Living with chronic pain - a longitudinal study of the interrelations between acceptance, emotions, illness perceptions and health status

Alexandra-Lelia Dima

A thesis submitted in fulfilment of requirements for the degree of Doctor of Philosophy

to

School of Health in Social Science
University of Edinburgh

September 2010

Declaration

I hereby declare that this thesis is of my own composition, and that it contains no material previously submitted for the award of any other degree. The work reported in this thesis has been executed by myself, except where due acknowledgement is made in the text.

Alexandra-Lelia Dima

Abstract

Psychological adjustment to chronic pain has been recently explored within three separate frameworks: a behaviour-focused account of chronic pain acceptance within the broader remit of Acceptance and Commitment Therapy; an emotion-focused approach with various research programs investigating the role of anger, fear, depression and also shame and positive emotions in chronic pain; and a cognition-focused perspective more recently reframed in terms of illness perceptions as part of a wider model of response to health threats, the Self-Regulatory Model. Although these frameworks have broad areas of overlap, limited research has been directed at integrating acceptance, emotions and illness perceptions into a common, comprehensive account of psychological adjustment to chronic pain. Such an account would be beneficial both for providing a parsimonious approach that would guide further research and for developing pain management interventions that would take advantage of existing research from all three domains.

The aim of the present thesis was to explore the possibility of integrating these separate areas by studying the relationships between the main concepts (acceptance, emotions, and illness perceptions) in the context of chronic pain.

Based on a review of the relevant conceptual and methodological issues of each domain, a theoretical analysis of the similarities and differences between them was developed, with particular emphasis on the potential of existing models to support an integrative account. This analysis provided specific hypotheses regarding each domain and the interrelationships between them, which were investigated in a longitudinal study on a heterogeneous sample of 265 chronic pain patients using the services of the NHS Lothian Pain Clinic and several patient support organisations. Data were collected via postal and online questionnaires at 3 time points, at 4½-month intervals (21% attrition rate). Validated questionnaires were used to measure the relevant constructs, with additional questions obtaining information regarding health status, medical history and demographics.

The confirmatory analysis (employing a variety of statistical procedures, from correlation to multiple regression, factor analysis, cluster analysis and structural equation modeling) largely confirmed the expected relations within and between domains and was also informative regarding the most suitable data reduction methods. A detailed psychometric analysis of the questionnaires used offered a complementary view on the theoretical and methodological issues involved. An additional exploratory analysis focused on identifying the comparative characteristics of acceptance, emotions, and illness perceptions in predicting health status indicators, controlling for contextual factors such as medical history and demographics. Although no significant longitudinal changes were identified in most parameters (confirming the clinical observation of chronic pain as a stable condition), the longitudinal data allowed an analysis of the stability of the concepts and of the magnitude of their relationships in this patient sample. The analysis of intra- and interpersonal variation via hierarchical longitudinal modeling confirmed the stability of the data, highlighted the necessity of studying variation at both levels, and revealed interesting moderation effects, explained via the proposed concept of 'discrimination ability' and several alternative mechanisms.

These results can be considered as first steps towards an integrative model of psychological adjustment to chronic pain. It is proposed that the behavioural, cognitive and emotional aspects need further conceptual clarification and these future efforts can be supported by the Cognitive-Affective Model of the Interruptive Function of Pain, within the wider framework of the Self-Regulatory Model.

Acknowledgements

Many thanks to my supervisors, Prof. Mick Power and Dr. David Gillanders, for their valuable guidance and advice, and for their encouragements and support. To my colleagues, especially to Anne Finucane and Nuno Ferreira, for the interesting and stimulating discussions and for their friendship.

To my husband and best friend, Dan Dediu, for being always beside me, for helping me find solutions to so many practical issues, for his confidence in my abilities, for inspiring me to set my standards high, to always try to expand my knowledge and to appreciate science despite its uncertainties and limitations; I would not be here without him. To my family and friends for their continuous emotional support, especially to Mirabelle Maslin, also for her help in editing the final letter to the study participants.

To the College of Humanities and Social Science, for offering me the possibility to study the fascinating domain of health psychology, and the financial support via the Principal's Award.

Last but not least, to the medical team and administrative staff within the Lothian Chronic Pain Clinic, and the members of Pain Association, PainSupport and BackCare, who made this study possible, and to the participants in the survey, who generously and patiently responded to the detailed questions and offered their feedback and encouragements.

Contents

List of Tables

List of Figures

Chapter 1

Introduction

For health psychology, chronic pain adjustment is one of the most difficult issues to investigate. The blurred boundaries between physical symptoms and subjective experience are particularly apparent in pain perception, not only making a biomedical approach ineffective, but also raising substantial barriers in the search for explanation and control of the phenomenon within a biopsychosocial framework. Multiple lines of investigation have been followed, leading to the inevitable problem of overlap and selection between models.

The necessity of integrative efforts, both theoretical and empirical, becomes increasingly evident given the proliferation of research and intervention programs. A comparison between programs in order to select the most effective under certain conditions would inevitably involve losing the valuable contributions of the rejected alternatives. A more fruitful approach would require a detailed theoretical and empirical investigation of parallel research directions in order to identify both their common elements and unique contributions, and use them as building blocks for future research within an integrative framework to inform intervention (professional and personal) aimed at improving adjustment to chronic pain. The present thesis attempts such an investigation.

1.1 The present thesis

Psychological aspects of living with chronic pain are currently explored within three main frameworks, which can be considered as mainly focusing on three psychological domains: behaviour, emotion and cognition. The behavioural approach is centred on the concept of chronic pain acceptance, as a special case of a more general characteristic, psychological flexibility, developed within the theoretical basis of Acceptance and Commitment Therapy (ACT; Hayes et al., 1999b; Hayes and

Smith, 2005). This recently developed therapeutic approach builds on the operant-behavioral and cognitive-behavioral traditions, adding new influences from humanistic and experiential traditions, among others, and a detailed philosophical foundation. The interest in emotion can be considered to unify various approaches to chronic pain research, from the study of emotion as a component of the pain experience to accounts of the role of discrete emotions, mainly anger, fear and sadness, but also shame and positive emotions. All these approaches share a common theoretical basis in emotion research, although they have been mostly studied separately. The approach that has recently emerged from the cognition-focused literature in health psychology is the self-regulatory model of health behaviour (SRM; Leventhal et al., 1992, 1997). Although in essence it is a model of the cognitive-affective interactions in guiding health behaviour, its research applications have mainly focused on the cognitive component, as described by the concept of illness perceptions.

All three frameworks have generated valuable empirical research and clinical interventions in health psychology and chronic pain. However there are substantial areas of overlap between them, and few efforts have been directed so far at integrating them in a comprehensive account of behaviour, emotion and cognition in adjustment to chronic pain. The aim of the present thesis is to explore the possibility of integrating these separate areas of research by studying the relationships between the main concepts of these approaches (acceptance, discrete emotions and emotion regulation strategies, and illness perceptions) in the context of chronic pain, and by examining existing models in order to identify the ones that could best support further integrative efforts.

1.2 Overview of chapters

Such an integrative attempt needs to be based on a thorough understanding of existing theory. The first four chapters review and critically analyse the literature focusing on each of the four substantive areas: pain, acceptance, emotions, illness perceptions.

Chapter 2 gives an overview of chronic pain, from the fundamental issues of definition, epidemiology, and classification, to the description of current theories, physiological mechanisms and treatment. Assessment of pain is discussed in more detail, and a brief historical account of the study of the psychological aspects involved is presented.

Chapter 3 reviews the theoretical and empirical support for the concept of chronic pain acceptance. ACT theory is described from its philosophical and general psychology bases to its account on psychopathology and the inherent difficulties in reconciling its distinct context-focused approach with the assumptions of the mainstream scientific approach. The concepts of acceptance, psychological flexibility and chronic pain acceptance are further described, and the relevant research briefly reviewed.

Chapter 4 addresses the various lines of research on the role of specific emotions and emotion regulation strategies in chronic pain adjustment, and also on affect as a component of the pain experience. A general overview of emotion research and the related issue of measurement is followed by a brief historical account of emotional issues in chronic pain. Research on the affective component of pain is next reviewed, followed by separate analyses on the role of five discrete emotions (anger, sadness, fear, shame and happiness), and on the role of emotion regulation and interactions between emotions.

Chapter 5 focuses on the SRM. Its theoretical foundations, model statements, measurement and applications in various areas of health psychology are presented first, followed by an overview of the SRM research in chronic pain within the wider perspective of cognition-focused literature.

Chapter 6 attempts to link behaviour, emotion and cognition by reviewing previous studies focusing on interrelations and existing models of pain and chronic pain adjustment. It also discusses methodological issues relevant for any integrative effort, such as the requirements of developing and testing theoretical models. It is proposed that the cognitive-affective model of the interruptive function of pain (Eccleston and Crombez, 1999) together with the SRM have the potential to support further integrative efforts. Based on the theoretical analyses so far, specific hypotheses and exploratory goals are set for the present empirical study.

Chapter 7 describes the present study, from the data collection procedure and sample characteristics, to the results of confirmatory and exploratory statistical analyses. Further details regarding the statistical approach used in structural equation modeling and the data preparation are given in Appendices B and C. Complementary analyses of the psychometric properties of the questionnaires used are presented in Chapter 8.

Chapter 9 brings together the theoretical and empirical findings and offers interpretations of the various results in light of the previous theories and research presented. In accord with Diefenbach et al. (2008), it is proposed that working towards an integrative account of behaviour, emotion and cognition in chronic pain adjustment is better served by multiple detailed analyses of the interactions between various related concepts rather than by adding variables to increasingly complex (and inevitably redundant) models.

Chapter 2

Living with chronic pain

2.1 Introduction

For many people, chronic illness is a challenge on many domains of their lives. Adapting to the changes that illness brings on one's personal, occupational and social life demands significant psychological resources. Research in health psychology builds on the observation that one's decisions in such circumstances can have a significant impact on one's health status, in terms of both symptom reduction and well-being. However it is often difficult to identify the 'right thing' to do, feel or think in such situations. Decades after the biopsychosocial model of illness has been proposed to replace the biomedical approach (Engel, 1977), a wide variety of theories are available to inform interventions and alleviate the sufferer's experiences of illness, yet many questions and difficulties remain in both research and practice.

The difficulties of understanding the psychological implications of chronic illness increase in the situation where pain is a major symptom. The organismic reaction to pain dominates the individual's consciousness to such a degree that the constant exposure to this phenomenon blurs the boundary between psychological and physical suffering and often defies efforts to manage the condition. The all-encompassing nature of pain leads to major difficulties in research as well. Separating this complex phenomenon into distinct and measurable constructs to make possible the accumulation of knowledge and improvement of intervention has proven to be a challenging task. This chapter aims to sketch the complexity of chronic pain as an object of study and to review the efforts to describe, measure, explain, and treat it.

2.2 Chronic pain - definition

Pain is defined by the International Association for the Study of Pain (IASP) as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey and Bogduk, 1994). According to IASP, pain "is a psychological state". An accompanying note follows this definition in the IASP document, highlighting the subjective nature of pain, and also its variable relationship with both nociception and language descriptions. The double nature of pain, sensory and emotional, is underlined to differentiate it from other similar experiences (such as pricking, or dysesthesias) which may not involve both qualities.

Two aspects of the IASP definition and note deserve mentioning at this early stage, as a preview of the more detailed analysis that follows. First, the emotional component of pain is considered related to its unpleasantness. This note is particularly relevant to the present study. The unpleasantness is considered to reflect the threat associated with tissue damage (Chapman, 2004). But from the point of view of emotion theory, unpleasantness is only one extreme of the valence continuum, which is only one of several dimensions of emotional life (Fontaine et al., 2007). It is usually related to withdrawal (as opposed to approach) behaviours, to negative emotions and to defense responses (as detailed in Subsection 4.2.1). Moreover, dimensional views of emotion are competing with discrete emotions theories and componential approaches in explaining affective phenomena. Briefly, there is more to emotion than unpleasantness, and behind the limited focus of this definition lies a complex relationship between emotional life and the affective and sensory components of pain and related behaviours (reviewed in Chapter 4).

The second aspect is pain reported in the absence of any pathophysiological cause. The IASP Task Force on Taxonomy mentions that, although in this case causes are usually psychological, such pain is undistinguishable from pain caused by tissue damage and they advise 'accepting' the patient's report as pain. This summarizes one of the long-lasting controversies in pain research and practice, with dramatic emotional consequences in the doctor-patient relationship. The relationship (and distinction) between psychological and physical in chronic pain is a sensitive topic, and central to the efforts to establish causality in order to devise effective treatment options.

Pain has been usually classified into acute (phasic), subchronic (prolonged) and chronic (clinical) (Millan, 1999). Acute and subchronic pain accompany tissue

injury and subside with healing. In contrast, chronic pain is defined usually as pain that lasts longer than the normal tissue healing time: 3 months, or 6 months for research purposes (Merskey and Bogduk, 1994). However, besides the situations when pain is prolonged beyond the course of the concurrent injury/disease, chronic pain describes also cases in which the co-occuring disease is chronic in nature. In such situations the distinction between acute and chronic pain is less clear. Another type of situation characterised by chronic pain is the presence of pain for long periods of time in the absence of any associated tissue injury, neither as an initial trigger or as an accompanying condition. In all three situations, the time dimension introduces changes that make the pain similar in many respects: physiological, social, psychological, functional.

A frequent distinction in chronic pain is made between malignant (cancerous) and non-malignant (benign) pain. Although there are no physiological differences known at present, the differences related to the implications of the treatment dynamics in the current medical system, the strong connection with tissue pathology and treatment toxicity in cancer pain and the distinct time implications of terminal cancer pain justify the separation (Bonica, 2001, Ch. 10). The focus of the present review and study is benign chronic pain.

2.3 Epidemiology and impact of chronic pain

A recent systematic review (Harstall, 2003) reported a mean prevalence of chronic pain in the general population estimated at 35.5% with a range from 11.5% to 55.2% (according to data from mostly Anglo-Saxon and West European countries). The prevalence estimates vary depending on the definition used, country, study methodology. For example, severe chronic pain prevalence has been estimated at 11%, chronic widespread pain at 7.2%. A later study in 15 European countries and Israel (Breivik et al., 2006), reported that Spain has the lowest estimate of 12% and Norway the highest, 30%. In Scotland, sampling in primary care has led to an estimate of 18% according to McEwan, 2004, while a community study, Elliott et al., 1999, reported 46.5%.

The prevalence of chronic pain depends on gender and age, with women more likely to report pain than men (due to both social and biological factors), and some conditions such as joint pain and fibromyalgia increasing in prevalence with age. Various surveys reported pain prevalence estimates in older adults of up to 86% (Hadjistavropoulos and Craig, 2004).

Chronic pain impacts the society dramatically on multiple levels, from the physical and mental health, employment and daily life functioning of the sufferer to the economic costs associated with unemployment, welfare and healthcare. For example, a study by Maniadakis and Gray (2000) estimated the total economic burden of chronic backpain in the UK in 1998 at £6 to 12 billion. Chronic pain sufferers use healthcare services more frequently and frequency increases with level of pain-related disability, according to reports of respondents to a community telephone health survey (Blyth et al., 2004).

2.4 Pain theories - from the body-mind split to the body/self neuromatrix

Understanding of pain has evolved dramatically in the last decades. Until the middle of 20th century, pain was described in terms of a response proportional with the amount of damage to the physical body. Descartes' mechanistic model of pain (part of his dualist view of the human being) is considered the first theory in the field, and states that the pain is the direct result of an external nocive stimulus, just as the sound of a bell which hangs at one end of a rope is the direct result of someone pulling the other end (Melzack and Katz, 2004). This "alarm bell" theory of pain led to the development of the specificity theory: pain is the result of the activity of a pain center in the brain, activated via impulses through pain fibers the spinal cord by specific local pain receptors responding to injury. Lack of visible injury or disease was diagnosed as a psychiatric condition (in Descartes perspective, a matter of the mind and not of the body). It is important to note that, despite the mismatch with clinical data (Melzack and Wall, 1970) and the dramatic theoretical developments that overrode it, this theory still prevails in medical education today, according to some reports (Jay, 2007, p. 259), and also to a large extent in the mainstream culture.

Initial attempts to overcome the limitations of the specificity theory are generally grouped under the name of pattern theory (Melzack and Wall, 1970). The main proposal consists in stipulating various mechanisms of pain modulation to account for the lack of direct correspondence between the nocive stimulus and the pain response, such as summation of intensities of various stimuli in the spinal cord, the existence of specific circuits in the spinal cord that reverberate in situations of intense stimulation, the existence of control mechanisms that normally prevent summation and

are damaged in pathological conditions, or the production of spatiotemporal patterns that differentiate pain from other sensations (Melzack and Wall, 1970). These theoretical advances changed the focus from periphery to the central nervous system in explaining pain. A further step forward was Melzack and Wall's gate control theory of pain: modulation of impulses from the periphery is achieved by a gating mechanism in the spinal cord. The gating mechanism is influenced both by the amount of activity in the nerve fibers that transmit non-nociceptive impulses from periphery and by nerve impulses descending from the brain. Their seminal article in 1965, in addition to stimulating research on the physiology of pain modulation, represented the starting point of a broader understanding of pain as encompassing also psychological phenomena: "the model suggests that psychological factors such as past experience, attention and emotion influence pain response and perception by action on the gate control system" (Melzack and Wall, 1965).

As a result of several decades of research into the psychophysiological mechanisms of pain, theory has recently gone beyond the peripheral and spinal processes involved towards the understanding of the brain mechanims. A theory that addresses this aspect is the neuromatrix model of pain proposed by Melzack in recent years (Melzack, 2001; Melzack and Katz, 2004). It stipulates the existence of a body/self neuromatrix comprised of sensory, affective and cognitive neuromodules. It receives inputs from sensory signalling systems and cognitive and emotion related brain areas and delivers as outputs pain perception (in its sensory, affective and cognitive dimensions), action programs and physiological stress-regulation programs. Therefore, according to this model, pain is a multidimensional experience produced by a distributed neural network which includes but is not limited to the somatic sensory pathways, and most importantly can even act without them in certain conditions (e.g. phantom limb pain).

The neuromatrix model is a telling summary for the current advances in understanding the psychophysiology of pain, which will be briefly described in the next section. Moreover, it bridges the psychophysiological gap between pain and our understanding of emotion and cognition: the neuromatrix is seen as a unified system which performs a "cyclical processing and synthesis of nerve impulses" (Melzack and Katz, 2004) and constantly generates patterns (neurosignatures) that update the current state of the body as a whole[1] . This unified pattern includes subsets of

[1]This constant update conceptually resembles theoretical proposals of several emotion theories, such as the conscious access to continuous changes in the organism's neurophysiological state in the core affect model (Russell, 2003), but also with the continuous interpretation of experience stipulated by process models of emotion (such as appraisal models, Ellsworth and Scherer, 2003),

neurosignatures created by neuromodules which correspond to (but are not equal to) events in various parts of the body, which determine awareness (via a so-called "sentient neural hub") and movement (via the action neuromatrix). Some of these neurosignatures are experienced as different qualities of pain and generate specific actions and physiological changes. From this point of view, the neuromatrix model is more than a model of pain, is a model of central processing. It speaks eloquently of the difficulty of separating the physical, psychological and social aspects in both research and treatment. This unitary conception has extremely important implications for pain measurement (detailed in section 2.7.3).

2.5 Pain - physiological mechanisms

A brief insight into the physiology of pain is absolutely necessary for understanding the phenomenon of chronic pain. At present, our knowledge about pain distinguishes between peripheral, spinal and supraspinal systems that transmit and modulate the neural impulses which generate pain perception and pain behaviours. There are both neural and chemical mechanisms that take part in this process (Jay, 2007). While pain is often the product of nociception (i.e. the perception of noxious stimuli), the two terms are not interchangeable, as pain can also be experienced in the absence of nociception (Melzack and Katz, 2004). Nociception is described in terms of four processes: transduction, transmission, modulation and perception (Suchdev, 2002).

According to Jay (2007), Suchdev (2002), and Bonica (2001), at the peripheral level, in case of tissue injury, a local biochemical response takes place and various inflammatory and algesic substances are released (including prostaglandins, histamines, bradykinins, substance P). The local sensory neurons are activated (and their sensory thresholds modulated) by these substances and/or other mechanical, termal or biochemical stimuli. The conversion of the local biochemical response to injury into a neural response is called transduction. Transmission of the neural impulse from the periphery to the spinal cord is achieved by mainly two types of nociceptive sensory fibers: A-δ fibers are myelinated and have smaller nociceptive fields (they participate in generating the immediate, sharp pain after the injury); C fibers are unmyelinated and have terminations spread over a wider area (therefore participate

while the existence of specific patterns of activation resembles discrete emotion theories (Ekman, 1999). It is yet unclear how these models would best communicate; one first attempt to bring together emotion and pain theory is the sequential processing model of pain affect (Price et al., 2001), presented in Chapter 4. The neuromatrix model still awaits more precise conceptualisation and testing; the difficulties of testing it with linguistic data will be discussed in Subsection 2.7.1.

in the generation of a delayed, diffuse pain). The smaller proportion of A-δ fibers in the visceral structures (innervated by the autonomic nervous system) is part of the explanation for the lack of specificity of visceral, sympathetic and muscular pain.

The dorsal horns of the spinal cord are the location of the first synapse and an important center for the integration of sensory information from both noxious and benign stimuli. Modulation of the neural impulse related to tissue injury is mainly performed by the substantia gelatinosa, which (as stipulated by Melzack and Wall's gate theory) is a set of interneurons with an inhibitory effect on the transfer of information to the ascending pathways towards the brain. Activity in the non-nociceptive sensory fibers has an excitatory effect on substantia gelatinosa, as have descending pathways from some brain structures (such as cortical and diencephalic systems, the medulla, periaqueductal gray).

There are multiple and complex ascending pathways that participate in various aspects of pain perception. The spinothalamic tract is the main ascending pathway and is divided into two systems. The neospinothalamic tract has large myelinated fibers connected via the thalamus directly with the somatosensory cortex, and thus participate in generating the sharp, localised pain immediately after tissue damage which possibly helps locate the injury and assess its severity. The paleospinothalamic tract has both large and small fibers, less myelinated, which synapse in various structures (periaqueductal gray, hypothalamus, reticular formation, thalamus and other brain stem and midbrain structures) which transmit the impulses diffusely to cortical and limbic structures; this pathway is responsible for the long-lasting and poorly localised pain experienced some time after injury, and possibly also for some affective and sympathetic responses and arousal. Other ascending pathways participate: the spinoreticular tract and the spinomesencephalic tract (involved in autonomic, behavioral and motivational aspects of pain), the trigeminothalamic tract (equivalent to the spinothalamic tract for the head and neck), the dorsal column system (with a role in transmitting visceral nociceptive information, and possibly inhibition of pain), the propriospinal tract (with possible role in maintaining chronic pain), the spinohypothalamic tract (with possible affective and motivational roles).

Multiple cortical and subcortical centers receive information from the ascending pathways and interact with each other to generate the pain experience. The most notable, according to present knowledge, are: the reticular formation (influencing arousal and motivational, affective and autonomic responses), the thalamus (a

major relay station connecting with various cortical areas), the limbic system (apparently involved in motivational and emotional responses), the hypothalamus (participating in the autonomic and neuroendocrine control of pain), the somatosensory cortex (responsible for the discriminative aspects of pain perception), the frontal cortex (involved in behavioural and motivational aspects). Research in this area is still at the beginning. However, according to Melzack's neuromatrix model, it is thought that the distributed processing of the sensory information by all these centers generates neurosignatures that are perceived as pain and acted upon. In some situations (such as phantom limb pain), current sensory information is not necessary for production of neurosignatures. These centers also participate in feedback mechanisms of pain modulation, via various neuroendocrine pathways that release serotonin, norepinephrine, cholecystokinin and endogenous opiates in which the periaqueductal gray plays an important role. Other neurotransmitters and neuropeptides have been found important for pain modulation: calcitonin gene-related peptide, somatostatin, substance P, dopamine (Jay, 2007; Suchdev, 2002; Bonica, 2001)

The complexity of the neural networks involved in pain perception, modulation and behaviour is just beginning to be unveiled. However, the broad array of centers and pathways involved in pain (very few dedicated only to pain perception), speaks quite eloquently of the psychological implications of experiencing pain: it is an organismic response, that dominates (and overwhelms at times) the individual and involves all aspects of his/her psyche: emotional, motivational, cognitive, behavioural, autonomic. It also highlights the difficulty of disentangling all these aspects, in both research and treatment. The situation becomes even more complicated in chronic pain, where the interrelationships between them are modified by the long-term experience of pain.

Given the complexity of the pain process, there is no surprise that many things can go wrong. Depending on etiology, various peripheral, spinal and central mechanisms have been described to participate in the onset and perpetuation of chronic pain.

One of the main mechanisms is the process of sensitisation: repeated stimulation, instead of decreasing sensitivity as in other sensory fibers, determines a lowering of the threshold and an increase of response duration. In the case of continuous stimulation of nociceptors due to various accompanying health conditions (for example osteoarthritis), a peripheral mechanism that contributes to pain is the sensitisation of the sensory neural fibers to both noxious and non-noxious stimuli by the inflammatory and algesic substances via both neuroactive and vasoactive pathways. At

the spinal level, sensitisation of afferent fibers determines spinal reflexes that enhance nociceptor reponse, muscle tension and sympathetic activity (Bonica, 2001). Spinal receptive fields suffer various anatomical and physiological changes that lead to hypersensitivity to new sensory inputs. The phenomenon of wind-up has been proposed as an additional mechanism: wide-dynamic range neurons, under constant C fiber stimulation, are changing their on-off functioning to a constant activation, which also impacts on the sympathetic regulation of the affected area. At the central level, sensitisation is the result of physiological and morphological changes that influence the descending modulation of pain-related neural impulses (Jay, 2007). The phenomenon of sensitisation is often referred to as neuroplasticity (Melzack and Katz, 2004).

In neuropathic pain (pain due to partial or total nerve lesion), several other mechanisms have been proposed, such as spontaneous activity in damaged nerve fibers, demyelination due to chronic irritation, changes in the physiology of dorsal root ganglia, or anatomical changes such as the formation of axonal sprouts or neuroma, which lead to spontaneous activity (Jay, 2007). If the lesions are at the level of the central nervous system (for example due to spinal injury, thalamic lesions, or multiple sclerosis), the pain patterns are also due to deafferentation and neuroendocrine imbalances between various centres which lead to disinhibition of the pain network. The study of the mechanisms involved is progressing rapidly, and various competing hypotheses are under scrutiny (Bonica, 2001, Ch. 23).

The lack of any adaptive significance of chronic pain as opposed to acute and subchronic pain has lead to the latter to be considered as good (physiological), while former is seen as bad (pathological; Millan, 1999). Evolutionary interpretations of chronic pain describe it as a by-product of neural plasticity that is adaptive in learning and memory processes (Sufka and Turner, 2005).

2.6 The problem of classification

The complexity of the mechanisms involved in chronic pain is reflected in the difficulties of identifying distinct types of chronic pain manifestations. From the initial, time-related definition of chronic pain, several attempts have been made to characterise this phenomenon from a more comprehensive perspective.

Several taxonomies have been developed based on expert consensus and multiple criteria. One such example is the classification of the International Association

for the Study of Pain (IAPS), which includes five axes: region of the body, body system involved in pathology, temporal characteristics, patient-reported intensity and time since onset, presumed etiology (Merskey and Bogduk, 1994). A list of chronic pain syndromes following similar criteria is made available in the same document. Although more clinically meaningful than the various classifications based on single criteria, these taxonomies are difficult to use in research and clinical practice, as categories are not mutually exclusive, not fully reliable and not directly related to clinical interventions and outcomes; they also exclude information on psychosocial characteristics, which make them less applicable to interdisciplinary pain management interventions (Bonica, 2001, Ch. 2).

Another set of taxonomies have been developed based on empirical data. For example, a graded classification of chronic pain severity was developed based on measures of pain intensity and temporal characteristics and pain-related disability (Von Korff et al., 1992). According to this classification, four Chronic Pain Grades can be distinguished reliably: Grade 1 - low intensity, low disability, Grade 2 - high-intensity, low disability, Grade 3 - high, moderately limiting disability and Grade 4 - high, severely limiting disability. Another, complementary, classification of chronic pain based on various indicators of pain severity, perceived responses of significant others and interference with activities distinguishes between three profiles: dysfunctional (increased pain severity, life interference and affective distress), interpersonally distressed (with little perceived support) and adaptive copers (decreased pain severity, life interference and distress, with higher perceived control; Turk and Rudy, 1990). The authors recommend using this taxonomy in addition to the medical classification of pain conditions.

A recent approach (Von Korff and Miglioretti, 2005) extends this multivariate perspective on chronic pain by including the time dimension: on the basis of several measures of current chronic pain status and other variables that have been proven to predict future pain severity, a risk score is computed that leads to distinguishing possible and probable chronic pain. Possible and probable chronic pain correspond to the 50% and 80% probability thresholds of future clinically significant pain. This prognostic approach shifts the focus from labelling the current status of the patients to the possible risks they face (and the ways to reduce them), while viewing chronic pain as a continuum within which the severity of the condition can change and is inherently uncertain, rather than a fixed, immovable, stigmatising diagnosis. The risk score has been shown to be a good predictor of future chronic back pain according to Chronic Pain Grades classification (Von Korff and Miglioretti, 2005), as

well as a better predictor, compared to pain duration, of future clinically significant pain, physical function, pain-related worry, unemployment and long-term opioid use in back pain, headache and orofacial pain patients (Von Korff and Dunn, 2008). Results have been replicated on a UK low back pain sample (Dunn et al., 2008). Although these empirical classifications lead to more objective and reliable results, their utility is limited by the theoretical focus of the variables included (Bonica, 2001, Ch. 2).

To date, there isn't a commonly agreed taxonomy of chronic pain and classification is constantly evolving. For example, previous diagnoses of reflex sympathetic dystrophy and causalgia have been recently transformed into complex regional pain syndromes I and II (without or with nerve damage; Merskey and Bogduk, 1994), and a proposal has been made to describe fybromyalgia and other related diagnoses as central sensitivity syndromes (Yunus, 2008). Many of the individual health conditions fall in more than one of the current diagnoses and are described by a complex mix of pain-related symptoms, triggers and aggravating factors, comorbid illnesses and idiosyncratic reactions and adaptations.

In addition to the idiosyncratic features, manifestations common to all conditions are present in most epidemiological data, especially with regards to psychosocial-behavioural aspects (Turk and Rudy, 1990). Chronic pain is generally characterised by a lack of a close temporal and spatial relationship with a stimulus (as opposed to acute pain) and uncertainty regarding the future course of the condition, which lead to physiological, psychological and social changes. Interference with autonomous nervous system functioning can lead to muscle tension, decreased heart rate and blood pressure, and failure of immune responses. The continuous challenge of the chronic condition also leads to associated physical and psychological problems such as fatigue, insomnia, anorexia, depression, apathy. This common profile has lead to considering chronic benign pain as a relatively homogeneous condition, for which similar interventions can be applied (Bonica, 2001, Ch. 10).

2.7 Assessment

The complexity of the physiological mechanisms of pain and its difficulties of classification are parallelled by the multitude of issues involved in measuring pain and pain related outcomes and risk factors. I will refer here only to the clinical assessment of pain in humans, and exclude the issues related to measurement in animal

subjects and to experimental methods in acute pain research, for which I would direct the interested reader to other sources (e.g. Kruger, 2001).

2.7.1 Assessment of pain intensity and pain quality

It is commonly acknowledged at present that pain is a multidimensional phenomenon. However, clinical assessments usually focus on pain intensity, even if it is known that this aspect addresses only a small part of the whole phenomenon and its impact on the individual (Turk and Okifuji, 2001, Ch. 1). Several methods of measuring pain intensity have been developed: threshold approaches, magnitude estimation, signal detection theory, unidimensional subjective rating scales, observation and verbal pain assessment (Skevington, 1995, Ch. 2). While the first three methods focus on aspects related to nociception and are part of a set of methods known as quantitative sensory testing (QST; Fillingim and Lautenbacher, 2004), the last two address aspects related to the overall experience of suffering.

Threshold approaches focus on assessing a few related concepts: sensation threshold, pain perception threshold (the intensity at which the sensation becomes painful), pain tolerance level (the minimum intensity at which the pain becomes unbearable), the pain sensitivity range (between pain perception and pain tolerance levels), and the drug request point. They are measured with various methods of pain induction via thermal, mechanical, electrical or chemical stimuli applied usually in ascending or descending intensity series. These approaches have been used extensively in experimental conditions in researching various factors that influence the link between a physical stimulus and a pain sensation, from individual characteristics to context and interactions of factors (Skevington, 1995, Ch. 2). Especially because of these interfering factors they are of limited use in global clinical assessments of chronic pain, where little control is possible over individual differences even if some context characteristics can be kept constant. Their use in clinical assessment is focused on helping diagnosis of abnormalities in pain processing, predicting response to acute surgical pain or chronic pain interventions and measuring intraperson pain perception changes, for example due to treatment (Edwards et al., 2005b; Arendt-Nielsen and Lautenbacher, 2004).

Magnitude estimation is a psychophysical ratio scaling method adapted to the study of pain: the subject is asked to estimate the relative intensity of a set of painful

stimuli. Thus, instead of the dichotomous information obtained in threshold measurement, the researcher obtains continuous data regarding the whole range of intensity perception. The estimate is expressed in either verbal terms or in different other modalities, such as using handgrip force (procedure known as cross-modality matching; Skevington, 1995, Ch. 2). Although it constitutes an improvement in the precision of measurement compared to threshold approaches, magnitude estimation has a limited use due to the cognitive complexity of the task. Moreover, it has a similar focused use in clinical practice as threshold measures. In the efforts to eliminate subjectivity from pain self-report, it excludes from the concept of pain intensity many aspects that mediate the relationship between stimuli and pain sensation; therefore it pays for reliability of measurement with a limited operationalisation of the concept itself.

Signal detection theory (SDT) is based on similar pain induction trials with stimuli of different intensities; two receiver operating characteristics are computed: the index of discriminability (low values would indicate an interference with the sensory processes) and the pain report criterion (high values might reflect a stoic attitude towards reporting pain). Separating sensory discrimination from response bias has been considered an important advantage of SDT. However the method has similar drawbacks to the previous two, limiting its applicability in clinical settings (Skevington, 1995, Ch. 2).

Evidence is accumulating for the usefulness of QST in clinical settings, for identifying patterns of pain perception relevant for diagnosis and treatment and as an additional measure of pain severity or treatment outcomes, besides its application in the experimental research of pain mechanisms (Fillingim and Lautenbacher, 2004). However, it is difficult to use in everyday clinical practice due to the equipment and control requirements of performing accurate testing and it has limited application in the study of chronic pain. Moreover, it is based on a definition of pain as nociception and therefore on the assumption of stimulus-sensation equivalence. Paradoxically, this limited focus has made it useful in revealing the influence of a multitude of factors on pain reporting and thus the complexity of this phenomenon (Fillingim and Lautenbacher, 2004). The key behaviour in all methods is the self-report of pain perception for each trial, which inevitably is subject to various social and psychological influences in addition to physical conditions (such as presence of an analgesic). Efforts of eliminating self-report together with its various influences from pain assessment have focused on physiological measures (muscle tension, cardio-vascular parameters, nociceptive withdrawal reflexes, evoked potentials, brain imaging) but

has not yet resulted in identifying a reliable indicator of the pain response (Arendt-Nielsen and Lautenbacher, 2004). Instead of trying to eliminate these influences, other methods reviewed below have focused on pain intensity as a subjective experience viewed as the result of interactions between multiple person and context related factors.

The simplest and most widely used methods are the unidimensional subjective rating scales, such as the visual analogue scale (VAS): usually a horizontal line of 10 cm with verbal labels at the two ends and no numerical divisions. Variations are VAS for pain relief or for sensory and affective aspects of pain intensity. The advantages of VAS are the speed and facility of application and the high correlations with more comprehensive methods, although there are doubts regarding its accuracy (Skevington, 1995, Ch. 2). Its disadvantages stem from the same simplicity, which leads to limited information obtained and therefore limited relevance. As a global measure, it is impossible to assess what factors have the biggest impact on the unique scores; for example, research suggests that emotional qualities of pain are most represented in the global score in some patient groups (Clark et al., 2002). Also, the use of a single scale does not allow controlling for intraindividual variability and this leads to a decrease in the reliability of the measure. Other forms are the Verbal Rating Scales (VRS, lists of adjectives describing pain at increasing intensities) and Numerical Rating Scales (NRS, usually 11-point scales, from 0 to 10, on which the respondents are asked to locate their pain intensity); both are frequently used due to reasons similar to the VAS and with similar drawbacks (Jensen and Karoly, 2001).

One obvious source of information regarding the individual's pain is the observation of behaviour. It is actually the only method applicable where self-report is not available, such as in infants or unconscious patients. Gestures such as guarding, bracing, restricted movements, vocalisations such as moaning, grunting, and facial grimaces are coded by trained observers and used to estimate pain intensity based on its impact on these behaviours (Skevington, 1995, Ch. 2). Besides being time-consuming, observation has the obvious drawback of not being able to ascertain the specific meaning of the behaviour without the addition of self-report (for detailed accounts of the assessment of facial expression and behaviour in pain see Keefe and Smith, 2001, and Craig et al., 2001).

Verbal pain assessment overcomes to some extent the drawbacks of unidimensional pain intensity scales and observational methods: more comprehensive than the former, easier to administer than the latter. However in doing so it needs to tackle

the limitations generated by the subjectivity of pain language. The McGill Pain Questionnaire (MPQ; Melzack, 1975) is an illustrative example of these methods.

MPQ was the first attempt to go beyond pain intensity into the field of the subjective experience of pain and try to measure pain quality and it remains one of the most used instrument for pain assessment today. The idea behind MPQ developed during discussions between its author, Ronald Melzack, and chronic pain sufferers. He noticed the rich vocabulary used to describe pain and developed a list of pain descriptors which were categorised by respondents in a later study into three classes: sensory - discriminative, motivational - affective and cognitive - evaluative (Melzack, 2005).[2] Efforts to use this qualitative data in a quantitative manner led to the development of the MPQ. The questionnaire consists of a list of 20 sets of between two and six adjectives from which the subject is requested to choose the words that best describe their pain, maximum one word per set. The questionnaire includes additional questions related to global assessment of present pain intensity on a 5-level scale with anchor words, location of pain on line drawings of the human body, and temporal properties of pain. In each set, adjectives are ordered based on previous research regarding their relative level of pain intensity, which indicated a high degree of consensus between subjects' ratings (both doctors and patients). Melzack recommended administration by a trained researcher or nurse. Four types of data can be obtained: two highly correlated pain rating indexes (based on mean scale values and on the rank values of words), the number of words chosen and the present pain intensity (Melzack, 1975). Alternative scoring methods have been subsequently proposed (Melzack and Katz, 2001).

The reliability and validity of MPQ is supported by studies that have identified similar adjective categorisation patterns for different populations, a good test-retest reliability coefficients for short periods, confirmation of the 3-dimensional structure (although findings are not consistent, especially regarding the distinction between emotional and evaluative dimensions), sensitivity to pain reducing interventions, and a relatively good discriminative capacity between various pain syndromes (Melzack and Katz, 2001). It has been used for measuring effectiveness of clinical interventions, distinguishing pain qualities in different disorders, developing experimental pain induction methods that are similar with clinical pain regarding pain qualities (Holroyd et al., 1992).

[2]Crawford (2009) however notes the existence of a different discourse in the initial articles: descriptors were derived from existing clinical literature.

However MPQ is subject to many critiques. Its validity has been questioned due to response format (Keefe, 1982), reliance on the questionable assumptions of precision in language use and lack of variability in word comprehension and on questionable questionnaire development methodology (Skevington, 1995), unclear structure and lack of discriminant validity of the subscales (Holroyd et al., 1992), misclassification of some descriptors (Fernandez and Towery, 1996), and even its role in redefining specific medical conditions as invariably painful (Crawford, 2009). The evidence supporting its clinical utility in differentiating patient groups is also not consistent across studies (Skevington, 1995). Moreover, the list of descriptors might be actually a way of imposing a language of pain on the patients without taking into account the different meanings that the words have for individual patients, or other more suitable words or expressions that could communicate their experience; lacking a context to interpret the descriptors more reliably, patients might simply respond by 'guessing' to comply with the questionnaire completion task (Skevington, 1995). Despite these critiques, MPQ is still used in clinical practice and research in its original format, and very few modifications have been proposed (Melzack and Katz, 2001, p. 45).

The controversy related to MPQ reflects more global issues of measuring pain intensity and pain qualities through language. The subjectivity of personal pain experiences combined with the high variability of language use lead to real difficulties in pain measurement. This problem is mirrored in emotion measurement (see Section 4.2.3). In using pain descriptors or emotion labels, one cannot simply rely on the assumption that words are directly encoding some 'real' neuro-psychological entities and ignore the intersubjective nature of language use and the interaction between language and neurophysiology. Although the neurophysiological basis of these phenomena cannot be denied, a perhaps more adequate description is that pain experience (as emotion) is actually co-constructed during the interaction at diadic or societal levels rather than directly extracted more or less reliably from a 'true' inner reality (such as Melzack's 'neurosignatures', or the Ekman's 'innate emotion programs'). Thus, any pain measure is participating in the construction of pain perception (Crawford, 2009). Intersubjectivity might account at least partly for the problem of instrument variance in some verbal pain measures (Holroyd et al., 1996). Generally, when using language in measurement, we are inevitably encumbered by one of its most useful features: its flexibility in creating meaning in interaction.

2.7.2 Assessment of other pain related aspects

Measuring pain perception is obviously not enough to describe such a multifaceted phenomenon. There is an increasing interest in the multidimensional assessment of pain in both initial diagnosis and treatment evaluation. Various aspects of the sufferer's experience, such as functional impairment, emotional distress, health-care-seeking behaviours, work status, are considered both indicators of pain severity and distinct constructs which influence and are influenced by pain perception.

One of the most used measures of physical and emotional functioning is the Sickness Impact Profile (SIP), a 136-item questionnaire administered via interview or self-report which generates a 12-dimension profile of physical and psychosocial disability: ambulation, mobility, body care and movement, social interaction, communication, alertness, emotional behaviour, sleep and rest, eating, work, home management, and recreation (Bradley and McKendree-Smith, 2001). The variety of the aspects included is indicative of the extent of the impact of pain (shorter, more focused versions of the SIP have been developed consequently).

Another, more comprehensive, instrument is the West Haven - Yale Multidimensional Pain Inventory (WHYMPI; Kerns et al., 1985), which was developed from a cognitive-behavioural perspective and consists of twelve empirically derived scales. Five of them focus on the pain experience: interference in various domains of life, support from significant others, pain severity, life control, and affective distress. A second set of three scales measures others' punishing, solicitous, and distracting responses to the sufferer's behaviour. A third set of four scales assesses participation in 4 types of activity: household chores, outdoor work, activities away from home and social activities (Jacob and Kerns, 2001).

Both SIP and WHYMPI are applicable to chronic pain sufferers irrespective of the diagnosis or body area affected. Indeed, multidimensional pain assessment have led to new classifications of chronic pain sufferers, intended to help the development of biopsychosocial interventions applicable across diagnoses (Von Korff et al., 1992; Turk and Rudy, 1990; Von Korff and Miglioretti, 2005, as mentioned in Section 2.6).

A comprehensive assessment becomes increasingly important when efficacy, effectiveness and efficiency of the treatment methods are under scrutiny. Numerous concepts need to be assessed in such studies: pain reduction, patient satisfaction,

quality of life, reduction in health-care utilisation (medication, number of treatments), increases in functional activities (including return to work and reduction in disability payments; Okifuji and Turk, 2001). Turk and Dworkin (2004) reported on a consensus reached in the chronic pain research field regarding the assessment of treatment outcomes from six core domains: pain reduction, physical functioning, emotional functioning, patient satisfaction, negative health states and averse events, and patient disposition.

2.7.3 General issues in pain assessment

None of the measures described above could be considered a gold standard in pain assessment. Pain experience is not reducible to any of them, a more achievable goal is to aim to assess validly and reliably the various aspects of pain experience (Arendt-Nielsen and Lautenbacher, 2004), or manifestations present at various levels of the chronic pain condition: nociception, pain perception, pain appraisal, pain behaviour, and social roles for pain and illness (Dworkin and Sherman, 2001). But even this comparatively limited aim is encumbered by issues intrinsic to the phenomenon itself.

The correlated dimensions of pain

One of the obstacles in measuring pain dimensions is the difficulty of distinguishing between one concept and other related concepts. To ensure validity, a measure needs to be specific, and it is difficult to get specific when measuring a phenomenon that is as pervasive as pain. We have seen in the previous sections that pain is a systemic response, and it is perceived at the phenomenological level as an event that invades the whole person and has important repercussions at all levels of the person's life. According to the neuromatrix theory of pain, the neurosignatures that signal pain are subsets of a unified and continuously updated pattern which brings to awareness the state of the body as a whole (see Section 2.4).

Particularly in self-report, the distinctiveness of these subsets is not easily apparent. For example, measuring pain intensity or duration via self-report can activate evaluative processes which scan all the other levels of pain experience. Beyond the momentary physical sensation, the respondent estimates the intensity by its causes and consequences ('it hurt so much I could not move'). Thus, pain intensity reports can be influenced by assessment of physical or psychosocial disability due to the

measurement method, not only to the impact of one dimension over another in the sufferer's life.

As a consequence, it is difficult to assess to what extent the associations between pain dimensions are due to measurement methods or 'real' causal effects (unidirectional or reciprocal). Some studies show low or even no correlation between reported pain intensity and pain related disability (Jensen et al., 1992), while others consider intensity and associated disability as indicators of a main latent factor, pain severity (Von Korff and Miglioretti, 2005)[3].

Recall bias

Respondent recall bias is another issue that can affect reliability. Pain assessment via self-report usually involves asking sufferers to remember their experiences during the previous period of time. Recently, ecological momentary assessment of pain using electronic diaries has become more widely used (Jamison et al., 2001). Its main advantages are the lack of recall bias and the potential to analyse intraindividual variation (Litcher-Kelly et al., 2004). A number of studies have indicated a high rate of agreement between retrospective reports and momentary pain ratings (for example, Jamison et al., 2006). While pain intensity, persistence and associated disability are shown to be relatively reliable, other indicators of pain-related health status are more influenced by recall bias (such as pain related disability, doctor visits, pain onset time). It is influenced by individual characteristics (present pain severity, depression, frequency of health-care use, etc.) and special consideration needs to be paid to minimise this effect (Von Korff, 2001).

The subjective nature of pain

Apart from measures of physical function, which address only the most basic level of the pain phenomenon and thus are less influenced by self-report bias, all the other instruments for assessing different pain dimensions are confronted with the issue of subjectivity (Dworkin and Sherman, 2001).

For example, there can be no generally agreed criteria for pain intensity. Each person sets his/her own norms regarding what it means to be experiencing a lot of pain, and how much one can bear; this inevitably decreases between-subjects

[3]This issue becomes more sensitive in studies that aim to distinguish between determinants and outcomes of pain, in both cross-sectional and longitudinal designs. Parameters of association between concepts can be over or underestimated due to measurement; this aspect can become particularly relevant in structural equation models (as discussed in Chapter 6).

reliability, as differences between subjects can be due to their perception of present or retrospective pain intensity but also to their endorsing different intensity criteria. Also, the sufferer's own norms might not necessarily be stable across time, decreasing within-subjects reliability. These difficulties have contributed to the concerns related to malingering: when assessing a symptom that does not have an objective measure, and when reports can be influenced also by other personal gains (emotional, financial), there may be a doubt about the authenticity of the report. These doubts make the issue of pain reporting more contentious, although the incidence of pain fabrication is estimated to be low (Craig and Hadjistavropoulos, 2004, p. 309).

A related problem is the idiosyncratic nature of pain. Each sufferer may have unique experiences that would fit in general categories such as pain intensity, quality, pain related cognitions and emotions, etc. Selecting only a few examples in a questionnaire (and thus obtaining a low score) might mean that one has a low level of the particular characteristic, or that one simply did not find in the selected list the examples that related to one's own experiences. A partial solution is the development of illness-specific questionnaires, such as the Neuropathic Pain Scale (Jensen and Karoly, 2001) or the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC; McConnell et al., 2001).

What makes pain measurement more difficult is the intersubjective nature of pain (Crawford, 2009). As mentioned in section 2.7.1, it is unlikely that the neurosignatures possibly identifiable in brain activity (Melzack and Katz, 2004) are directly and uniquely related to words such as throbbing, shooting or stabbing, or even to other pain related nonverbal behaviours. A transformation of pain is taking place from tissue trauma to the subjective pain experience and the expressive behaviour of the sufferer, to the attribution of pain and response behaviour of the carer; multiple intrapersonal and contextual factors intervene in all stages (this process is further described in the communications model of pain; Hadjistavropoulos et al., 2004). These social influences have active roles in the construction of a verbal interpretation of one's inner pain experience, and are not easy to distinguish even via multidimensional pain assessment.

2.8 Treatment

Considering the complexity of physiological mechanisms involved and the difficulties related to assessment and diagnosis, the sometimes overwhelming problems

related to the treatment of chronic pain are not surprising. Usually, the treatment involves a mix of pharmacological approaches, physical therapy, psychosocial interventions, and a long list of complementary treatments such as acupuncture, chiropractic, aromatherapy, natural remedies, homeopathy, nutrition, biofeedback, hypnotherapy, reiki, etc.

The mechanisms of pharmacological and physical treatments target various locations of the pain response (Bennett, 2006; Bonica, 2001; Jay, 2007), starting with local anaesthesia (such as lidocaine and capsaicin), local reduction of inflammation (aspirin, ibuprofen), local reduction of muscular tension and restoration of muscular function (muscle relaxants, trigger-point injections, physiotherapy, relaxation), modulation of the pain perception mechanisms at the spinal level (transcutaneous electrical nerve stimulation, spinal cord stimulation), chemical modification of the activity of various neurotransmitters (opioids such as morphine, codeine etc. acting directly on opioid receptors; antidepressants such as amtriptyline and imipramine blocking the reuptake of norepinephrine and serotonin; anti-epileptics such as gabapentin and pregabalin, acting as gamma-aminobutyric acid analogs), local inhibition or interruption of pain transmission through specific pain pathways (e.g. intrathecal drug delivery, neurolytic blocks), to stimulation of central inhibitory mechanisms (e.g. Deep Brain Stimulation). In case of an associated condition (e.g. osteoarthritis, angina), interventions can be targeted at diminishing the progression of the chronic health condition and its effects on the nervous system. Most of the interventions are still in need of more clinical trials. Their properties in relation to specific patient characteristics are still unclear, as the responses to most of the treatments are characterised by significant variability. Therefore treatment algorithms are continuously changing based on new research, and in individual cases one often meets with a history of multiple prescription changes and interventions. Changes in legislation (especially related to prescription of opioids) add to the complexities of medication treatment (Jay, 2007).

Psychosocial interventions vary widely, and include operant-behavioral therapy (OBT), cognitive-behavioral therapy (CBT), and more recently acceptance and commitment therapy (ACT). The theoretical aspects of these three psychological interventions will be reviewed in the next section. Other approaches involve patient education, ergonomic training, biofeedback, vocational therapy and family and marital therapy.

These services are offered increasingly by multidisciplinary pain management teams, which have been shown to give the best results for various categories of patients,

compared with waiting lists, treatment as usual or other non-multidisciplinary treatments (Gatchel and Okifuji, 2006; Scascighini et al., 2008). A multidisciplinary treatment would usually mix medication, physical and psychosocial interventions and sometimes complementary therapies in various combinations depending on the patient group and the strengths of the pain team. It is not known which components of the multidisciplinary programmes most benefit which types of patients (Scascighini et al., 2008). Yet these programmes are not readily available to all sufferers due to financial reasons, and their aims are rarely about total elimination of pain but rather target symptomatic control, decrease in healthcare utilisation, increase in function and return to work (Gatchel and Okifuji, 2006). Patients however often expect a cure (particularly early in the patient's chronic pain trajectory), and the limited results are directing them to alternative services, often involving complementary therapies, which although offer relief in a certain proportion of the population, do not meet the patients' expectation either (Rosenberg et al., 2008).

The treatment difficulties add to the patients' continuous perception of pain, their feelings of social isolation, their frustration for the associated disability and treatment side effects, and many other problems related to the consequences of the condition for their personal, occupational and social life. The psychological implications of the chronic pain experience are not easy to cope with, nor are they easy to describe and conceptualise in research. These efforts are the topic of the next section.

2.9 Psychological aspects of chronic pain

The conceptualisation and treatment of chronic pain has been shaped considerably by the evolution of psychological thought. The initial development of the psychophysical approach was simultaneous with the reign of psychoanalysis. The former focused on pain as a result of external stimuli such as physical damage according to the specificity theory of pain, while the latter viewed pain as a manifestation of repressed psychological conflict. The two disciplines were thought to address types of pain that were mutually exclusive and therefore any psychological factor was considered to intervene only when pain did not have an identified organic basis. Psychoanalytic and later psychodynamic approaches had mainly a descriptive scope and did not lead to a model with significant applications in pain management, or any empirical support (Hadjistavropoulos and Craig, 2004; Asmundson and Wright, 2004)

The development of Melzack and Wall's gate theory of pain and the propagation of behaviorist approaches led to the appearance of the first psychological approach to pain with direct practical application, detailed in Fordyce's seminal book, "Behavioral methods for chronic pain and illness" (Fordyce, 1976). Fordyce's theory attempts to go beyond the previously opposed diagnostic categories of organic and psychogenic pain, and introduces a distinction between respondent and operant pain: as any other illness or health behaviour, pain is partly a response to specific antecedent stimuli (respondent) and partly subject to the positive or aversive consequences of its occurrence, either in the person's history or in the present circumstances (operant). Fordyce presents a new perspective on chronic pain in behavioral terms: as a set of chronic illness-related behaviours subject to learning and change given the right input from behaviorally-trained health care professionals and social environment. OBT interventions address the operant pain that occurs as a by-product of the underlying disease or trauma. Three areas of possible change are identified: direct and positive reinforcement of pain, indirect positive reinforcement of pain by avoidance of aversive consequences, and failure of positively reinforcing well behaviour. Fordyce analyses each separately and offers structured programmes of intervention; this work represents the beginning of pain management as it is practised today and methods such as working to quota, pacing, working with the patient's family are still applied to a large extent as they were first conceived, often under the term of "contingency management" (Bonica, 2001, Ch. 88).

The behaviorist approach is a significant progress compared to the psychoanalytic theory, as it minimises the importance of untestable personality factors in chronic pain and highlights the role of environmental factors, paving the way to a multifaceted understanding of pain. Moreover, Fordyce dismisses the explanatory power of the organic-psychogenic distinction and any use of a personality-related variable as a cause of pain behaviour. The following quote summarises clearly Fordyce's position and also highlights the difficulties in measuring any constructs related to pain and in studying their interaction:

> the essence of the problem lies in assuming that there are real mental and physical events which can and do interact. In fact, there are simply phenomena which we describe in physical language or mental language; we delude ourselves to believe that because we can impose both mental and physical concepts on such an abstraction as 'pain', that, in fact, such a causative sequence exists (Sternbach and Fordyce, 1975, as cited by Fordyce, 1976, p. 28)

Critics of the operant-behavioral view highlight the fact that, in practice, specific behaviours and contingencies are not invariably desirable or negative, and the relations between them are rarely simple. It is difficult and even ethically questionable to intervene by withholding emotional and financial support without the full cooperation of the sufferer. And it is unrealistic to consider all the network of contingencies in the sufferer's environment outside clinical settings (Hadjistavropoulos and Williams, 2004). Moreover, between the external stimuli and the person's behaviour, there are numerous mediating psychosocial factors, such as attitudes, beliefs, expectations, emotions (Skevington, 1995, p. 87), even if these prove particularly difficult to measure and study. For example, a sustainable increase in exercising habits cannot be achieved only by making changes in the sufferer's environment; his/her thoughts and feelings about how exercising will help might also need to be addressed. The acknowledgement of the inadequacy of the behaviorist approach to internal events and the change of clinical and research focus towards these factors led to the development of the CBT approach to chronic pain.

CBT uses structured techniques to teach patients to identify, monitor and change maladaptive thoughts and feelings related to pain and behaviour. The patient needs to actively participate in learning skills that help them manage their condition, such as relaxation, imagery and coping self-statements (Bonica, 2001, Ch. 89). The initial formulation focused more on cognitive aspects and specific skills, while the integration of affective factors is a more recent effort. At present, CBT programs integrate a broad variety of techniques, including operant methods, patient education, fitness training, problem-solving, stress management, etc. (Hadjistavropoulos and Williams, 2004). This change of focus from immediate observable behaviour to the patient's inner experience of the world led to some extent to a welcomed change in treatment outcomes from immediate behavioral changes to slower, but more stable improvements via cognitive change (Turner and Clancy, 1988)[4].

In practice though, both OBT and CBT have some major drawbacks sourcing from the control-based approach to behaviour change, to which Acceptance and Commitment Therapy (ACT) offers a solution. Dahl et al. (2005, p. vii) describe the frequent motivational issues this approach generates. The pain management team generate therapeutic agendas based on a functional analysis of the specific activities the patients' avoid and the maladaptive thoughts they hold. These agendas might not match the patients' own views and plans, and without their full involvement it

[4]The application of cognitive psychology and CBT in chronic pain will be reviewed in more detail in Chapter 5.

is difficult to obtain improvement especially via a time-limited intervention. The issue of self-motivation is made more salient by the use of techniques such as exposure and thought reconstruction, which require patients to expose themselves to more pain and in a sense acknowledge that their current way of thinking is wrong. As Kratz et al. (2007, p. 299) explain, "approaching a recalcitrant problem like chronic pain with control-based strategies alone may leave the chronic pain sufferer vulnerable to frustration, demoralization, and endless preoccupation with reducing pain. The addition of pain acceptance may offer the potential for improved quality of life by filling the often substantial gaps in pain management left by control-based strategies.".

The application of ACT to chronic pain aims to fill this gap by a distinct approach, summarised by Dahl et al. (2005, 2004) via three main characteristics. First, the agenda is built by the patient through value clarification. ACT aims at reorganising the values system of the person in order to motivate therapeutic change instead of persistence in avoidance behaviours. Second, although experiential avoidance remains a core pathological process, in ACT it refers not only to situations and movements, but also to the unwillingness to remain in contact with pain-related experiences (sensations, thoughts, emotions) and efforts to change or reduce these. Thus, the exposure in ACT is oriented towards the thoughts and sensations themselves as part of the situation[5]. Third, the thoughts are not challenged, as in CBT, the person's reaction to them is the target of the therapeutic change: avoidance reactions are to be replaced with a mindful and accepting attitude. Several studies indicate ACT-based interventions show promise when compared to OBT or CBT-type interventions (Dahl et al., 2004; McCracken et al., 2005, 2007a; Vowles et al., 2009; Wicksell et al., 2007, 2008a, 2009), although critics of the ACT approach question the theoretical basis and the technical novelty of the method (e.g. Ellis, 2005).

Pain management methods have developed concurrently with theoretical efforts to identify concepts and measurement tools that would support the search for causal mechanisms and tests of treatment efficiency. Fordyce's model remained faithful to the simple behaviourist framework of positive and negative reinforcement of pain behaviours measured by observational methods within clinical diagnosis

[5]In contrast to OBT, the functional analysis in ACT acknowledges the presence and influence of internal events, but views them as part of the behavioural context.

and treatment (Hadjistavropoulos and Williams, 2004). In contrast, the cognitive-behaviourist approach has generated a richness of constructs and models in the attempt to operationalize its main domains: cognition and affect.

The development of ACT for chronic pain led to the concept of chronic pain acceptance. A detailed review of its theoretical basis and its relationship to pain will be the topic of Chapter 3. The search for a better way to measure emotion led from generic terms such as depression or stress to constructs such as fear avoidance, anxiety sensitivity, anger expression. The search for a better operationalization of cognition led to the development of constructs such as pain-related beliefs and expectations, coping, catastrophizing, illness perceptions. Detailed views of the attempts in each of these two areas are described in Chapters 4 and 5. Obviously, these three main domains do not function as separate factors in chronic pain. There are conceptual overlaps and complex interactions between them. Relationship patterns might also differ depending on other psychosocial factors such as demographics and various characteristics of the chronic pain condition or of the environment. These will be detailed in Chapter 6, as a first attempt to integrate the three perspectives on psychological adjustment to chronic pain.

Chapter 3

Acceptance

3.1 Introduction

The ACT approach to chronic pain, as briefly stated in Chapter 2, focuses on behaviour. But in contrast to the OBT approach, it is not only observable, external behaviour that is targeted, but also internal, mental behaviour. Thus, its main concepts, psychological flexibility (PF) and chronic pain acceptance (CPA), include in their definition phenomena otherwise described as part of cognition or emotion. This overlap would suggest a solid basis for integrating behaviour, emotion and cognition. However, ACT's distinct approach to scientific investigation represents a sizeable obstacle for an integrative attempt.

This chapter starts with a review of the theory behind CPA and the difficulties of testing it from a mainstream scientific perspective. It then attempts to clarify the concept of acceptance and to describe its application in other health-related areas. The research and measurement of acceptance in chronic pain are next reviewed. The chapter ends with several proposals for future research and several caveats regarding the interpretation of research results in a chronic pain context, in light of the theory.

3.2 The theory

To get a better understanding of the concept of acceptance in chronic pain, one needs to situate it in the wider perspective of functional contextualism (FC) and behaviour analysis (BA), on which ACT is based. Understanding the theory is especially important in the context of comparing the three frameworks (acceptance, emotions and illness perceptions) as applied to chronic pain and the exploratory efforts to integrate them. In the previous chapter I have mentioned three changes that ACT

introduces to the practice of chronic pain management: value clarification, exposure to avoided mental content, and mindfulness learning. I will next describe the theory developed to bind these methods together and then discuss the meaning that it projects on the construct of acceptance.

ACT is a therapeutic method with a wide application in clinical and health psychology. It was developed by Steven C. Hayes and colleagues in recent decades in an attempt to overcome the limitations of cognitive-behavioural therapy. It borrows methods from various sources: the human potential movement (gestalt, experiential, client-centered), behaviour and cognitive-behavioural therapy, various Eastern and Western mystical traditions. Technically (i.e. from the point of view of the therapeutic tools), it is an eclectic approach to therapy. However its authors add to it the effort of integrating these methods in a detailed multi-layered system: it is a clinical theory based on a psychological theory located in a distinct philosophical approach to science (Hayes et al., 1999b, p. 16).

At the philosophical level, they locate ACT within FC: an approach to science based on pragmatism and context. It opposes mechanistic approaches to science which assume that reality is organised into discoverable parts and its real structure is stable and generalisable irrespective of the researcher's intentions and situation. By contrast, FC assumes that "what is true is what works" in a particular situation depending on the set goals of the scientist (Hayes et al., 1999b, p. 20). It strives to understand all phenomena as "acts-in-context" and organise them in functional units depending on the situational goals of analysis. As the general goal of FC is to predict and influence behaviour (the world)[1], direct causes of behaviour need to be external to the behaviour and manipulable (at least in principle): "all analysis must trace phenomena back to the environmental context, both historically and situationally" (Hayes et al., 1999b, p. 23).

At the psychological level, Relational Frame Theory (RFT) further develops the idea of contextual relationships. "The basic premise of RFT is that human behaviour is governed largely through networks of mutual relations called relational frames. These relations form the core of human language and cognition, and allow us to learn without requiring direct experience."(Hayes and Smith, 2005, p. 17).

As opposed to animals for which learning is mainly based on classical and operant conditioning (in other words by direct interaction with the environment), human

[1] "Functional contextualists have an intensely practical goal for analysis: the prediction and influence of events as an integrated goal" (Hayes et al., 1999b, p. 22).

cognitive abilities extend further to include these networks. A relational frame is defined as "a specific class of arbitrarily applicable relational responding that shows the contextually controlled qualities of mutual entailment, combinatorial mutual entailment, and transformation of stimulus functions" (Hayes et al., 2001, p. 33). They are characterised as 'frames' and 'arbitrarily applicable' because they can be applied to any events and can be unrelated to the physical properties of the stimuli. They are 'relational' as they associate events. Context control reflects the necessity of a given situation in which the frames apply. The three properties refer to bidirectionality of the associations (mutual entailment), which can be applied to relations between multiple stimuli (can be combinatorial) and lead to changes in any particular stimulus depending on the relations it develops with other stimuli (transformations of stimulus functions). As it is not a central theory for chronic pain, but a separate behaviorist account of cognition on which ACT is developed, RFT will not be further detailed. Suffice it to say that these processes are considered to enable humans to achieve a more extensive control of the environment, but to have drawbacks in the domain of internal events (Hayes and Smith, 2005, p. 17).

RFT uses terms such as human language, cognition, knowing (based on language), mind, minding and verbal event as equivalent terms. These denote a process that is more than human vocalisation or social communication: "we mean symbolic activity in whatever domain it occurs (gestures, pictures, written forms, sounds, and so on)" (Hayes et al., 1999b, p. 10). A variety of mental processes such as "evaluating, categorizing, planning, reasoning, comparing, referring, and so on" (Hayes et al., 1999b, p. 49) are included in this definition. The distinction verbal - nonverbal is not to be confused with the common usage of the term: "Gestures, signs and pictures are 'verbal' if their effects depend on their participation in relational frames, but they are 'nonverbal' if that is not true" (Hayes et al., 1999b, p. 42).

The characterisation of symbolic activity as both beneficial and harmful is framed in evolutionary terms, as evolving for purposes of social control and danger signalling (Hayes et al., 1999b, p. 71). Benefits are reaped when dealing with the external world, while harm is inflicted when applying it to internal events. "Humans are tremendously advantaged by this ability. We can create physical stability and comfort by interacting cognitively with the world. We can verbally construct dangers, needs, and futures and take action based on these formulations. But we can also struggle for no reason and hold on when we should let go" (Hayes et al., 1999b, p. 69)

At the clinical level, ACT places symbolic behaviour at the centre of its theoretical construction: "most forms of psychopathology and human suffering are verbal behavior gone awry" (Hayes et al., 1999b, p. 51). In contrast with CBT which distinguishes between adaptive versus maladaptive thoughts, ACT targets the process itself as a source of pathology: "It is not that people are thinking the wrong thing - the problem is thought itself and how the verbal community supports its excessive use as a mode of behavioral regulation" (Hayes et al., 1999b, p. 24).

In earlier formulations, ACT summarises the problem in four words: fusion, evaluation, avoidance and reasons (FEAR). Fusion characterises symbolic activity in situations where the symbol, the event it describes, and the person experiencing it are seen as identical (fused together): for example, the word "depressed", depression as an internal event (or crying as an external event) and the person living it are in an inflexible self-perpetuating relational frame. The existence of such a relational frame allows attribution of valence functions to events (evaluation) according to conventions. The negative valenced events are then avoided in order to sustain the fusion with opposite positively valenced events and symbols. Reason giving further enhances such inflexible relational frames by construing internal events (and their symbols) as causes of overt behavior, thus encouraging the fusion and the avoidance behaviours (Hayes et al., 1999b, pp. 72–77).

More recent formulations (Hayes et al., 2006; Blackledge and Barnes-Holmes, 2009) further develop this model into six core processes centred around the concept of psychological inflexibility versus flexibility, defined as "the ability to contact the present moment more fully as a conscious human being, and to either change or persist when doing so serves values ends" (Hayes et al., 2004b, p. 5). Three of them are categorised as mindfulness and acceptance processes: acceptance (as opposed to experiential avoidance), cognitive defusion (versus fusion) and being present (opposed to attachment to the conceptualized self). The other three are commitment and behaviour change processes: contact with the present moment (versus dominance of the conceptualized past and feared future; weak self-knowledge), valued living (versus lack of value clarity/dominance of pliance and avoidant tracking) and committed action (versus inaction, impulsivity and avoidant persistence). These processes are rather ways of organising the intervention goals and techniques, than distinct phenomena amenable to individual measurement.

ACT aims to change not the content, but the function of symbolic behaviour by manipulating the context that determines their relations with overt behaviour (Hayes et al., 1999b, p. 24). For this purpose they employ a variety of techniques that are

claimed to target the above processes, sometimes described as ACT: accept, choose, take action.

The client is exposed to the realisation that previous attempts of control have brought only temporary relief and worked against him/her in the long term, by amplifying and adding to the initial problem. The resulting state of creative hopelessness makes the client open to a new approach: observing mental processes without acting upon them. Mindfulness, willingness or acceptance are used interchangeably to describe this state.

This state of observation is accompanied by "establishing contexts in which the distinction between derived and direct stimulus functions is more experientially evident, and in which verbal stimuli have multiple effects, only some of which are derived" (Hayes and Smith, 2005, p. 150). In other words, exposing the clients to situations in which they could perceive their problem from different perspectives, and thus be able to reduce its influence over their behaviour. The process is called defusion, or deliteralisation and is aimed at reducing the inflexibility of relational framing by creating a context in which thoughts have a reduced control over behaviour, for example by focusing on the real or imagined physical properties of a thought: its sound, its colour, its size, etc. They trace defusion back to the CBT concept of distancing. However while distancing is only a first step towards evaluating and disputing thoughts, defusion means noticing without acting upon them; not even judging them as good or bad, as attributing valence contributes to the problem, as explained above (Hayes and Smith, 2005, p. 53). Via defusion, the role of thoughts in regulating behaviour and emotion is undermined, rather than reinforced.

Another barrier to behavioural flexibility is an exaggerated attachment to a rigid set of verbal rules about the self, which ACT labels 'the conceptualized self'. Diminishing the influence of these rules reduces the client's involvement in the war between verbal polarities that threatens his/her sense of stability. Reestablishing flexibility involves supporting the development of a style of self-relating more sensitive to 'here-and-now' contextual influences (labelled 'ongoing self-awareness' and 'self-as-observer'). Focusing on the self-as-observer, which is described as the unchanging perspective or "watcher" of all personal experience, reestablishes this sense of stability, while focusing on the awareness of the changing momentary experience widens the opportunities for change and also for observing previously avoided mental content.

The above methods target the rigidity of the problematic behaviour pattern. But for real change to take place, initiation of new behaviour patterns need to be guided by something situated beyond verbal processes. ACT proposes that values are above evaluation and judgement. Values are life directions that need to be clarified or defined and then chosen, and it is assumed that a person already has the ability to do so. Value clarification enables the client to select appropriate goals and commit to the relevant actions. Commitment is not towards goal attainment per se, but towards the value as a direction which guides the selected goals. Thus, failure to reach a goal is only an opportunity to regroup and continue the never-ending journey. "Values are vitalizing, uplifting and empowering. They are not another mental club to beat yourself with or another measurement to fail against [...] Values are chosen life directions." (Hayes and Smith, 2005, p. 155). In ACT, commitment is also not a guarantee that verbal events will not take place; committed, value-based action only prevents rigidity of relational frames to escalate, and is described as contrasting with the vicious cycle of evaluation, avoidance, reason giving, fusion.

In my opinion, ACT brings several positive changes to mental health practice. For example, it brings a welcomed focus on changing standards regarding normality versus mental illness, and thus concurs with the increasing understanding of the positive aspects of interpersonal variability in the mainstream culture. Instead of striving to conform to a increasingly limiting socially defined ideal, the celebration of life and experience in all its forms is a liberating idea in itself. The therapeutic effect of legitimating the presence of all mental content is acknowledged in many therapeutic traditions. Value clarification is another needed addition. The access to intrinsic motivation (even if it is, essentially, still based on assimilated cultural norms) is considered as one important source of healthy psychological and social development and it is diminished by negative performance feedback, which supports the increased success of an accepting approach compared to challenging of maladaptive thoughts (e.g. Ryan and Deci, 2000). The focus on distancing is another valuable ingredient. Putting life into perspective and considering a wider variety of options and influences than one's usual routine can be in itself a much needed breath of fresh air. And whether one does it by repeating a phrase many times, by humour, by contemplating a paradox or by an open discussion with someone with a different perspective, it is a good opportunity for change, and can be considered in essence a problem-solving strategy. These three examples are only a few subjective considerations, and certainly the therapeutic practice may highlight many more. In research though, the flexibility of ACT leads to some limitations, described in the next section.

3.3 Difficulties of testing ACT from the perspective of the scientific method

The elaboration of RFT as a psychological model of cognition and of FC as a distinct approach to philosophy of science can be considered a strength of the therapeutic approach of ACT. However, the fact that FC is described as incompatible with the mainstream approach to scientific investigation represents in my opinion a considerable barrier to theory testing, especially in the context of an effort to integrate ACT concepts with other approaches, as in the present thesis[2]. A clarification of these incompatibilities is therefore necessary.

Hayes et al. (2001, p. 4–7) made a distinction between the mainstream approach, viewed as mechanistic (concerned with reducing a phenomenon to parts, relations and forces) and mentalistic (considering internal events as causes), and functional contextualism, which focuses on the pragmatic value of the analysis and the contextual determination of the phenomenon studied[3]. They stated that, while contextualists criticise mechanists for splitting phenomena into components and reifying them, the main mechanist argument against contextualism is vagueness and imprecision generated by the focus on situational variability (Hayes et al., 1988). Certainly, what is generally labelled 'mainstream science' or 'the scientific method' is not a methodological monolith, but rather a collection of methods of investigation which share a certain rigour (Sankey, 2008). Nevetheless, by positioning itself explicitly in opposition to "elemental realism" (Hayes, 2009), ACT is exposed to methodological inconsistency. As Hayes (1993, p. 24) states,

> The weakness of functional contextualism is that its methods threaten its root metaphor. Contextualists can borrow mechanistic methods in the services of their goals, but they can in turn be swayed by the implicit values of these methods and become mechanists. Accomplishing practical outcomes requires a division of the whole into parts. Most especially, if one becomes interested in behavioral influence, one must distinguish between events that are – at least in principle – manipulable and those that are not.

[2]The consequences of this stated philosophical incompatibility for an integrative theory will be further discussed in Section 6.3.

[3]This distinction is based on an earlier categorisation of philosophical systems into four major models, or "world hypotheses": formism, mechanism, organicism and contextualism (Pepper, 1942, as cited in Hayes et al., 1988).

The focus on context and function-related variability leads to two practical problems in theory testing, one related to measurement, the other to identifying structural (or functional) relations. In concept description ACT uses a distinct paradoxical language to highlight contextual variability, which makes the operationalisation difficult. Moreover, the mechanisms by which behaviour is determined by contextual influences (external and internal events) are also described as variable without clear specifications of predicted patterns, limiting the testability of the theory. These aspects might not have a dramatic impact on therapeutic practice, where the context, methods and relationship are more salient, and language use is flexible and subordinated to the principle of workability. In research however they do pose problems. By adopting "mechanistic methods" in investigating ACT, the researcher must also meet the requirements of these methods (e.g. unidimensionality and homogeneity of causal processes in structural equation modeling; see Chapter 6). The following section is a critique of ACT from the perspective of the research methods used.

3.3.1 Contextual variability in concept definition

In ACT, the analysis of behaviour patterns is based on workability, which is an essential element of keeping one's behaviour in line with the chosen life directions. Behaviour change does not take place by replacing one set of behaviours with another, less 'maladaptive', set, but by the process of changing their functioning from inflexibility to flexibility. Thus, no specific observable or internal behaviour (thought or emotion) can be characterised as consistently related to a specific concept. In communicating ACT, the authors use metaphor and paradox to convey this message, while the therapeutic methods rely on experiential techniques to induce change. The words and definitions used are therefore considered only means to an end, not fixed or general descriptions of a assumed reality.

The authors state that the use of metaphors and paradox is more suitable to ACT and represents a solution to the problematic situation in which "attempts by both the writers and readers of this book to understand destructive verbal processes will themselves be based on verbal processes" (Hayes et al., 1999b, p. 12). The following paragraph eloquently reflects this approach:

> Don't believe a word in this book. It is one of the burdens of ACT that if the model is correct, then the model must be held lightly.[...] Act is not a dogma, but it is not a nondogma either. The confusion and incoherence in this paragraph is deliberate, not because we are trying

> to confuse the reader, but because language is fundamentally incapable of going beyond itself except in the experiential glimpses provided by paradox and confusion" (Hayes et al., 1999b, p. 281)

A few examples of context-dependency might clarify this general point. Exerting control over events is considered beneficial or detrimental, depending on the type of event targeted (e.g. external or internal; Hayes and Smith, 2005, p. 53[4]. Rigidity as induced by rule-governed behaviour is sometimes helpful (e.g. in committed action), an often it is not (e.g. when resulting in avoidant behaviours; Hayes et al., 1999b, p. 29, 237). Both flexibility and consistency are recommended (Hayes and Smith, 2005, p. 189), but only general criteria are given (such as changeability, or adequacy in relation to chosen values), leaving the reader (therapist, or patient) the freedom to decide how to apply them flexibly in their specific situation. Any situation can have multiple contrasting interpretations, as what is changeable and adequate is often a matter of negotiation between the parties involved.

In practice, it is up to the therapist and patient to apply these criteria adequately and ACT certainly recommends a nuanced and context-related interpretation of these issues. In research however this flexible view unfortunately translates into lack of clarity, which poses significant problems in operationalising central concepts such as psychological flexibility, acceptance, avoidance, etc. in self-report measures. For example, an item referring to efforts to control one's life can be answered positively by both an avoidant person and an accepting person, depending on the contextual interpretation. How would such items differentiate reliably between the two?

The therapeutic relation offers the chance to negotiate and agree upon a most suitable interpretation. But as questionnaire items do not offer the option of lengthy clarifications, measuring these concepts by self-report is prone to error. Of course, the issue of measurement error is not limited to ACT, it extends to the whole field of psychological and social sciences, and the role of context has been often described as an enduring dilemma for psychological measurement and theory testing (Meehl, 1978). The differences between ACT and other conceptual frameworks is the inclusion of contextual variability as a fundamental component of the theory, the acknowledgement of the limitations of language and thought to communicate about RFT and ACT, and the rejection of the 'mainstream' method of searching

[4]It can be argued that the description of acceptance in a self-help book (Hayes and Smith, 2005) is not adequate for the present argument, as it is not an example of technical language but of every day language. However, these descriptions are particularly important for acceptance measurement via self-report, as questionnaire items involve a translation between these two registers.

for distinct elements of a phenomenon and the stable relationships between them. These aspects raise doubts about the reliability of self-report in relation to ACT processes and bring forth the fundamental question of whether using measurement in testing ACT empirically at a higher level of generalisation is actually consistent with the philosophical approach of ACT.

Without trying to answer such a fundamental theoretical question, it is important to highlight that, in practice, ACT does attempt to define psychological flexibility as consisting of six overlapping processes, and specific measurement tools for the six core processes have also started to be developed (e.g. of acceptance and cognitive fusion in pain; Wicksell et al., 2008c). However, the contextual focus of ACT does not agree with predicting clear distinctions between these processes, or stable functional relationships between them, which makes theory testing difficult, as explained next.

3.3.2 Contextual variability in theory testing

ACT's contextual approach to concept description extends to the process of theory formulation and testing. Statements of causal relationships can be found in the literature, for example concerning cognitive entanglement, negative self-referential evaluations and negative evaluations of private experiences leading to unproductive attempts to regulate private experience (such as suppression of thoughts and emotions), which in turn lead to inability to take action (and immediate reduction of avoided content, followed by long term increase of such content; Hayes et al., 2004a, p. 555). However these statements are rather guidelines for therapeutic practice than predictions of stable relationships between distinct elements, and are difficult to test statistically as general structural relations, due to the contextual variability and lack of conceptual distinctiveness stipulated by the functional contextualist approach of ACT.

Moreover, ACT states that, according to FC, thoughts and feelings, as all other public or private events, cannot cause the behaviour of the same person, as they are not external to the behaviour, but can however participate in overall causal relationships (Hayes et al., 1999b, pp. 55–6). This statement cannot be translated unambiguously into models of structural relationships at an intrapersonal level. If taken literally in a behaviorist framework, ACT states that there are no causal connections between any internal events, therefore a structural equation model would

only relate to the theory if it measures external factors as causal variables and behaviour as outcome variable, possibly with a moderating role of internal events.

The theoretical emphasis on the role of context is in my opinion not sufficiently harmonised with current theory testing, which relies heavily on results of quantitative studies and gives a central role to measurement and statistics. Hayes et al. (2006) argued for the effectiveness of ACT practice mainly based on correlational studies, research on the impact of ACT techniques and on change processes, most of the using measures such as the Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004a) or comparing ACT processes with various other constructs related to physical and psychological health and functioning. While experimental studies and randomised controlled trials can be considered theoretically closer to a context-dependent application of ACT principles (although they also involve dividing the phenomenon into component parts), correlational studies that focus on identifying general relationships between multiple variables related to ACT, alternative theories and relevant outcomes address a higher level of generalisation. This analysis at a general and conceptual level, while certainly offering a valuable addition to the more applied studies, is more accurately described in terms of the 'mainstream' search for behavioural regularities and less in tune with strict functional contextualism.

The role of context would require a clearer description in relation to theory testing. The meaning of the term "context" in ACT is closely connected to the concept of "relational framing" (see Section 3.2) and therefore refers to the functional associations between events (internal and external), both historically and situationally. However in research this contextual dependency would need to be translated into more specific predictions or methodological choices. Contextual dependency might refer to causal heterogeneity: the structure of causal relationships is potentially changing depending on environmental and personal characteristics. This would request the formulation of multiple local models in which to include variables such as "family support", "work conditions", "prior medical history" etc.[5] Or it could be a way of recommending qualitative methodology and de-emphasize the use of quantitative research, which relies at least on some degree of generalizability of measurement and causal relationships, as stated above. The lack of specific methodological recommendations encumbers theory testing with the available scientific tools.

[5]Such variables are usually considered as contextual in a broader sense, therefore the term context will also be used in the following chapters to refer to demographics and other social or environmental influences.

The efforts of ACT researchers to inform practice based on quantitative studies could be considered as indicating their commitment to theory testing, even if this objective is subordinated to the wider goal of events prediction and influence, as stated in Section 3.2[6]. In order to pursue this objective further, I consider that a more clear and testable formulation at the level of concepts and their relationships would be necessary both for guiding practical applications and for making ACT more amenable to comparisons with other research areas in the same applied domains.

3.4 The concept

The difficulties detailed above make 'acceptance' a difficult concept to measure and research. In ACT, acceptance sometimes is considered a synonym of mindfulness and willingness and sometimes includes all other processes: defusion, mindfulness, self-as-observer and active value-based behaviour.

> "The ACT model itself suggests that acceptance can involve many different psychological components, including cognitive defusion, choice, abandonment of a control agenda, exposure, and active willingness and commitment. It is not known whether acceptance functions differently with or without any of these elements" (Hayes et al., 1999b, p. 282)

Acceptance is viewed as an antonym of experiential avoidance (EA), a central concept to the ACT model of psychopathology. EA is defined as "a phenomenon that occurs when a person is unwilling to remain in contact with particular private experience (e.g. bodily sensations, emotions, thoughts, memories, images, behavioral predispositions) and takes steps to alter the form or frequency of these experiences or the contexts that occasion them, even when these forms of avoidance cause behavioral harm" (Hayes et al., 2004a, p. 554). The authors source EA in most systems of therapy: behavioural, client-centered, gestalt, existential, as emotional or cognitive avoidance (Hayes et al., 1999b, p. 58). They provide research results from the fields of thought and emotional suppression and coping styles literature to support their statement about destructive effects of experiential avoidance. The paradoxical effects of thought or emotion suppression and the negative outcomes related to the

[6]For example, Hayes and Shenk (2004) advocate concept clarifications and theory improvement as opposed to attachment to specific techniques from religious practices or earlier theories, when commenting on the issue of mindfulness and meditation.

use of emotion-focused and avoidant coping strategies is considered related to experiential avoidance as forms of behavioural control (Hayes et al., 1999b, pp. 60-1). Therefore it can be stated that EA behaviourally is a synonym of these concepts, and measures of one can be an indicator of the other, while reversed scores can indicate the degree of acceptance. No specific differences are stated.

Acceptance is also opposite to effortful control. Defining acceptance by exclusion has multiple anchor-points: it is not wanting, you cannot do it half-heartedly, although you can set some appropriate conditions to it, it is not trying, it is not a matter of belief, it cannot be self-deceptive or manipulative. "Willingness is not resisting your pain, ignoring your pain, forgetting your pain, buying your pain, doing what the pain says, not doing what the pain says, believing your pain, not believing your pain" (Hayes and Smith, 2005, p. 125). These paradoxical clarifications and the use of metaphors (e.g. "holding your pain as you would hold a delicate flower in your hand" in Hayes and Smith, 2005, p. 125) are not very helpful for delimiting the connotations of the concept from a measurement perspective, although they could be useful in a therapeutic context for catalysing a change of perspective.

With all these conceptual clarifications, acceptance as used in ACT seems an elusive idea. The authors state that the meaning of active forms of acceptance is hard to describe (Hayes and Smith, 2005) and that it "is tricky because it's an action that humans can learn but minds cannot" (Hayes and Smith, 2005, p. 125). This has obvious implications for self-report measures of acceptance. A person that answers 'yes' to a question like "Anxiety is bad" or "It's ok to experience pain" might be usually behaving in ways that we think are usually grouped in the category/continuum we name acceptance, or might adhere only verbally to this attitude, while behaving in an avoidant manner. A certain degree of overlap between behavior and verbal report is likely to exist in any self-report measure, as is a degree of measurement error. However the difficulty to assess this extent of this error is increased in concepts with less clear operationalisations.

Another issue that makes acceptance a difficult concept to define and operationalize is the multiple contradictory meanings that the word has in common use:

> Acceptance, in the sense it is used here, is not nihilistic self-defeat; neither it is tolerating and putting up with your pain. It is very, very different than that. Those heavy, sad, dark forms of "acceptance" are almost the exact opposite of the active, vital embrace of the moment that we mean (Hayes and Smith, 2005, p. 7)

Recent theoretical formulations replaced acceptance (versus experiential avoidance) with "psychological flexibility" (versus inflexibility). By giving-up control and adopting this open-minded attitude to experience, one opens up to an entire new world of experience in which both suffering and satisfaction are possible and meaningful. The main goal is behavioural flexibility which gives one the freedom to pursue the chosen values (Hayes and Smith, 2005, p. 125). The term acceptance now labels only one of the six core processes in ACT (see Section 3.2).

A recent investigation of the construct validity of acceptance (Kollman et al., 2009) points out the multidimensional nature of the concept. In an effort to obtain a unidimensional measure, they exclude other aspects such as present-focused attention, openness, valued action, etc. Their operational definition of acceptance is "the active following of internal events (e.g., feelings, thoughts, memories and physiological reactions) without 'taking steps to alter the form and frequency of these experiences'" (p. 206). They compare it with "cognitive reappraisal" and "perceived control over emotional reactions" on a sample of anxiety or mood disorder patients, and conclude that this limited operationalisation of acceptance is significantly different from these two other processes, although it is not related to relevant outcome measures such as worry, social interaction anxiety and well-being aspects. The lack of clinical validity is explained by the limited definition: "actively allowing internal events" might not be effective by itself, but in combination with the other processes (additively or interactively), or it might participate in more complex temporal or contextual relationships that ensure its clinical effectiveness. The commonly used acceptance measures, such as Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004a) pay for their clinical utility with a less exact concept definition. Kollman et al.'s (2009) study speaks eloquently of the difficulties in measuring and researching acceptance, and especially in matching the mainstream scientific approach with FC.

3.5 Acceptance and health

In chronic illness, acceptance has been mostly researched in relation to coping, as one of the many coping strategies available or as an alternative to coping. In a review of the theoretical frameworks on adjustment to chronic illness as applied to rheumatoid arthritis, Walker et al. (2004) placed acceptance among the psychological models, in close connection with stress and coping and the role of stable characteristics such as neuroticism and optimism, and in opposition to biomedical and biopsychosocial models. Acceptance was described as a cognitive process that

participates in making sense of the illness experience, gaining control and restoring self-esteem (according to Taylor, 1983, as cited by Walker et al., 2004). To support their interpretation of acceptance, the authors cited previous studies linking acceptance with increased sense of control, lower psychological distress and better psychological adjustment, but also mentioned research reporting associations between acceptance and increased anxiety and depression, and lower well-being. Acceptance was also described as a synonym of positive yielding control in a study of adjustment to breast cancer (Astin et al., 1999). These operationalisations of acceptance are different from its meaning in ACT and RFT, which consider themselves contextual and not mentalistic (see Section 3.2).

Acceptance as described in ACT has recently been applied in several health-related research areas (in addition to applications in mental health): diabetes (Gregg et al., 2007), epilepsy (Lundgren et al., 2008), substance abuse (Ostafin and Marlatt, 2007; Luoma et al., 2008; Heffner and Parker, 2003), insomnia (Lundh, 2005), weight control (Lillis and Hayes, 2007), anorexia nervosa (Heffner and Eifert, 2002), HIV (Batten and Hayes, 1997). While some of these applications are involved strictly with the theory and practice of the therapeutic method, some also involve research and measurement and in this case either use general measures of acceptance (AAQ; Hayes et al., 2004a) or mindfulness, or versions of the AAQ adapted for the specific condition.

A detailed comparison of different operationalizations of acceptance in health literature is beyond the purpose of this study. Suffice it to say that given the wide variety of interpretations, the meanings attached to the concept via a specific measure deserve special attention. I will next focus on clarifying these meanings in relation to chronic pain.

3.6 Chronic pain acceptance

Even if in chronic illness acceptance has also been used with no relation to ACT, in chronic pain the concept has been developed based on ACT and research is related to improving acceptance-based pain management programmes. The behaviorist roots of acceptance fit well theoretically and practically with the strong behaviorist tradition of chronic pain management, and with the successful application of fear-avoidance models in this area (reviewed in Chapter 4).

3.6.1 Measurement and research

Chronic pain acceptance (CPA) was initially defined as: "acknowledging that one has pain, giving up unproductive attempts to control pain, acting as if pain does not necessarily imply disability, and being able to commit one's efforts toward living a satisfying life despite pain" (McCracken, 1998, p. 22).

The Chronic Pain Acceptance Questionnaire (CPAQ; McCracken, 1998) is the measure of choice in the field. A first version was developed based on a pool of 34 items describing different aspects of acceptance (from AAQ), with a 7-point Likert response format (from 'never true' to 'always true'). Item selection was based on item-total, inter-item and reliability statistics; the resulting 24-item version had good internal consistency (Cronbach's $\alpha = .85$; Geisser, 1992, in McCracken, 1998).

McCracken (1998) reported good item distributions, item-total correlations and internal consistency ($\alpha = .84$), lack of correlation with gender, age and chronicity of pain (probably representing time since pain onset). Acceptance scores were also significantly related to pain-related anxiety, avoidance, depression, physical and psychosocial disability, uptime and work status when controlling for related demographics and pain intensity (as measured by VAS). Correlation with pain intensity was relatively low ($r = -.28$). It was acknowledged that this data supported the construct validity of chronic pain acceptance, although shared method variance, self-report related biases, the preliminary status of CPAQ development and the cross-sectional design recommended a cautious interpretation. An alternative interpretation was considered: "those who suffer with less severe pain, disability, and distress may find their pain more acceptable as a result" (p. 26).

McCracken et al. (1999) compared patients classified as dysfunctional, interpersonally distressed and adaptive copers based on WHYMPI scores (Turk and Rudy, 1988) and found significant group differences on pain-related anxiety and acceptance, when controlling for pain intensity and depression. These two pain-related concepts significantly participated to correct classification of 72.5% of dysfunctional sufferers and 90.9% of adaptive copers (none of the interpersonally distressed sufferers were asigned correctly[7]). As WHYMPI scores include similar concepts (such as perceived life control, affective distress), these results also reflect the similarity of instruments and do not represent a strong test of the theory.

[7]The authors mention two possible reasons: interpersonally distressed sufferers might be differentiated by characteristics of their social environment rather than their own responses to pain, and the small numbers in this group might have biased the classification procedure

Initial factor analyses of the CPAQ 34 item pool (McCracken, 1999) suggested a 3-component structure: Life Focus ("engaging in normal life activities"), Acceptance of Chronicity ("recognising the pain might not change") and Avoid/Control ("needing to avoid or control pain")[8]. Another component, Cognitive Control ("Believing that controlling thoughts controls pain"), was eliminated due to low correlations with the other factors and lack of fit with the concept definition (suggesting that cognitive control is not related to acceptance). The remaining factors had good internal consistency. The author stated that the limitations of relying on the original pool of items, and the lack of cross-validation on independent samples recommended further studies.

McCracken and Eccleston (2003) compared acceptance (CPAQ) with coping (measured by the Coping Strategies Questionnaire; CSQ, Rosenstiel and Keefe, 1983[9]) in predicting distress and disability in chronic pain patients. Acceptance was associated with better work status, more daily uptime and less pain, physical and psychosocial disability, depression, and pain-related anxiety, but also with some coping strategies (coping self-statements, ignoring pain and praying and hoping). In multiple regression analyses, acceptance contributed to a bigger share of the variance explained in most of the variables above, compared with coping strategies. The study was replicated by McCracken and Eccleston (2006) to take into consideration the newer psychometric advances on CSQ and CPAQ. Similar results were found with the new coping subscales (distraction, ignoring pain, distancing from pain, coping self-statements and praying). They interpreted the positive associations between some coping strategies and difficulties in functioning as indicating they are "unhelpful strategies", while acceptance showed its utility in relation to patient functioning by being associated with positive outcome measures[10].

[8]Items were first selected based on item distributions, item-total correlations and examination of initial factor analyses for single-item scales. A principal component analysis of the remaining 27 items (oblique rotation) suggested a 4-factor solution. Checks of stability and reliability of factor structure were subsequently performed on part of the sample.

[9]Catastrophizing was excluded as the authors did not consider it a coping strategy, but rather an emotional distress response.

[10]This common error is the researcher's permanent temptation, although in numerous other studies the authors do warn against interpretations of causality. In this case, an alternative interpretation is that praying, distraction and distancing are most frequently used by people that report greater difficulties in functioning. This can be due to sufferers praying more when they suffer more, to increased suffering leading to more praying or to reporting more praying and suffering because of a different process influencing both variables (e.g. a belief that this is what is expected from them, that they will negotiate a better position in the relationship with the others via this discourse). Only a thorough analysis of all these possibilities might shed more light on the causal relationships.

It is important to note that the capacity to 'predict' scores on such measures is statistical, not substantive, and can be due not to the clinical utility of acceptance-based intervention compared to coping or to its having "more utility than coping for understanding adjustment to chronic pain" (McCracken and Eccleston, 2003, p. 201), but to conceptual overlap between acceptance (CPAQ) and measures of distress and disability. McCracken (1999) admits to the possibility of content contamination between the concept of acceptance and outcome constructs such as daily functioning: for example if acceptance is defined by engaging more in daily life activities, its high correlation to measures of daily functioning does not reflect its predictive power or a causal mechanism, but an overlap of meaning. Given the behavioural focus of ACT, CPA is defined simultaneously as a set of contextual influences, the resulting behaviour, and the flexible relational pattern between them; and this overlap is a direct consequence. No clear solutions to this problem are given, except statements regarding the usefulness of measuring acceptance by overt behaviour and the researcher's freedom to focus on less contaminated factors (p. 98).

Viane et al. (2003) further reported results regarding the clinical utility of acceptance as a predictor of mental well-being (but not physical well-being) when controlling for pain intensity and catastrophic thinking. They also investigated the construct validity by analysing CPAQ factor structure and comparing it with another measure of acceptance, a subscale of the Illness Cognitions Questionnaire (ICQ; Evers et al., 2001)[11]. In their study, contrary to McCracken (1999), Cognitive Control showed medium positive correlations to Life Focus and Acceptance of Chronicity, while Avoid/Control was barely related to the other factors. Total CPAQ scores were only moderately related to ICQ acceptance scores (15% shared variance), suggesting they measure distinct concepts. The authors noted that, while the relationship with control is inconsistent, acceptance is consistently defined by focus on living a satisfying life and accepting that pain might not change and search for a cure might be in vain.

Risdon et al. (2003) further examined the concept of acceptance and the difficulty of measuring it by identifying multiple understandings of acceptance in pain-related discourse in British culture, using a Q-methodological analysis[12]. One resulting account equated acceptance with taking control: recognising that pain is part of your

[11]This subscale focuses on beliefs of adjustment and effective coping with the condition, not on specific behaviours.

[12]This method required participants with diverse understandings of the term to sort 80 statements selected from the acceptance literature based on the similarity with their views

life, trying to control it, without including disability or helplessness in its meaning. A second account, living day to day was characterised by uncertainty regarding the future and own identity, in the same time with recognising pain. A third one, acknowledging limitations, also involved recognising pain, but without efforts to control or feelings of uncertainly, rather a restructuring of goals given the current limitations. Other discourses were empowerment (finding strength from within), accepting loss of self (rebuilding a new identity which includes awareness of the negative aspects related to loss), more to life than pain (focusing on other meaningful aspects in life), don't fight battles that cannot be won (acknowledging the lack of control one has over many life events, including pain), and spiritual strength (finding strength in spirituality) (p. 375). Some themes overlapped between the some accounts, but each had its own nuanced interpretation. The authors proposed three overarching themes: focusing on other aspects of life, acceptance of chronicity (in accord with the findings of McCracken, 1999) and refusal of acceptance as sign of equating pain with failure or inferiority. The theme of controlling pain was inconsistently related to acceptance in this study, as some accounts included control while others excluded it. This inconsistency is mirrored in the controversy regarding the role of cognition in ACT.

Given the above findings regarding CPAQ, McCracken et al. (2004b) replicated the previous analysis (McCracken, 1999) on a new sample and reached a similar 4-factor solution: activity engagement (AE), pain willingness (PW), thought control and chronicity (overlapping to a large extent with Life Focus, reversed Avoid/Control, Cognitive Control, and Acceptance of Chronicity respectively[13]). Thought control and chronicity were further eliminated based on low correlations to pain-related measures in 3 categories: medically oriented variables (pain intensity, medical visits, pain medications), physical functioning (hours of daytime rest, physical disability and work status) and psychosocial issues (depression, pain-related anxiety and psychosocial disability). Thus, the two factors remaining in the current version of CPAQ were PW (comparatively more related to pain intensity, medical visits and pain medications, physical disability) and AE (more related to work status[14]; McCracken et al., 2004b). According to the updated definition, "acceptance of chronic pain entails that an individual reduce unsuccessful attempts to avoid or control pain

[13]Items that did not belong to the same scale in the two studies were redistributed based on correlations with subscales, contributions to their internal consistency and face validity.

[14]Both scales were equally related to hours of daytime rest and psychosocial measures, according to multiple regression equations.

and focus instead on participation in valued activities and the pursuit of personally relevant goals"[15] (McCracken et al., 2004b, p. 159).

Significant research efforts have been focused on investigating the clinical utility of CPA, with both cross-sectional and longitudinal designs. A further cross-sectional study (Viane et al., 2004) reported that acceptance (the ICQ subscale) was associated with lack of attention to pain (measured by the Pain Vigilance and Awareness Questionnaire) when controlling for pain intensity and demographics. This association was replicated in a diary study that related acceptance scores with lack of attention to pain and higher engagement, motivation and efficacy related to daily activities, as measured by averaged responses to individual items in electronic diary assessments for a 2-week period (also controlling for pain intensity and demographics). The authors considered these results as supporting evidence for the two factors of CPAQ: PW and AE. They proposed that the link between acceptance and lack of attention to pain is either mediated by engagement in activities or generated by a strong motivation to control and escape pain. They also noted the yet unknown mechanisms that relate acceptance to engagement in activities, but hypothesised that it is achieved by adjusting goals to the new limitations. Further mediation analyses of cross-sectional data (Vowles et al., 2008a) provided limited support for a role of acceptance as a mediator of the effects of catastrophizing on patient functioning.

The limitations of the cross-sectional design have been acknowledged by proponents of CPA (McCracken et al., 2004b), and longitudinal designs have also been attempted. In a 2-stage longitudinal study, McCracken and Eccleston (2005) presented additional evidence in support of the role of acceptance in chronic pain, by analysing correlations between scores of acceptance at time 1 and variables related to functioning at time 2 (on average 4 months apart). They found scores of greater acceptance at time 1 associated consistently with better emotional, social, and physical functioning and less medication use, controlling for pain at time 2 and demographics[16]. Unfortunately, not controlling for the influence of the health status at time 1 prevented these analyses from representing a strong test of the temporal relationship. Without such control, even if the test minimises the bias due to common measurement context (situational influences of current pain, mood,

[15] This new format of CPAQ suffers from two main problems: increased overlap with measures of pain-related disability, and method bias (all PW items are reverse coded, while all AE items are positively worded). These are discussed in detail in Chapter 8.

[16] Results of multiple regression varied depending on the outcome measure and specific subscale considered.

or social context[17]), contrary to the authors' affirmation it does not "strengthen the case for a directional relationship". The alternative hypothesis that CPA scores are to a large extent overlapping conceptually with measures of functioning remains unchallenged[18], although method overlap was partially addressed in this study by diversifying measures of patient functioning beyond self-report.

A stronger test of the clinical utility of acceptance in relation to functioning in chronic pain would be the identification of a relationship between acceptance as a stable trait (or changes in acceptance scores) and improvements in health related outcomes over time, with or without a pain management intervention. Vowles et al. (2007a) described the contributions of changes in pain, acceptance and catastrophizing scores in explaining changes in treatment outcome variables (depression, pain related anxiety, physical disability, psychosocial disability, daily rest due to pain, and direct physical performance measures) following an acceptance-based intervention (contextual cognitive behavioural therapy, CCBT). Results indicated a small but significant contribution of acceptance, catastrophizing and pain scores to improvements of treatment outcomes. However, concept overlap was not controlled in this design either, as all measures might indicate physical and psychosocial improvement from different perspectives[19].

Obviously, the controversies and weaknesses of chronic pain acceptance measures and of the methods used to test the ACT model cannot be considered as proof against the usefulness of acceptance in chronic pain interventions. In addition to questionnaire-based studies, the role of acceptance in pain is also supported by experimental studies of pain induction. For example, participants in a cold pressor task showed more tolerance of pain if presented with an acceptance-based instruction, compared with control-oriented or placebo rationales (Hayes et al., 1999a). Chronic low back pain patients experienced greater post-activity pain if given a distraction task (random interval repetition) during a pain-inducing activity, compared with a non-distraction condition (Goubert et al., 2004b). Gutierrez-Martinez

[17]For a description of this type of method bias see Podsakoff et al., 2003, p. 885

[18]The authors argued that "the stable levels of acceptance and functioning over time that subjects show do not provide an opportunity to demonstrate relations between changes in acceptance and changes in functioning" (p. 168), although they reported moderate stability for PW, AE and total CPA scores and functioning measures, and even significant increases in PW, total CPA scores and daily uptime. In a more recent study using the same methodology (McCracken and Vowles, 2008), time 1 values were controlled only for dependent variables with significant changes from time 1 to time 2. In these cases acceptance scores were no longer significant predictors.

[19]Using predictors measured simultaneously with the treatment outcomes (whether computed as gain scores or controlling for pre-treatment values) does not allow causal inferences based on results of regression models (Gelman and Hill, 2007, p. 190–2).

et al. (2004) tested acceptance-based instructions (disconnecting pain-related internal events from literal actions) with control-based instructions (changing or controlling such events) in an experimental pain inducing task (an identity matching-to-sample task involving successive exposures to increasingly painful electrical stimuli). Choosing to continue the task and be exposed to painful stimuli was motivated by material rewards and instructions related to the usefulness of the experiment in learning about chronic pain strategies. Among patients that chose to be exposed to more stimuli, ACT instructions led to higher tolerance (higher number of shocks the participants chose to be exposed to) and lower believability of pain (percent of participants who stopped the task after reporting high levels of experienced pain after a stimulus[20]). Evidence from experimental studies continues to accumulate, although not all in support of an unconditional superiority of acceptance compared to other methods (examples include Keogh et al., 2005; Feldner et al., 2006; Kingston et al., 2007; Masedo and Esteve, 2007; Roche et al., 2007; Vowles et al., 2007b)[21].

Evidence has started to accumulate from assessments of acceptance-based interventions. Dahl et al. (2004) reported good results of a brief ACT intervention for chronic stress and pain in a sample of public health sector workers at risk of high sick leave utilisation, compared to medical treatment as usual. The ACT intervention resulted in fewer sick days and treatment use, but no differences in level of pain, stress or quality of life. Related approaches such as mindfulness training were shown to be useful in addressing the initial stages of response to loss in chronic pain patients, with reduced depression and state anxiety in comparison to a control group (medical treatment or waiting list for psychological treatment), but no differences in measures of later stages of grieving or trait anxiety, which is unsurprising for a brief 8-week intervention (Sagula and Rice, 2004). An acceptance-based interdisciplinary treatment has proven to be successful in decreasing chronic pain related physical and psychosocial disability and distress in a group of patients with longstanding pain, in comparison to a waiting phase control group (McCracken et al., 2005). Further assessments have shown promising results of acceptance-based interventions for the rehabilitation of adolescents with idiopathic chronic pain (Wicksell et al., 2007), treatment of highly disabled chronic pain patients (McCracken et al., 2007a), applied in outpatient settings (Vowles et al., 2009), longstanding chronic pain and whiplash associated disorders (Wicksell et al., 2008a) and longstanding pediatric pain (Wicksell et al., 2009).

[20]Implying that they "believed" their pain sensation and acted upon it.

[21]A review of this literature is beyond the scope of the present study

However, limitations of current methods and findings should be considered an incentive to continue research in order to better describe and explain the processes at work and devise improved measurement tools. Areas of further development are outlined next.

3.6.2 Future directions

In my opinion, clarifying CPA will benefit from taking into consideration four major aspects: *a*) its relation to psychological flexibility in newer ACT formulations; *b*) its similarities and differences compared to earlier and present concepts and theories used in pain management (such as operant pain and coping, or emotions and illness perceptions); *c*) its connection to outcome measures such as pain intensity and functioning and; *d*) its component processes and the relationships among them. I will briefly detail these four areas.

Relation to psychological flexibility

The current measurement of CPA is based on earlier ACT formulations (CPAQ) or coping traditions (ICQ). As reviewed in Section 3.2, more recent ACT formulations describe avoidance of inner events such as pain, emotions, thoughts as only one aspect of the phenomenon that leads to increased pain related disability. Other aspects are: fusion with pain-related thoughts, loss of contact with the present moment, lack of values clarity, lack of consistent values-based action, and dominance of the conceptualised self. All these aspects work in a self-amplifying loop that leads to psychological rigidity and loss of vitality. A functional analysis of the patient's situation needs to tackle all these aspects in order to develop an intervention (Dahl et al., 2005).

It is time for the measurement tools to be aligned to the new therapeutic practice. For AAQ, this has only meant the replacement of 'experiential avoidance' with 'psychological flexibility'. For CPAQ, even if it is based on adaptations for chronic pain of the initial AAQ item pool, the questionnaire development process took a very different path. Thus, it is not clear if the relationship between CPAQ and AAQ is one of equivalence, nor if it reflects accurately the conceptual relations between CPA and psychological flexibility. This particular aspect deserves further study and is beginning to be addressed.

McCracken and Vowles (2007) developed a revised version of the Brief Pain Coping Inventory (BPCI-2), which includes 2 subscales: pain management strategies

(PMS), and psychological flexibility (PF, with reversed items referring to avoidance and pain control, and non-reversed items describing engagement in activities, awareness of pain related internal events and taking value-based actions). Validity analyses indicated that both scales are correlated with CPAQ subscales, but only PF is associated also with mindfulness and pain intensity, which indicates a broader definition of PF compared with the definition of acceptance as measured by CPAQ. The authors specified that their previous research on acceptance referred to its meaning as one of the 6 processes of psychological flexibility (p. 705), and that they have also addressed mindfulness, values and cognitive fusion separately (e.g. the development of a values inventory for chronic pain by McCracken and Yang, 2006), but not committed action and self-as-context. They also characterised the newly developed PF subscale as including acceptance, mindfulness, values and cognitive defusion. However, distinctions are not clear cut. For example, it can be argued that the CPAQ items referring to engagement in activities despite pain are conceptually similar with the ACT description of committed action.

A new alternative measure of psychological inflexibility was developed recently by Wicksell et al. (2008c) and focuses on avoidance of pain and cognitive fusion. The questionnaire development process started from an item pool describing avoidance, acceptance, cognitive fusion and values orientation following a similar methodology to McCracken et al. (2004b). Thus, the questionnaire suffers from weaknesses similar to CPAQ related to the use of the EFA methodology (not modelling method bias and original intended components and relying on sample fluctuations rather than theory to guide item selection), in addition to including items characterised by content contamination in relation to measures of psychosocial and physical functioning. The use of more theory-driven initial item writing and statistical methods would enable the development of a more comprehensive measure of psychological flexibility.

Further research clarifying the extent to which the six core processes of psychological flexibility overlap and the possible causal relations between them is bound to also shed light on CPA processes and the degree to which chronic pain related flexibility overlaps with general psychological flexibility, although these efforts will have to address directly the opposition between functional contextualism and elemental realism and clarify the usefulness of establishing these distinctions and relations at a higher level of generalisation. An empirical analysis of the relationship between CPA and PF is presented in Chapter 7.

Relations to other concepts

The situation of acceptance in the wider context of chronic pain management is a more complex issue. Even if acceptance is presented as a novel approach compared to OBT and CBT (see Section 2.9), the differences between CPA and concepts such as operant pain and coping still await further clarifications.

Is the concept of chronic pain acceptance a restatement of the operant pain construct? As described previously, Fordyce (1976) mentioned three instances of behaviour modification: direct positive reinforcement; indirect positive reinforcement via avoidance of aversive consequences other than pain; and lack of positive reinforcement of well behaviours. The first two seem the opposite of pain willingness, while reinforcement of well behaviours seems to describe engagement in valued activities despite the presence of pain. Thus, the two CPAQ subscales seem to be closely related at a conceptual level to operant pain instances, even if there are substantial differences in the interpretation of these phenomena in OBT as compared to ACT and RFT. Further investigation of this relation could be informative both historically and practically.

Is the concept of chronic pain acceptance a slightly different coping strategy? The questionnaire studies reviewed above showed that the differences are significant. "Acceptance-based models may provoke the average cognitive behavioral chronic pain therapist to consider more broadly the targets of his/her therapy, and whether self-control of maladaptive thoughts and feelings is the most workable way forward for the individual case in their particular circumstances" (McCracken and Eccleston, 2006, p. 28). The similarities are also important to investigate.

Research on the relationship between acceptance and other pain-related concepts such as emotions and illness perceptions is still at the beginning. Researchers recommended further "investigations to examine issues of 'experiential avoidance' in chronic pain more broadly, including avoidance of emotions and other cognitive content" (McCracken and Eccleston, 2005, p. 168). They hypothesised that "behaviour showing acceptance is produced from an interaction of past and ongoing experience with current social, emotional, and verbal influences" (McCracken et al., 2004b, p. 165). These connections will be detailed in Chapter 6 and explored statistically in Chapter 7.

Relation to outcome measures

In theory and practice, ACT has a paradoxical and unpredictable relation with the symptoms themselves. In discussing the results of a brief ACT intervention with no impact on pain and stress, only in sick leave and medication utilisation, Dahl et al. (2004) stated:

> "On the one hand, ACT does not target the symptoms directly. On the other hand, the ACT model proposes that avoidance of symptoms may actually compound symptoms that might exist absent avoidance. Thus, the overall level of symptoms ought to lessen as acceptance of negative psychological content increases and as focus on valued life domains increases. According to the model, increasing acceptance should precede lessening of symptoms." (p. 798)

Correlational studies report either weaker relationships to perception of pain intensity compared to other measures of patient functioning (McCracken and Eccleston, 2006; McCracken et al., 2004a), or none (McCracken and Eccleston, 2005). This low connection is interpreted as the result of acceptance not targeting pain, but the change in attitude regarding the sufferer's relationship with pain. This differentiation between pain perception and physical and psychosocial functioning is central to the concept of acceptance, as it refers exactly to the process of separating the pain experience from its consequences by diminishing its impact on the person's life.

This approach is presented as an advantage of ACT interventions in comparison to previous approaches:

> "If pain is indeed chronic and not eliminated by whatever behavioral effort is expended, coping may be an exhausting process of constant attempts to engage with pain as means for lessening its impact. [...] a process of acceptance, receiving or having pain without attempts at control, appears likely to give more freedom from pain, and allow more effort toward meaningful life functioning." (McCracken and Eccleston, 2006, p. 28)

Higher correlations are reported with other chronic pain outcomes such as physical and psychosocial disability, work status, reported average daily uptime in the past week, depression and pain-related anxiety. In this context, the content overlap

of CPA and patient functioning measures (discussed in Section 3.6 and Chapter 8) poses a significant problem, which might need to be specifically addressed in further research. More specifically, development and selection of measures for both correlational and longitudinal studies needs to consider the specific mechanisms targeted by ACT processes[22] and their impact on the relevant areas of functioning, rather than the use of unspecific instruments[23]

Acceptance is potentially more efficient in selected aspects of the pain experience: "the pain experience includes many components, the sensory and affective aspects of pain, emotional distress, physical impairment, and functional changes in many areas of daily activity. Some aspects are likely to be more responsive to change oriented strategies while other may be best accepted" (McCracken, 1998, p. 25).

Also, other contributing factors are important: "The acceptance-related processes we examined are not critical issues for all patients to the same extent. There are likely other issues such as social factors, practical problems of life, and other health problems, that also contribute to varying degrees to patient functioning over time." (p. 168) (McCracken and Eccleston, 2005)

The role of social factors in acceptance began to be examined with a study by McCracken (2005), which suggested that both solicitous (e.g. offering to help) and punishing responses (e.g. getting angry) of others are negatively associated with both CPA scores, while social support and distracting responses are only associated with PW. More research is needed to clarify these issues.

Component processes

As detailed in Chapter 8, the two CPA components of pain willingness and activities engagement are questionable. First, it is yet unknown whether they are distinct aspects of CPA if method bias were controlled. Second, the existence of additional components equivalent to the six components of psychological flexibility, or components characteristic only to the chronic pain experience has just began to be explored (Wicksell et al., 2008c).

[22] A brief discussion on measurement of change processes in RCTs is available in Dahl et al. (2004, p. 799)

[23] On the other hand, use of purpose-built tools will not enable comparisons across interventions.

3.6.3 Interpretation caveats

Given this strong connection to clinical practice, some cautionary statements regarding the implications of ACT in chronic pain research are necessary. More specifically, the paradoxical statements within ACT reviewed in Section 3.3 land on sensitive ground in the area of chronic pain. What to accept and what to change are sensitive issues for the patients, and no generally applicable guidelines can be given. In talking about emotional pain, ACT advises to accept "forms of pain that either necessarily come along with healthy actions or are historical in their nature, conditioned, and not based on the current situation" (Hayes and Smith, 2005, p. 122). However the chronic pain sufferers find it difficult to figure out which part of their pain comes necessarily along with their actions. Most actions that they performed without pain before the onset of their medical condition are now associated with pain, experienced intensely and overwhelmingly. McCracken (1998) underlined that the question of what and when to accept and control is "must be carefully considered on an ongoing basis by the provider of services as well as the patient", that "there may be more than one route to acceptance of pain" and that it is not to be inferred that "all patients with pain should give up trying to reduce it" (p. 25). Efforts to control pain are problematic "when they *(a)* dominate the patients life and do not succeed, *(b)* lead to unwanted side effects or complications, and *(c)* move the pain sufferer increasingly away from the things that are important to them, such as health, work, friends, and family. " (McCracken et al., 2004a, p. 4).

Naturally, improvement in individual cases is a matter of applying flexibly the ACT principles considering the specific context. However for assessment of both initial status and treatment outcome as well as for interpreting research findings and relating them to practice, it is important to acknowledge that considering individual CPA or PF scores as locations on latent continua is not consistent with ACT theory, but rather an 'elemental realist' approximation of an 'act-in-context'.

3.7 Conclusion

This chapter has reviewed the theoretical and measurement aspects of CPA, as the main concept of ACT applied to chronic pain. ACT is a promising new approach to clinical practice, based on explicit and detailed accounts of psychological (RFT) and philosophical (FC) assumptions, and offering and integrative view of psychopathology, focused on the function of internal and external events in influencing behaviour.

It contrasts inflexibility, avoidance and rule-based behaviour patterns with flexible, mindful and value-based functioning. On the other hand, the focus on flexibility and contextual variability leads to a reduced precision in construct assessment and theory development which may hinder research, especially attempts to integrate ACT concepts with other related approaches.

CPA, defined as willingness to experience pain and engagement in valued activities despite pain, has stimulated considerable research into chronic pain adjustment, mainly supporting its clinical utility. Nevertheless, the drawback of reduced precision also reflects in CPA via an overly broad definition and a degree of content overlap with outcome measures of health status, which might partly account for the positive results in the literature. Further examination of the relations between acceptance and other psychological constructs and health status indicators in chronic pain is necessary. The next two chapters will focus on two of them: emotions and illness perceptions.

Chapter 4

Emotions

4.1 Introduction

The previous chapter has focused on avoidance as a behavioural conceptualisation of chronic pain adjustment, from the perspective of ACT. Although cognition and emotion play an important role in ACT theory and practice, they are not considered as determinants of behaviour as they cannot be directly manipulated and are not independent events (e.g. Hayes et al., 2001, p. 176). In contrast, many other approaches to chronic pain management have given emotion a central causal role. This chapter reviews a variety of approaches to pain-related emotion and its role in living with chronic pain. First, it summarises several important issues from emotion theory which are relevant for the study of emotions in chronic pain. It then briefly reviews the study of emotions in health psychology. The rest of this chapter is dedicated to emotional life in chronic pain, from the emotional qualities of the pain experience to the role of discrete emotions such as anger, fear, sadness, shame and happiness and their regulation and interactions.

4.2 The theory

The role of emotional life in chronic pain is better understood when situated in the bigger perspective of emotion theory and research. The difficulties and controversies at this general level are comparable with the challenges facing pain and acceptance research and impact on the application of emotion theory to chronic pain.

The field of emotion research, also termed 'affective sciences', focuses on a fuzzy category of phenomena which guide the individual's adaptive response to the environment. Related terms such as affect, feelings, mood, motivation are often used interchangeably with 'emotion', and sometimes differentiated in relation to specific

research goals. I will use here the term 'emotion' to describe the entire range of affective phenomena, and specify limited definitions where necessary.

There are numerous definitions and theories of emotion, and it is beyond the scope of this chapter to provide a comprehensive review of the theoretical complexity of this field. I will therefore focus on a few key issues that will enable a better understanding of previous research on the emotion-pain link. First, I will give an overview of the process of emotion elicitation as it is understood by the most influential emotion theories, covering both categorical and dimensional approaches to emotion, and focusing on the important issue of the biological and cultural determinants of emotion. I will then outline some relevant theoretical views on emotion regulation and conclude this theoretical section by overviewing some implications for the measurement of emotions.

4.2.1 Emotion as a process - between biology and culture

Despite the variety of emotion theories, there seems to be a relative consensus regarding the basic components of an emotional episode. In a typical sequence, an event (whether external or internal) is followed by an interpretation, leading to an appraisal, which generates physiological changes, conscious awareness and an action potential resulting in behaviour (Power and Dalgleish, 2008, p. 131). The sequence formed by these components has been however the topic of heated debate, with two main competing proposals. One states that the event is followed automatically by physiological changes and the ensuing behaviour, and the awareness and contextual interpretation of these changes lead to the conscious emotional experience. The second focuses on the appraisal of the antecedent event as an essential component in the generation of physiological, experiential and behavioural aspects of the emotional episode. The debate, recently represented by proponents of network theories versus appraisal theories of emotion, can actually be traced back to the contrast between Platonian and Aristotelian views of emotion, and has marked the development of philosophical and psychological thinking about emotions throughout the centuries (Power and Dalgleish, 2008).

These two apparently contradictory proposals have been integrated in multi-level emotion theories (e.g. Leventhal and Scherer, 1987; Izard, 1993; Power and Dalgleish, 2008), which stipulate that a number of automatic and appraisal-driven processes can take place in the generation of emotional episodes, and the complexity and flexibility (and also the difficulty and associated risks) of emotional life

lie in the interplay between these pathways. For example, Leventhal and Scherer (1987) described three levels of emotional appraisal: perceptual-motor, schematic and conceptual-cognitive. Izard (1993) differentiated four hierarchically organised systems of information processing, neural, sensorimotor, motivational and cognitive, each placing constraints on the other systems and participating in various degrees in emotion generation and regulation (only the first being both a necessary and sufficient condition). Power and Dalgleish (2008) stipulated four interacting representation systems (schematic, propositional, analogical and associative; SPAARS), and two major processes of emotion elicitation, one via the schematic level (i.e. based on an appraisal of the present event in relation to the individual's goal structure and his/her knowledge of the world, self and others), and one via the associative level (i.e. short-circuiting the appraisal process at the present time, although the development of such a direct route assumes the occurrence of an appraisal in the individual or evolutionary past).

Most emotion theorists now acknowledge that emotional responses result from various automatic and cognitively-mediated processes determined both biologically and culturally. An essential question in emotion elicitation regards the interaction of biology and culture in the development and manifestation of our everyday emotional experience. Many models can be located on a continuum from predominance of biological factors to a more important role of culture and cognitive processes, from which different arguments are advanced regarding the question of whether emotional experience is better described in terms of distinct categories or extends along several selected continua.

The categorical approaches are based on an evolutionary and functionalist perspective: specific categories of physiological and behavioural responses have evolved in relation to specific types of environmental challenges (Levenson, 1994). Despite sharing this fundamental claim, discrete emotions theories take different stances regarding the nature-nurture issue.

Some discrete emotion theorists focus on the biologically predetermined aspects as the core of emotion. Ekman's neuro-cultural theory of emotion (Ekman, 1972, as cited in Ekman, 1994) focuses on anger, sadness, happiness, disgust, fear and surprise as a set of evolved biological functions which have a number of properties: distinctive universal signals, specific physiological changes, automatic appraisal mechanisms, universal antecedent events, distinctive appearance developmentally, presence in other primates, quick onset, brief duration, unbidden occurrence, distinctive thoughts, memories, images and distinctive subjective experience (Ekman, 1999).

The theory specifies two sets of determinants of emotional expression: pan-cultural factors (possibly due to evolution, innate neural programs or common learning experiences) are responsible for the universal aspects, while cultural differences would be seen in the eliciting circumstances, consequences and display rules of specific affects (Ekman and Friesen, 1971).

More recent views within this model (Keltner et al., 2003) take into consideration the evidence for cultural and context-related variability of emotional expression and perception and state the importance of studying individual variability, but their main interest is in emotions as biological functions, which, in their perspective, can be identified in everyday emotional experience. However, critics of the neuro-cultural model assert that, precisely due to the intervention of multiple cultural and contextual factors, these innate biological functions do not manifest directly in the everyday emotions of an adult, whether they are categorical or not. Therefore they are not easily and reliably measurable by self-report, facial or behavioural expression, or physiological changes, as components of everyday emotional responses (e.g. Fernandez-Dols and Ruiz-Belda, 1997).

Izard's Differential Emotions Theory, although it agrees with the neuro-cultural model regarding the existence of a number of biologically predetermined emotion systems, also grants a more important role of cultural and individual factors in emotional development. It considers emotion as one of six personality subsystems (with the homeostatic, drive, perceptual, cognitive and motor subsystems), and details the increasingly complex interaction between them in the individual development. Therefore Izard acknowledged the possibility of complete independence between emotional components in adult life and focused on early life development in order to identify the role of these biological processes (Izard, 1994).

Other theorists, while agreeing with a biological influence on emotion categorisation, describe emotion categories as resulting from the present interaction with the environmental challenges, as filtered by the individual's interpretations. These categorical criteria are termed 'core relational themes' (Lazarus, 1991), 'action tendencies' (Frijda, 2008), 'cognitive evaluations of a juncture in action' (Johnson-Laird and Oatley, 1992), or simply appraisals (Power and Dalgleish, 2008), and emerge from the interaction with types of tasks that individuals are frequently confronted with in similar ways. The number of discrete emotions varies in these theories, but an important common aspect is the acknowledgement of the cognitive and environmental determination of emotion categories. For example, Lazarus (1991) states

that many emotions that "may have emerged on the basis of greater human cognitive and social complexity [...] may be [...] no less primary in the process of survival" (p. 81).

Partly as a consequence of viewing emotion categories as the result of the interaction between innate and environmental factors, these theories give a different interpretation to emotion measurement. Instead of being indicators of innate programs, measures of emotional experience via self-reports, changes in physiology or behaviour (including facial expression) tap into various emotion components which acknowledged to have a "loose and variable relation" (Frijda and Tcherkassof, 1997) in everyday experience, or even become dissociated in certain situations (Power and Dalgleish, 2008).

This approach concurs with other theories of a more sociological inclination which also highlight distinct emotion components from a communication perspective. For example, Buck's (1999) developmental-interactionist theory describes emotion as resulting from the activation of primary motivational-emotional systems (primes) by a challenging stimulus and manifesting in three readout systems: arousal, expression, and experience. These readouts develop to serve distinct functions: the arousal system aims to ensure physiological adaptation and homeostasis, the expressive behaviour system is developed for communication and social coordination, and the subjective experience system targets self-regulation. The different pressures they are subject to in the individual's developmental history result in a relative independence of these systems, but also in an increasingly complex interaction pattern. It also describes a developmental process in which primes (viewed as phylogenetic adaptations to typical environmental challenges), interact with general purpose systems (such as conditioning, learning, higher-order cognition and language) and the social and physical environment in the course of individual development to generate higher-level affects (social, moral and cognitive).

The proponents of dimensional theories of emotion are generally of a more social-constructivist orientation, although they certainly acknowledge the role of evolutionary mechanisms, and adopt equally varied approaches. For example, Watson and Tellegen (1985) proposed two orthogonal dimensions to best represent the structure of emotional experience, positive and negative affect, based mainly on factor analyses of self-reports of mood states, and viewed them as complementary to categorical approaches; an alternative and compatible (but less preferable in their opinion) description of this two-dimensional space is via pleasantness and arousal.

In a reformulation of this model, Watson et al. (1999) described a three-level hierarchical structure with the pleasantness-unpleasantness dimension at the highest level, followed by the positive and negative affect dimensions (relabelled Positive Activation, PA, and Negative Activation, NA), and discrete affects at the lowest level, and allowed for context-dependent structure flexibility. PA and NA were viewed as reflecting the subjective components of two evolved general behavioural systems, namely approach and withdrawal[1], while the pleasantness dimension is also a result of an "innate and essentially universal classifactory response" (p. 828).

Russell (2003) and Barrett (2006a) proposed an alternative model built around the concept of core affect: "the constant stream of transient alterations in an organism's neurophysiological state that represents its immediate relationship to the flow of changing events" and is available to consciousness via assessments of valence and arousal either as object-less mood or in relation to present stimuli (Barrett, 2006a, p. 48). Attributed core affect, together with its momentary change (alone or combined with information processing and behavioural planning), are considered the primitive building blocks of all emotional experience, including prototypical emotion episodes. The conceptualisation of core affect into discrete emotion episodes is also the focus of Barrett's (2006b) conceptual-act model and can be considered an effort to bridge dimensional and categorical views. This model is somewhat similar to an earlier attempt to combine the dimensional and categorical approaches, this time from the perspective of achievement and decision-making: Weiner's (1985) social-cognitive (attribution) theory of emotion. It states that the experience of the outcome of an event is assessed initially based on the criterion of success or failure, thus generating an "outcome dependent-attribution independent" positive or negative emotion. A consequent causal attribution based on several dimensions (locus of control, stability, controllability, and perhaps also intentionality and globality) generates specific emotions, such as pride, anger, pity, shame etc.[2]

Some appraisal theorists go beyond the discrete versus dimensional approach and describe a dynamic process, in which different types of appraisals (regarding stimulus characteristics, its motivational relevance, the individual's power and abilities to cope, and the social implications) are continuously and often simultaneously

[1]These are similar to two of the three systems stipulated by the Reinforcement Sensitivity Theory (Gray and McNaughton, 2003), which distinguishes between behaviour inhibition, behaviour activation and the fight-flight system, and to Lang's (1995) two motivational systems based on neural functioning, appetitive (approach) and aversive (avoidance), although in the latter theory they support the alternative dimensions of valence and arousal.

[2]This theory, although only part of a broader theory focused on motivation and not emotion, has had a considerable impact on health psychology via its cognitive aspects (i.e. attributions).

interpreting and reinterpreting the environment and thus guiding the individual's physiological, experiential and behavioral responses (Ellsworth and Scherer, 2003). Both phylogenetically and ontogenetically, emotional differentiation is the result of cognitive development which allows an increasing number of appraisals. The focus shifts from a category/dimension analysis to a process analysis, and from this perspective the difference between the regulatory role of emotions and emotion regulation becomes increasingly difficult to perceive.

The diversity of competing theories and the difficulty to interpret the accumulating evidence and gather decisive data make the quest for the 'best' theory impossible, at least at present. As an example, neuropsychological evidence, although already substantial and considered essential to these disputes, is amenable to various competing interpretations. Many emotion researchers supporting the categorical structure of the biological determinants of emotion interpret the neurophysiological evidence as proof of separate neural systems, although there is limited consensus on the number and functions of the distinct systems (Panksepp, 2000, 2008; Murphy et al., 2003; LeDoux, 2000). Dimensional interpretations of the existing evidence have also been presented (Watson et al., 1999; Barrett and Wager, 2006), and several other interpretations can be located between dimensional and categorical views (e.g. Buck, 1999). Beyond the involvement of multiple subcortical and cortical structures (e.g. thalamus, hypothalamus, amygdala, hippocampus, nucleus accumbens, the anterior cingulate, prefrontal and frontal cortex) and neurotransmitters (e.g. dopamine, serotonin, norepinephrin, oxytocin) in the generation of emotional experience, the clarification of these controversies still awaits further evidence.

I have presented above only a general overview of the multitude of issues and interpretations in this complex field, but I will attempt based on this limited description to draw an important conclusion for the application of emotion theory in health and chronic pain research. The biological influence on emotional experience is certainly amenable to different interpretations, from categorical innate programs to dimensional appetitive and aversive mechanisms. However there seems to be a consensus regarding the fact that emotional life is described categorically in most cultures (with both common elements and cultural variability involved; e.g. Scherer and Wallbott, 1994). These distinctions are in the Western culture summarised by discrete emotion terms, among which anger, joy, sadness, fear and disgust are reliably identified. Based on these convergent research results I would argue that discrete emotion terms play an essential role in both the description and the structuring of emotional experience. In everyday emotional life, the selection of adaptive

responses relies on one's ability to use the distinctions between broad categories of situations operating in one's cultural environment. As tokens for intra- and interpersonal exchanges of meaning, these labels are the product of the interaction between biological and cultural forces and can be considered useful both in terms of characterisation of emotional episodes, and as instruments for emotion regulation.

4.2.2 Emotions and emotion regulation

Emotions are both regulatory forces aiding adaptation and objects of regulatory efforts. The field of emotion regulation (ER) developed mainly from the literature on emotion-focused coping, with significant influence from psychoanalytic literature, and studies deliberate and automatic efforts to influence the type, duration, subjective experience and expression of intra-individual emotional episodes (Gross, 1999). Other authors include in the ER definition not only the modification of emotional reactions, but also their monitoring and evaluation, and underscore its goal-directedness (Thompson, 1994).

As opposed to coping, which includes actions directed at non-emotional goals and can extend on longer periods of time, ER refers to efforts directed at modifying a time-limited emotional reaction, and can use both coping strategies such as rumination, suppression, or social comparison, and other mechanisms such as regulation of expression and physiological changes. These regulatory efforts can aim at decreasing or increasing both positive and negative emotions and can address any of the components of an emotional episode, from selecting and modifying the antecedent events (via attentional processes), to changing its cognitive interpretation to modulating the response components (Gross, 1999, 2008). ER is also different from emotion sensitivity, which refers to the onset of emotional episodes rather than the control on its total duration or other properties (Koole, 2009).

Two main approaches can be distinguished in the study of ER. One focuses on the immediate consequences of specific ER strategies (such as reappraisal, suppression, social comparison, or rumination) in specific contexts. The second addresses individual differences from the perspective of trait-like abilities or deficits in ER (such as alexithymia, coping style, or emotional intelligence) and their relation to achievement or mental and physical health[3] (Gross, 1999). Between the multitude

[3]A more recent approach adopts a dynamic outlook and attempts to identify the effects of ER efforts via differences in the intensity or frequency of a specific emotional state in a given time period, and their connection with other phenomena such as reports of physical health (e.g. Paquet et al., 2005).

of ER strategies and the general trait, there is a third level of analysis which tries to classify ER efforts depending on various relevant criteria. For example, in a review of the literature, Koole (2009) used a dual classification, based on the target of ER efforts (attention, cognition, physiology), and its functions (satisfying hedonic needs, supporting goal achievement, optimisation of personality functioning)[4]. However the author acknowledged the purely theoretical nature of this taxonomy and the lack of empirical evidence to support it.

To date, the only taxonomy with preliminary empirical support is a recent proposal which distinguishes between internal and external, and functional and dysfunctional strategies (Phillips and Power, 2007). The functionality of ER strategies refers to the possible adaptive or maladaptive medium-to-long term consequences of the tendencies to use particular types of strategies, while the internal-external distinction follows an established tradition in developmental and clinical psychology and refers to the intra- or interpersonal resources the individual accesses. The simplicity of such a taxonomy, far from intending a judgemental outlook on ER, is imposed by the limitations of the self-report method (Power, 2008). Preliminary data on the validation of the Regulation of Emotions Questionnaire are supportive of these distinctions and their relevance for emotional and behavioural problems, general health and quality of life in adolescents (Phillips and Power, 2007). This approach however necessitates further empirical testing, especially concerning the causal relationships with psychological distress and the distinctiveness of these categories in other populations.

4.2.3 General issues in emotion assessment

As with pain and acceptance, the complexity and elusive nature of the phenomenon invite the question: can we measure emotion validly and reliably? Beyond the issues of recall bias and the intersubjective nature of emotion, which are similar to the measurement of pain, emotion assessment is subject another difficulty: while separating related dimensions was an issue in pain measurement, emotion measurement is encumbered by the low coherence of emotional response components.

As discussed in the previous section, some functionalist views hold that the emotional reaction implies coordinated changes in multiple systems, which sustain the adaptive role of emotions to this process. Thus, a researcher studying emotion

[4]In this classification, ACT methods might be considered ER strategies, for example mindfulness as targeting attention, or value-based action as cognitive integration, both functioning to increase flexibility of personality systems.

would need to consider the three main systems where emotional activation manifests: physiology, evaluative language and behaviour (Lang, 1995). But coherence of the emotional activation systems is still a matter of dispute.

Physiological 'signatures' for distinct emotions have been identified in controlled experiments (Levenson, 2003). Other studies reported coherence between self-report, facial behaviour and physiology in lab film viewing induced emotions of amusement and sadness, although a higher correlation was found between subjective experience and behaviour than between both and physiology (Mauss et al., 2005). But the very fact that strict experimental control is needed to achieve coherence speaks against the reliability of physiological measures of emotion in daily life. For example, Myrtek (2004) reported a lack of correlation between heart response to emotional and mental work load versus momentary self-report of conscious emotions, in both laboratory and ambulatory settings[5].

The expression of emotion via facial behaviour or actions is also loosely related to self-report in everyday situations. Some authors assert that facial behaviour is an accurate indicator of emotion (Keltner et al., 2003), based on evidence of small to medium associations between facial displays and other emotion aspects (self-reports of emotional experience, physiological responses, brain activity, environmental events and cognitive appraisals). But for a valid and reliable measure, these associations also need to be specific (the measure is associated only with its related phenomenon) and generalizable (the association is detectable irrespective of context), while current evidence indicates that facial behaviour is related to many other factors and is context-dependent (Dima, 2009).

Given these considerations, many authors recommend the use of indicators from all three systems in order to gain a more reliable measure of emotional life, especially due to the high degree of independence between systems (Wilhelm et al., 2006). As both physiology and behaviour measures are more difficult to apply outside laboratory settings, self-reports are the most frequently used measure of emotional experience. But this loose connection between physiology, experience and behaviour raises the question of what do self-report measures of emotion actually assess.

As discussed in Subsection 4.2.1, emotional life is the result of the interplay between biology and culture. Thus, emotion labels cannot be a direct reflection of

[5]The lack of a strong link between physiology and emotion might be explained by a mediation role of abstract representations of emotion-related physiological changes (Philippot and Rime, 1997), which are likely to be influenced by both biological and cultural factors, as there are substantial similarities between considerably different cultures (Breugelmans et al., 2005).

universal/innate emotional systems, nor uniquely culturally constructed. The development of emotional language at the individual and societal level is still a matter of research. Beyond this theoretical issue, self-report measures (whether responses to questions related to the frequency or intensity of emotion categories or dimensions, or emotion narratives) are momentary constructed descriptions, and are thus influenced by a multitude of factors (the individual's recent experience, contextual characteristics of the questionnaire application, individual characteristics of language use, etc.), which need to be considered in the interpretation of self-reports both in terms of meaningful sources of variance and as measurement error.

In essence, emotion labels can be considered as the currency of our verbal communication of emotional experience, loosely related to generic themes of experience relevant for the culture in which they evolved and probably related to a certain extent to some species specific adaptive behaviours. Actually, this mixed heritage makes them a suitable instrument for emotion regulation efforts, as differentiation of emotional experiences based on these labels helps select the most suitable responses in the given cultural context. Their central position in emotion regulation via communication justifies their relevance for emotion research[6]. Nevertheless, their dependence on the variability of personal experiences and language use leads to difficulties in emotion measurement (as with pain, see Subsection 2.7.1). Emotion labels do not encode 'real' neuro-psychological entities, as pain descriptors do not encode 'real' pain qualities, but reflect the intersubjective nature of language use and thus the dynamics of human interaction. Thus, any emotion measure (as any pain measure, according to Crawford, 2009) participates in the construction of the individuals' momentary descriptions of their emotional life (based on recent experiences, social interactions, emotion regulation efforts, the context of the questionnaire completion, etc.).

4.3 Emotions and health

The study of emotions and chronic pain has been naturally influenced by research on the role of emotions in health, specifically in chronic illnesses. Both categorical and dimensional approaches to emotion have been studied in health-related contexts. The dimensional approach has tended to support the role of positive emotions (joy, optimism, hope, love) and related behaviours in preventing or curing illness and

[6]However, it is important to note that English terms such as anger, fear etc. are characteristic only for English speaking cultures, any crosscultural study would need to redefine the relevant categories to a certain extent.

increasing health and well being, while negative emotions (aggression, hostility, depression, anxiety, guilt) were viewed as increasing the risk of various physical conditions such as cardiovascular diseases, immune deficiencies, cancer (Cacioppo, 2003). However promoting positive emotions as a curative force risks becoming an additional stress for the patients facing an already threatening illness who find themselves restricted in their emotional responses by the prescription of a positive outlook and the interdiction of the justified distress elicited by the symptoms and consequences of illness (Moskowitz, 2008).

Categorical approaches have brought a fundamentally different idea that both negative and positive emotions guide adaptation and decision making. In this perspective, emotion regulation, not an unyielding optimism, is the key to increase the adaptive functions of emotions and decrease the associated risks. However, basic emotions have mostly been studied separately in different health conditions. Among the advantages of studying basic emotions in health contexts is the possibility to study emotions in shorter time frames. While dimensional models operate in an accumulation and chronicity model, basic emotions are more adequate for studying the impact of state-like emotionality on health behaviour and decision making (Consedine and Moskowitz, 2007).

The mechanisms by which emotion and health interact could be situated on a time line from primary causative and preventive (physiological changes that exceed the body's capacity to keep homeostasis lead to functional and structural changes, immune response suppression leads to increased vulnerability to external factors), to secondary causative and preventive (mediation via health behaviours - nutrition, exercise, sleep pattern etc.), to symptom attention, sensitivity and reporting (self-perceived emotions, self-perceived health/illness, well-being), medical contact, detection and screening behaviours, treatment decision making and treatment adherence (Consedine, 2008). In this classification, the issue of managing life with a chronic condition such as pain could be considered an aspect of the last two categories: the continuous adaptation to the challenges brought by pain and its personal and social consequences necessitates numerous decisions related to diminishing its impact and its aggravation, as well as adhering flexibly to various pain management behaviours (from medication to self-help methods).

4.4 Emotions and chronic pain

Pain is a special case both as a symptom associated with other health conditions, and as a stand-alone condition, in that it is both a sensory and an emotional experience. This characteristic makes pain a particularly adequate topic of study for biopsychosocial approaches to health (see Chapter 2). Thus, the role of emotions in chronic pain has been fundamental to research efforts regarding definition, etiology, diagnosis and treatment.

Early accounts of the relationships between emotions and pain were formulated in a psychoanalytic framework. Engel (1959) placed the affective tone of pain at the centre of the individual's development and functioning in terms of body image, relationships and related feelings of guilt, aggression, loss and sexual excitement. He distinguished a special type of 'pain-prone' patient for whom pain plays an adaptive role and is thus paradoxically associated with pleasure, even if only by avoiding other more unpleasant experiences. If peripheral pathological processes do not account for the character of the pain experience as reported by the patient, clinical explanations need to be sought in the 'psychic distortion and elaboration' of pain. His description of this psychological level was in terms of emotions. Pain, he stated, can be used as self-punishment to ease feelings of guilt, closely related to difficult childhood experiences involving aggression, loss and the development and socialisation of sexual impulses. Although this approach was revolutionary at the time and the astuteness of the clinical observations presented is still impressive, the abuse of this label in practice (especially when associated with the limits of diagnosing the medical condition) and lack of empirical support has not led to effective interventions (Keefe et al., 2001).

The OBT approach to pain (Fordyce, 1976) reinterpreted in a behaviorist language the relationships identified in the psychoanalitic literature regarding emotional factors and pain. In Fordyce's interpretation, the link with early childhood experiences of pain became a history of conditioning relevant for the current development of pain behaviours. Anxiety is inevitably experienced throughout the individual's life in relation to potential harm or loss, and it is naturally associated with depression as a sign of loss. Punishment can also be associated with pain, and in this context depression and guilt are present and with them probably withheld anger and hostility. These represent opportunities for learning and it follows logically that the more such events were present in the person's life, the stronger the association between emotional distress and pain.

Fordyce described a "snowballing or vicious cycle effect" by which frequent emotional distress prevents normal relationships and therefore predisposes the individual to more inner focus on distressful feelings or bodily sensations. Strong associations make all these separate states behave as a "single response class", and stimuli that elicit one state may also elicit another. Discrimination between these states is difficult at the level of conscious experience, and so is labelling them internally or when communicating these states to others. This automatic discrimination error does not imply a causal link between emotions and pain, and Fordyce warned against using the presence of distress for using the diagnosis of psychogenic pain in the absence of an identifiable physical injury. He stated that proving the existence of a causal link in a specific case involves identifying systematic relationships between emotional stimuli and reports of pain, but did not develop this subject further. Fordyce made no further distinctions between the roles of separate emotions, and did not mention any relationship between happiness (or any positive feelings) and reports of pain. His approach focused strictly on pain behaviours as operant, and the methods to diminish them and promote and maintain well behaviours.

The CBT approach to pain has focused mostly on cognitive constructs such as illness schemata, beliefs, attitudes, attributions, cognitive distortions and their impact on decision making and coping (see Chapter 5). Cognitive models have generally neglected the role of emotions (Skevington, 1995, p. 173), but in the last decades the general interest in emotions has also kindled research on their role in chronic pain, in close connection with research on stress and health (Keefe et al., 2001).

Research on the relationship between stress and pain has revealed a complex pattern according to which the impact of stressful events depends on the interpersonal context, the individual's appraisal, the coping resources available and the action taken to counteract these effects (Keefe et al., 2001). In certain conditions, stress has also been shown to induce analgesia, via multiple neural and hormonal mechanisms (Bonica, 2001, p. 137–140). These findings exposed the necessity of focusing on specific emotional states and emotion regulation strategies. Thus, while dimensional views of emotions have been more influential in other health-related areas, the relationship between emotions and chronic pain has been formulated more in terms of basic emotions. Fear/anxiety and anger have been by far the most studied, while sadness, shame and happiness have been usually incorporated in the study of depression and only recently addressed more directly. However, one of the biggest challenges of emotion research in chronic pain is actually separating emotional aspects of the pain experience itself from aspects of the individual's emotional life

that are distinct from pain. I will start by describing the affective component of pain and then focus on each of the five basic emotions.

4.4.1 Emotion as a dimension of the pain experience

Emotion has been considered a constitutive component of the pain experience. As discussed in Chapter 2, it is the characteristic unpleasantness of pain that distinguishes it from sensations. The degree to which the affective component is distinct from the sensory and evaluative components is however controversial (Fernandez and Turk, 1992).

Well-established measures such as the MPQ include motivational-affective terms in the assessment of pain quality. Melzack and Katz (2001) asserted that the 3-dimensional structure of the MPQ has a strong support, especially the distinction between the affective and sensory dimensions, even if some evidence exists against the relevance of two separate affective and evaluative subscales. The existence of alternative proposals for the MPQ structure consisting in a variable number of factors was explained in terms of the differences in samples and factor analytic methods used. In contrast, Holroyd et al. (1992) reported high correlations between the subscales and lack of discriminant validity in relation to measures of psychopathology. In their analysis, a second-order pain-distress factor (on which the affective dimension had the highest loading) explained 62% of the variance in MPQ scores, while the primary factors had minor contributions[7]. But high correlations do not necessarily imply lack of distinctiveness, and other authors argued for the necessity of separate measures of pain affect and pain sensation based on research showing the selective influence of experimental pain induction and treatment interventions on the two dimensions (Wade et al., 1990; Fernandez and Turk, 1992; Melzack and Katz, 2001).

Wade et al. (1990) went one step further in suggesting that the affective component is actually multidimensional, and reducing the emotional disturbance due to pain to a single 'unpleasantness' term is too simplistic. They differentiated between anger, frustration, anxiety, fear and depression as predictors of pain-related unpleasantness and clinical depression when controlling for pain sensation intensity and report

[7]The difficulty of clarifying the issue of component distinctiveness is increased by a particularly strong contribution of instrument variance to the high correlations between the subscales, as shown by Holroyd et al. (1996). The authors noted that the difficulties the patients face in describing their pain might make them inherently sensitive to format- and context-related error (p. 263).

that anger, anxiety and depression are associated with clinical measures of depression, while anxiety, frustration and anger are related to pain unpleasantness. They interpret these differences as evidence for the usefulness of distinct emotion reports and perhaps targeted interventions.

Along the same lines, Gaskin et al. (1992) reported that measures of anxiety, anger and depression are associated with pain intensity (MPQ) scores[8]. Fernandez and Milburn (1994) studied 10 distinct emotions in relation to affective, sensory and overall pain assessments and identified anger, fear and sadness as most relevant for predicting affective pain[9]. Based on this research, Fernandez and Boyle (2001) used anger, anxiety and depression as subcategories of the emotional component of pain to guide an analysis of the affective and evaluative descriptors of the MPQ, thus implicitly considering the three emotion categories as indicators of a single concept of emotional distress subsumed to the concept of pain[10]. Clark et al. (2002) showed that the emotional qualities of pain (and four of its components - anxiety, depression, fear and anger) can dominate the somatosensory and well-being dimensions in global intensity self-reports of postoperative pain, thus bringing further evidence for the importance of the affective dimension of pain. A stronger link between affective qualities and overall pain intensity was confirmed in a within-person, momentary assessment design (Litcher-Kelly et al., 2004).

A different proposal is the sequential processing model of pain which describes the affective dimension of pain as the result of multiple processes: pain sensation, arousal, autonomic and somatomotor activation and cognitive appraisals (Price and Harkins, 1992; Price et al., 2001). These processes take place in two stages: first-order appraisals related to pain sensation, arousal, activation and perceptions of the immediate context, followed by second-order appraisals based on the first stage and additional assessments of the long-term implications of pain.

In addition to relying on rich data on experimental pain induction, this model of pain-related affect shows a thorough understanding of current theories of emotion.

[8]The authors suggested that these relations might also be explained in terms of attributional processes (i.e. the responses to questionnaires might reflect respondents' interpretations of general arousal prompted by emotion or pain-related items).

[9]The emotions were selected based on Izard's differential emotions theory, and grouped in 3 categories: physiologically prewired negative emotions (anger, fear, sadness), cognitively modulated negative emotions (guilt, shame, disgust, contempt) and positive or neutral emotions (surprise, interest and joy).

[10]In this study, participants were asked to classify descriptors into the three subcategories plus an overall intensity-evaluative subcategory. Few affective descriptors were shown to relate unambiguously to a specific subcategory, which reflects the difficulties in assessing the emotional aspects of pain via self-report.

The authors refer to Damasio's (1999, as cited by Price et al., 2001) distinction of *core consciousness* and *extended consciousness* and to cognitive appraisal theories to highlight the interaction between meaning and physiological activation in the generation of emotional feelings. In the first stage, the qualities of pain sensations and associated activation and contextual factors are appraised as intrusion and threat at the level of *core consciousness*, resulting in an overall assessment of pain unpleasantness which leads to simple pain behaviour. The second stage processes the first stage input and additional context information at the level of *extended consciousness* to generate complex pain behaviour and various emotions (anger, frustration, sadness, anxiety, etc.) depending on the nature of the appraisals involved. Thus, the model shares a common perspective with many approaches to emotion theory. It follows multilevel models of emotion in its attempt to integrate both automatic, immediate processes and conscious, appraisal-mediated processes in the generation of emotional states. Also, it shares common elements with specific emotion theories, such as the core affect and the conceptual-act models (Russell, 2003; Barrett, 2006a,b) which also draw from Damasio's distinction and try to bring together dimensional and categorical approaches to emotion.

The sequential processing model is also supported by neurophysiological data (Price, 2000, 2002; Chapman, 2004). Limbic neuroendocrine physiological mechanisms (especially the locus ceruleus and dorsal noradrenergic bundle; the ventral noradrenergic bundle and the hypothalamo-pituitary-adrenocortical axis; and the central serotonergic pathways) participate in both emotion and pain perception and are likely responsible for an immediate response to threat that automatically discontinues ongoing attentional and behavioural processes and alter bodily states. Activity in multiple cortical areas results in second stage affective states. Some areas seem to participate selectively in pain related primary affect (e.g. the anterior cingulate cortex; Price, 2000). Interestingly, empathic reactions to other person's pain are thought to involve predominantly these areas (Singer et al., 2004; Singer and Frith, 2005)[11].

[11]As detailed in Chapter 2, pain perception, modulation and behaviour emerge from the activity of multiple ascending and descending pathways, cortical and subcortical centers and neuroendocrine mechanisms, many of them also participating in emotional processing, as described earlier in this chapter (for a different interpretation of the neurophysiological literature, see Mollet and Harrison, 2006). The extent to which specific neural formations are dedicated to specific functions and the correspondence between these functions and categories of behaviour as discriminated by observation are however controversial issues in the neuroscience literature (Cacioppo et al., 2000).

Although it is by far the most elaborate model of pain affect and has important implications for clinical practice, the sequential processing model faces a major difficulty when applied beyond experimental settings. Chronic pain patients find it difficult to distinguish between pain intensity, affect, disability and other related aspects when asked to report their health status. For example, in answering a question assessing pain intensity patients might corroborate information about his immediate affective reactions, multiple appraisals of the implications of their pain, the various emotional states they have recently experienced, emotion regulation efforts, etc. Thus, one might never obtain a 'clean' measure of a concept, and correlations between various measures also reflect similar processes patients employ when responding to particular questions. This issue of "discrimination error" is central to emotion measurement in chronic pain, in addition to the difficulties discussed in Sections 4.2.3 and 2.7.3.

In conclusion, depending on the characteristics of the measurement tools, sample and context, emotion can been conceptualised as overlapping with sensory characteristics of pain, as a distinct dimension, as contributing factor to overall pain intensity assessments, as a result of appraisal processes based on other pain and context characteristics, or even as a group of distinct concepts interacting with the pain experience. Some examples of the role of unique emotions in chronic pain or symptom perception and adaptation to illness are presented next.

4.4.2 Anger

Anger and frustration are reported as the most intense emotions experienced as concomitants to pain (Wade et al., 1990; Fernandez and Milburn, 1994; Price, 1988 as cited in Price et al., 2001). Yet the role of anger and anger regulation in chronic pain has received considerable attention only in the last two decades. As seen in earlier sections, previous attempts to link anger inhibition to pain severity were either based on psychoanalytic views and were thus only clinical descriptions (Burns et al., 2008c), or merged anger into a general construct of emotional distress.

Fernandez and Turk (1995) described two possible pathways through which pain may generate anger, according to emotion theories: an immediate, automatic, non-cognitive activation related to the sensory properties of pain, and a conscious activation mediated by cognitive appraisals of goal obstruction or mistreatment directed at the source of injury, medical and legal systems, significant others or self (see also Okifuji et al., 1999). They noted the high prevalence of anger and hostility

relative to other emotions reported in the literature, possibly underestimated due to denial, and also of anger suppression. They reviewed the limited research at the time supporting hypotheses regarding the role of inhibited and expressed anger in the etiology of chronic pain or in the adjustment to chronic pain by its influence on associated depression, interpersonal difficulties, comorbid health problems, poor health habits or failure to cooperate in treatment programs.

Initial research focused on the concepts of 'hostility' and 'anger management style' (with two dimensions, anger-out, representing a tendency to manage anger through verbal and physical expression, and anger-in, a tendency to inhibit expression). Several studies using self-report (i.e. the Anger Expression Inventory; AEI) revealed a complex picture of the relationship between anger and pain, mainly focused on the role of anger expression. In addition to the moderating effects of gender and level of hostility and the mediating role of spouse punishing responses (Burns et al., 1996), anger expression was shown to be linked with pain severity by various psychophysiological mechanisms related to stress responses: muscle reactivity, endogenous opioids activity, blood pressure reactivity, and sympathetic hypofunction leading to increased catecholamine sensitivity.

The most documented mechanism is the hypothesis of an endogenous opioid dysfunction. As reported by Bruehl et al. (2002), anger expression (but not anger suppression) was associated with absence of opioid analgesia during acute pain tasks for both chronic pain patients and healthy controls. Also, stronger opioid analgesia for experimentally induced pain partially was reported to mediate the relationship between low anger expression and reports of lower daily chronic pain intensity (Bruehl et al., 2003b). Variance in anger expression scores and variance in blood pressure reactivity (another physiological effect of endogenous opioids) overlapped in explaining variance in sensitivity to experimentally induced pain (Burns et al., 2004). Anger expression was associated with higher pain severity only in patients not taking opioids as a treatment, controlling for depression, anxiety and antidepressant intake (Burns and Bruehl, 2005). Research also identified a genetic moderation of the relationship between anger-out and analgesic demands after coronary artery bypass surgery (Bruehl et al., 2006b), and recently confirmed for the relation between anger-out and ratings of pain intensity following an experimental pain induction, for both chronic pain patients and healthy controls (Bruehl et al., 2008b). Greater anger expression scores significantly predicted smaller pain-induced increases in plasma endogenous opioids following pain induction, and this relationship mediated the effects of anger expression on pain unpleasantness, controlling

for negative affect (Bruehl et al., 2007b). The relation between anger-out and endogenuous opioid analgesia to acute pain in healthy individuals was apparently moderated by gender (Bruehl et al., 2007a). Recent work suggested that negative emotional responses to experimental noxious stimuli in chronic pain patients were also affected by opioid dysfunction in individuals with high anger-out scores (Bruehl et al., 2008a).

These studies present a strong case for the endogenous opioid deficit as important physiological correlate of the link between anger expression and pain severity. This finding might reflect several alternative mechanisms. Anger expression might contribute to a stressful personal environment, which stimulates secretion of endogenous opioids, leading in time to either increased tolerance or chronic depletion of opioid stores, resulting in incapacity to modulate pain perception and mood. Alternatively, chronically low opioid activity (due to genetic or environmental factors) might lead to decreased ability to regulate both anger and pain perception, resulting in overt expression of anger and increased pain sensitivity; or both deficits of endogenous opioids and ability to regulate anger might be enhanced by physiological changes characterising chronic pain in some predisposed individuals. The presence of this mechanism in healthy individuals rules out the possibility of the deficits being the direct result of chronic pain related changes (Bruehl et al., 2002, 2003b).

Several studies support the other physiological mechanisms. For example, Burns (1997) reported that anger management style, hostility and gender interactions contribute to the aggravation of chronic low back pain by increasing symptom-specific muscle tension during events eliciting anger, in addition to cardiovascular changes. In a study by Burns et al. (2003), healthy individuals with high anger expression scores reported higher pain intensity in experimentally induced pain only following a situation eliciting anger and the relationship was partially mediated by blood pressure reactivity. Bruehl et al. (2003a) found that anger-out was related to increased pain severity in patients diagnosed with chronic regional pain syndrome (CRPS, condition characterised by sympathetic dysfunction) but not in patients with non-CPRS limb pain.

In a recent review of the literature on anger expression, Bruehl et al. (2006a) noted the importance of distinguishing between trait and state anger-out. While the former is measured by the AEI subscale, the latter is experimentally manipulated by harassment methodologies (anger elicitation with or without the possibility to express anger). They proposed that trait and state anger interactions might have an

impact on pain perception: in anger-out individuals, expressing their anger might reduce arousal and negative health effects, suppressing might have opposite effects. Moreover, they replaced the previous opioid deficit hypothesis with that of an opioid triggering mechanism: high anger-out individuals might have a higher threshold for opioid release, for which behavioural anger expression might act as a trigger.

Even if most findings focused on anger-out, anger-in was also shown to interact with hostility in predicting increased low back pain via increased muscle tension during anger elicitation (Burns, 1997; Burns et al., 2006). Burns et al. (2008c) explained the unclear results regarding anger inhibition by limitations in the self-report measure used: unclear conceptualisation of trait and state anger-in and overlap with general measures of negative affect. They proposed adopting an experimental research paradigm based on Wegner's ironic process theory of thought suppression (Wegner, 1994, as cited in Burns et al., 2008c), which enables assessing the effect of state anger suppression on subsequent pain processing. According to this model, a monitoring process ironically makes the suppressed content highly accessible and thus able to influence perceptions of consequent events (in this case, pain) in a manner congruent with the suppressed content (anger).

Working within this paradigm, Quartana and colleagues have reported that experimentally manipulated anger suppression (but not anger control) during anger induction (but not anxiety induction) led to later reports of higher pain intensity (and more pain behaviour) following pain induction in both chronic pain patients and healthy individuals, and the relationship was partly mediated by ratings of anger-related affective pain qualities and anger levels (Quartana and Burns, 2007; Quartana et al., 2007; Burns et al., 2008b). For chronic low back pain patients with high anger-out scores, experimentally induced pain-related distress suppression (and not other forms of anger regulation) contributed to increased symptom-specific muscle tension during pain induction (Burns et al., 2008a), also when followed by a mental stressor and a recovery period (Burns et al., 2009). A study from a different research group brought preliminary support for the role of (written) constructive anger expression in improving perceived control over pain and depressed mood (and marginally pain intensity) in chronic pain patients; this relationship was partially mediated by the amount of anger and meaning making expressed in the written text (Graham et al., 2008).

The recent findings outline an increasingly clear, though complex picture of the relation between anger and pain. Also, the neurophysiological literature describes overlapping neural and endocrine mechanisms for both pain perception and anger

regulation, thus offering a strong support for this relation (Bruehl et al., 2009; Mollet and Harrison, 2006). In essence, it is not the emotion itself, but rather the strategies used to regulate it that interact in multiple ways with pain perception and modulation. Frequently expressing anger is associated with reports of increased pain severity via increased physiological responses to stress. However this relationship is moderated by various other factors, such as gender, state-anger, medical condition, opioid intake. Suppressing anger is also paradoxically increasing subsequent pain reports, suggesting that both regulation strategies, while useful in certain situations, need to be carefully balanced and used in combination with other strategies.

4.4.3 Depression and sadness

In Fernandez and Milburn's (1994) study of the relationship between distinct emotions and pain reports, sadness was identified as one of the negative emotions most closely related to pain, and the second most intense emotion experienced, after anger. However, sadness as a stand-alone emotion has not been studied in chronic pain, and research has focused on chronic pain-associated depression, which may be considered the clinical equivalent of sadness as a discrete emotion (Fernandez and Boyle, 2001).

Depression and chronic pain are often comorbid conditions. Moreover, depression is more frequent in chronic pain compared to other chronic conditions and development of depression is related to duration and severity of pain, number of pain locations, and frequency of pain breakthrough (Banks and Kerns, 1996; Fishbain et al., 1997). The simultaneous presence of chronic pain and depression affects negatively functional limitations, quality of life, and treatment utilisation and efficacy (Bair et al., 2003; Mossey and Gallagher, 2004; Arnow et al., 2006). The causal connection between depression and pain is however controversial and several alternative hypotheses have been proposed: depression is an antecedent of chronic pain, an immediate consequence, a predisposing factor (the *scar* hypothesis), a consequence mediated by psychological factors such as beliefs of life interference or decreased self-control (the cognitive-behavioural mediation model), or they both have a common pathogenic mechanism (Fishbain et al., 1997).

Depression as antecedent is mostly based on the psychoanalytic tradition and proposes that, for a patient for whom an organic cause has not been found, the chronic pain is a result of repressed depression; this hypothesis has been largely disconfirmed (Pincus and Williams, 1999). Other mechanisms have been recently proposed, such

as neglecting physical health and sleep problems, and several longitudinal studies indicate a higher risk of chronic pain onset for depressed individuals (Currie and Wang, 2005). Depression as immediate consequence, although largely supported (Fishbain et al., 1997), was an early approach lacking explanatory power as it did not address the mechanisms by which depression follows onset of chronic pain or increases in pain severity and therefore was replaced by more complex models.

The *scar* hypothesis (or the *vulnerability / diathesis-stress* model) is based on Beck's cognitive distortion model, Seligman's learned helplessness model and Lewinsohn's behavioral model of depression. It stipulates that premorbid psychological predispositions (such as negative schemata about the self, the world and the future; or the tendency to make internal, stable and global attributions; or restricted premorbid levels of instrumental activities and limited skills to obtain external reinforcers) are activated by stressful events related to pain: the symptom itself, the related impairment and disability, the secondary social and psychological losses and the interactions with the medical system. This activation leads to processing biases (such as overgeneralisation, personalisation, absolutistic thinking and catastrophizing), more frequent use of depressive attributional style, limitation in rewards and increase in punishing reinforcement, which maintain dysphoric mood and negative thought patterns (Banks and Kerns, 1996).

The cognitive-behavioral mediation model (Rudy et al., 1988) proposed that perceptions of reduced instrumental activities (life interference), and reduced control and personal mastery are necessary conditions for onset of depression in chronic pain patients. Support for this model was provided by mediation analyses using SEM methodology. The authors considered these results as evidence against the common pathological mechanism hypothesis, which relied mostly on neurophysiological data related to the stress response (with possible common mechanisms related to functioning of the hypothalamo-pituitary-adrenal axis, serotonin and norepinephrin; see also Blackburn-Munro and Blackburn-Munro, 2001, for a more recent review), but did not account for the low correlations between pain severity and depression.

In fact, instead of being a case of supporting one hypothesis in the detriment of another, the possibility of multiple causal factors intervening to various degrees in different populations is more likely. For example, several longitudinal studies reported significant (although weak) relationships between both initial depression and subsequent pain onset and vice versa (Magni et al., 1994; Currie and Wang, 2005). Turk et al. (1995) tested the cognitive-behavioral model in young and elderly patients and report stronger links between pain and depression in the elderly compared

with young patients. Within a sample of older adults, a longitudinal study found a moderating effect of gender, and not age (Geerlings et al., 2002). Averill et al. (1996) identified several demographic, pain-related and work-related correlates of depression in chronic pain: high depression was associated with lower education, single status, younger women and older men, pain duration, unemployment, working people planning future litigation and unemployed not planning litigation (work status, education, and marital status seem to be the most relevant predictors according to a stepwise regression model). Obviously, the impact of different factors depends on the medical condition related to chronic pain. For example, rheumatoid arthritis was more influenced by peripheral physical pathology, while other pain conditions involving changes in the central nervous system were possibly more sensitive to psychological and social input. These findings indicate the necessity of developing complex bio-psycho-social models addressing the depression-pain link (Campbell et al., 2003).

The study of the depression-pain relationship is encumbered by two main issues. First, many studies rely on the assumption that depression and pain are two distinct constructs. But most of the data pertaining to these hypotheses rely on either operational diagnostic criteria (such as the Diagnostic and Statistical Manual of Mental Disorders; DSM) or self-report measures (such as Beck Depression Inventory; BDI) which are both problematic when used in chronically ill population, due to overlap in symptoms, e.g. sleep problems and impairment in functioning (Banks and Kerns, 1996; Fishbain et al., 1997; Campbell et al., 2003)[12]. Some authors employed corrected cut-off scores for diagnosis instruments (Turk et al., 1995), but such recommendations haven't been widely adopted, therefore most current data related to the depression-pain link need to be treated with caution due to this criterion contamination (Pincus and Williams, 1999).

Second, depression is a complex and heterogeneous category. In chronic pain research, the term has been used to describe either mood, symptoms or syndromes, often leading to contradictory results regarding prevalence (Banks and Kerns, 1996). While the emotion of sadness is considered to occupy centre stage[13], depression includes a complex pattern of cognitions and affective responses. It is acknowledged

[12] Although other authors argue against eliminating such symptoms from a depression diagnosis in chronic pain and suggest considering them towards a diagnosis of reactive depression (Sullivan, 2001).

[13] Although depressed mood is not a necessary condition for the diagnosis of depression, anhedonia being the alternative condition, according to DSM-IV (Sullivan, 2001).

that other emotions are important in depression: primarily disgust (with its self-directed variants like guilt, shame and embarrassment), but also anger and anxiety (Power and Dalgleish, 2008)[14]. The models described above also highlight the significant role of cognition, but its interaction with emotional aspects in chronic pain-related depression is still largely unknown, as general depression models do not necessarily apply to chronic pain. Indeed, the presence of chronic pain and depression has been reported to influence depression symptoms differently, with little or no impact on loss of interest in activities and feelings of guilt, and higher impact on insomnia and sad or depressed mood (Ohayon, 2004).

This complexity does not justify the use of such an umbrella-construct in research, but rather focusing on specific components for which different psychological, social and physiological mechanisms might intervene. Studying sadness as a stand-alone emotion might provide different insights into these mechanisms. Recent experimental research on sad mood induction in healthy subjects suggests a role in reducing pain thresholds (thus increasing sensitivity) for acute pain (Wagner et al., 2009), while earlier research reports that depressed individuals actually have higher pain thresholds than healthy individuals (Dickens et al., 2003). In chronic pain, yet a different relationship might emerge, due to the pathophysiological and psychosocial changes involved; the existing evidence is inconclusive (Lautenbacher and Spernal, 2004).

4.4.4 Fear/anxiety

Fear is the third most reported basic emotion in chronic pain (after anger and sadness), according to Fernandez and Milburn's (1994) study, and also one of the negative emotions most closely associated with pain. Yet fear and similar emotional states such as anxiety, panic and worry have been studied much more than anger and sadness in chronic and acute pain. This is partly because they are prototypically used to describe response to physical threat, for which pain is an obvious signal, and emotional distress, with which chronic pain is associated in many ways, and partly because cognitive-behavioral research on anxiety disorders has been one of the main areas of scientific progress in clinical psychology in the last decades, from which many concepts and models have been adapted for chronic pain management.

[14]Yet information-processing bias experiments indicate that feelings of guilt and shame do not characterise chronic pain depressed patients, in contrast with depressed patients not suffering from chronic pain (Pincus and Williams, 1999).

The prototypicality of fear as a response to physical threat would lead to the conclusion that fear is invariably positively associated with pain, but research reveals a more complex picture. Part of the complexity stems from terminological issues. First, anxiety is used to describe the psychiatric disorder, the symptom present in many other emotional disorders, the emotional response, the mood/state, the personality trait, or even a coping strategy. Each of these phenomena can have a different relationship to chronic pain status variables. For example, there is a high comorbidity of chronic pain in panic disorder patients (48%, Kuch et al., 1991) and a high proportion of chronic musculo-skeletal patients meet the criteria for social phobia and post-traumatic stress disorder (Asmundson et al., 1996, 1998 as cited in Asmundson et al., 1999). A more recent general population survey also indicated high comorbidity between chronic pain and anxiety disorders, especially panic disorders and post-traumatic stress disorders (McWilliams et al., 2003). However in terms of sensitivity to acute painful stimuli, individuals suffering from anxiety and panic disorders are similar to healthy individuals (Lautenbacher and Spernal, 2004). When considered as a personality trait, anxiety (as measured by the Spielberger Trait Anxiety Inventory; STAI) is related to pain severity and disability (McCracken et al., 1996), coping strategies (Hallberg and Carlsson, 1998), and its relation to reports of pain intensity and unpleasantness and tolerance for acute pain is moderated by modality, gender (Jones et al., 2002), and attention focus (James and Hardardottir, 2002).

Second, fear, anxiety, panic and worry can be conceptualised as synonyms or distinct concepts. For example, anxiety can be considered as the anticipation of potential threat, while fear as a reaction to a present threat (James and Hardardottir, 2002). In experimental studies of acute pain in healthy individuals, induced fear (conditioning to electrocutaneous painful stimuli, or fearful images) was reported to reduce sensitivity to subsequent painful stimuli of a different modality, while anxiety (expectation of a painful stimulus not applied subsequently) either had no effect, or increased sensitivity to acute pain, depending on its relation to pain, attention processes and perhaps gender (Lautenbacher and Spernal, 2004; Kirwilliam and Derbyshire, 2008). In clinical settings however, fear and anxiety are used interchangeably (Leeuw et al., 2007).

Third, fear and anxiety can have a different impact on pain depending on whether they are about pain itself, or other events in the person's life. Trait and state anxiety unrelated to pain have no bearing on sensitivity to pain, but pain-related anxiety (such as dental pain), although it does not decrease thresholds, increases

ratings on pain intensity for the relevant body region (Lautenbacher and Spernal, 2004). Obviously, fear can stem from other chronic pain-related issues, such as the unpredictability of the condition and consequences of disability.

Fourth, fear has not been studied from an emotion perspective, but predominantly as part of a complex pattern (as with sadness and depression) which includes cognitive processes involved in its generation and perpetuation, as well as mechanisms of emotion regulation, and related behaviours, and no clear conceptual distinctions were made between them.

Most research has been based on the fear-avoidance model of exaggerated pain perception, first developed for chronic low-back pain (Lethem et al., 1983). In this model, fear of pain is a synonym of pain-related anxiety and represents the emotional response to current and future pain viewed as threat, for which the individual has two types of responses available: confrontation or avoidance. Depending on current life events, personal pain history, characteristic pain coping strategies and personality, individuals might chose predominantly confrontation strategies or avoidance. Confrontation is the adaptive response, as it leads to emotional responses synchronous with the sensory qualities of pain, and as the organic basis of pain subsides or reaches a plateau, so do the emotional response components (pain experience, behaviour and physiological responses). Avoidance is non-adaptive, as it decouples the affective and sensory components, and thus the individual can be increasingly emotionally affected by pain despite organic improvements in their back condition, leading to exaggerated pain perception and pain behaviours and physiology. In essence, this model is a reformulation of Fordyce's operant pain concept in cognitive terms. It has had an enormous impact on the CBT approach to pain management and is also reflected in the acceptance-based approach.

A first effort to distinguish the components of pain-related anxiety/fear was the development of the Pain Anxiety Symptoms Scale (PASS; McCracken et al., 1992), which separated cognitive, behavioral and physiological aspects into 4 subscales: somatic anxiety, cognitive anxiety (appraisals of cognitive interference during pain), fear (appraisals of negative consequences of pain) and escape/avoidance[15]. Another conceptualisation is fear of movement/(re)injury, as measured by the Tampa Scale for Kinesiophobia (TSK; Miller et al., 1991, as cited in Vlaeyen et al., 1995). Fear

[15]A different factor structure was identified by Larsen et al. (1997), with 5 factors labelled catastrophic thoughts, physiological anxiety symptoms, escape/avoidance behaviours, cognitive interference and coping strategies, while a 4-factor solution led to different item groupings, thus questioning the validity of the scale.

is here equated more with beliefs about pain causing injury and about the necessity to avoid physical activity and pathological somatic focus. Fear of work-related activities is yet another conceptualisation, as reflected in the Fear Avoidance Beliefs Questionnaire developed for back pain patients (FABQ; Waddell et al., 1993), which actually enquires about beliefs related to how work-related and general activities affect their pain. A fourth measure, the Fear of Pain Questionnaire (FPQ; McNeil and Rainwater, 1998), distinguishes between fear of severe pain, fear of minor pain, and fear of medical pain, and asks respondents to estimate how fearful they think they would be in a list of hypothetical events involving painful experiences.

All these measures have generated research indicating that fear of pain is associated with multiple health status indicators in chronic back pain and other conditions. Thus, fear of pain (PASS) was reported to be associated to disability and interference due to pain, controlling for emotional distress and pain severity (McCracken et al., 1992), to pain behaviours (McCracken et al., 1996), pain-related distress (Vowles et al., 2004), and increased non-specific physical complaints (McCracken et al., 1998), and was more characteristic of dysfunctional chronic pain patients as classified based on the Multidimensional Pain Inventory (Asmundson et al., 1997b; McCracken et al., 1999). Fear of work and activities (FABQ) was associated with work loss and disability in daily activities (Waddell et al., 1993; Fritz et al., 2001), although its usefulness in predicting poor outcomes was limited for work-related low-back pain (Cleland et al., 2008). Fear of movement (TSK) was related to measures of catastrophizing and depression and also to behavioral measures of avoidance (Vlaeyen et al., 1995), and was more associated to self-reported disability and behavioral performance, compared to negative emotionality (Crombez et al., 1999). An electronic momentary assessment study reported that attention to pain partially mediated the relationships between fear of movement and pain intensity as measured simultaneously (Roelofs et al., 2004). However recent longitudinal studies did not support the role of fear of movement in predicting future low-back pain outcomes (Sieben et al., 2005; Pincus et al., 2006). Also, fear of pain as measured by FPQ was related to behavioural measures of escape/avoidance in healthy individuals (McNeil and Rainwater, 1998), which in this case might be an adaptive response[16].

[16]It is also acknowledged that these associations may be partially due to content contamination between measures, for example between avoidance and disability, or anxiety and pain intensity, as they tap into the same behavioural and emotional phenomena (McCracken et al., 1996).

Another conceptualisation of fear in pain research is as an expression of anxiety sensitivity (AS). AS was adopted by chronic pain researchers from cognitive-behavioural theories of anxiety disorders. It is defined as the fear of anxiety-related bodily sensations due to beliefs about their harmful consequences, and was found to be associated to fear of pain and thus to pain-related avoidance (Asmundson and Taylor, 1996; Zvolensky et al., 2001), and to selective attention to pain-related information in chronic pain patients (Asmundson et al., 1997a). AS also increased reports of pain intensity and anxiety (but not physiological reactivity) following experimentally induced pain in patients with panic disorders (Schmidt and Cook, 1999). In healthy individuals, AS similarly increased reports of pain intensity in response to acute pain (Keogh and Birkby, 1999), and its relation to affective ratings of pain was mediated by pain-related interpretive biases (Keogh and Cochrane, 2002). Results are contradictory regarding its relation to decreased pain thresholds (Keogh and Birkby, 1999; Keogh and Cochrane, 2002). It was also related to lower levels of vitality, and poorer social and psychological functioning, controlling for demographic, work-related and pain-related variables (Plehn et al., 1998).

Greenberg and Burns (2003) highlighted the difference between fear of pain as a special type of phobia (of the painful stimuli as such) and AS as a more generalised vulnerability to various situations that generate anxiety-like symptoms, and set up a decisive experiment in which chronic pain patients were subject to a cold pressor task (to elicit pain anxiety) and a mental arithmetic task (to elicit social anxiety). In this situation, a phobia model would predict differences only in responses cold pressor task depending on fear of pain and AS scores, while the AS model would predict differences in both tasks. The results supported the latter model, as PASS scores accounted for significant variance of self-report and behavioural responses to both tasks, and most variance explained was common with AS scores[17].

However these models of fear-avoidance and anxiety-sensitivity are focusing less on the fear as a momentary emotional reaction (as distinct from anger and sadness, for example) and more on a wider meaning of fear which includes maladaptive cognitions about pain and pain avoidance. This made them more suitable as a framework to study the impact of life events, pain history, coping strategies and personality on chronic pain adjustment, than as models of emotion and pain. They also highlight the above-mentioned difficulty of separating the emotion of fear from its cognitive and behavioral correlates[18].

[17]The amplifying function of such a meta-emotion as fear of fear can be described as reflecting a dysfunctional emotion regulation mechanism.

[18]The issue of emotion-cognition-behaviour interactions is further detailed in Chapter 6.

The focus on momentary functions of fear/anxiety in chronic pain characterises another conceptualisation: the construct of 'worry'. It offers a more dynamic approach to pain-related distress, and also a theoretical link with its adaptive function at normal levels (Aldrich et al., 2000). Based on cognitive-behavioral research on rumination in sleep disorders and test anxiety, worry is central to the cognitive-affective model of the interruptive function of pain (Eccleston and Crombez, 1999).

The model places attentional processes center stage, defined as selection for action, and asserts that the selection of the pain signal from the multitude of competing signals in the environment is facilitated by properties of the signal (intensity, novelty, predictability, threat - and how it is perceived depending on personal characteristics such as habitual ways of processing threatening information, or somatic awareness), and the environment (e.g. emotional arousal properties, task difficulty). Once selected, pain interrupts the ongoing behaviour and motivates escape behaviours, competing with the motivation to continue the previously activated behaviour. Eccleston and Crombez (1999) view chronic pain as chronic interruption, a normal (not pathological) response pattern to continuous painful stimulation, for which high symptom reporting, depression and widespread avoidance behaviours are the natural consequences.

Another important consequence is an increase in worry: affect-laden, intrusive, threat-related rumination about pain and its consequences for the body and the self, which is actually a problem-solving effort focused on pain (Eccleston and Crombez, 1999; Aldrich et al., 2000)[19]. They describe the increased worry by 'vigilance' to threat-related information, which, together with hightened mental activity, should facilitate 'problem-solving' in situations with no immediate behavioral solution. In the context of chronic illness, worrying becomes a particularly difficult problem-solving effort, and it is at risk to become chronic itself, and thus dysfunctional, enhancing the distressful properties of the threat it tries to solve. Eccleston and Crombez (2007) describe this phenomenon as a perseverance loop, in which a person continues the misdirected problem-solving behaviour which increases hypervigilance to threat and the interruptive function of pain, which in turn amplify worry. Reframing the problem would potentially lead to a solution.

Evidence for the role of worry in chronic pain is accumulating. In a diary study (Eccleston et al., 2001), chronic pain patients described pain-related worries (about medical uncertainty, disability, pain experience or distress) as more distracting,

[19]In this framework, acceptance would be a redefinition of the problem-solving effort in terms of disability and distress, rather than pain.

intrusive and distressing than unrelated worries (about finances, relationships or self-presentation) experienced in a 7 days interval, and these characteristics were not related to general anxiety-related personality traits. Vlieger et al. (2006) reported similar worry (about relationships, confidence, future, work, finances) and problem-solving confidence levels for chronic pain sufferers requesting treatment in comparison to sufferers with no such requests, which supported the argument that worry is not a pathological phenomenon associated with health care use, but a natural consequence of continuous painful stimulation. Both worry and problem-solving reports were associated with reports of depression and catastrophizing, which the authors interpreted as suggesting that depression and catastrophizing are possible by-products of unsuccessful attempts to solve the difficult problem of chronic pain[20].

This model is by far more comprehensive than the previous models of fear and avoidance. However, it too defines worry as cognition (Aldrich et al., 2000, p. 466) and considers affective factors only as moderators of the pain's impact on attention. Eccleston and Crombez (2007) only tangentially mention the possible detrimental role of physiological activation in worry states. This suggests that a more emotion-oriented approach to chronic pain related fear/anxiety has the potential to bring more clarity, if applied in combination with cognition and behaviour related constructs.

4.4.5 Shame

Given the links between guilt and pain stipulated by psychoanalytic approaches, the dearth of research into this aspect can be considered a counter-reaction to the initial overemphasis. Two recent studies in medical anthropology addressed the issue of shame and stigma in chronic pain and brought forth aspects of living with chronic pain that are generally ignored (Werner et al., 2004; Jackson, 2005). Using the interview-based grounded theory approach, both studies depicted the stories of patients that have to cope not only with the illness, but with the distrust of others regarding the authenticity of their symptoms and with breaching others' expectations and social representations about illness behaviour. Patients are wary of being considered as "whining and complaining" and try to negotiate a strong and positive self-image. Fighting this stigma sometimes also implies refusing any suggestion regarding psychological causes, risk factors or treatments and looking for arguments to support the biological etiology of chronic pain.

[20]The relationships between these concepts are critiqued in more detail in the next chapter in relation to cognition.

Werner et al. (2004) interviewed 10 women with chronic muscular pain and interpreted their discourses in terms of negotiating credibility, and thus dignity and self-worth, according to normative expectations of the biomedical establishment which reflect moral rules. Interestingly, the positive discourse about personal strength to fight the stigmatising attitude of others was associated with expressions of disgust towards other women's talk about illness. This aspect is particularly relevant for theoretical views that hold disgust, shame, guilt, embarrassment, and blame as facets of the same basic emotion with an essential role in the transmission of social rules and regulating social interactions, and differentiate shame and disgust mainly by orientation towards self or other (Power, 2006; Power and Dalgleish, 2008).

Jackson (2005) discussed the chronic pain experience as a "liminal" phenomenon (i.e. an entity that exists between culturally defined categories) in relation to the Cartesian mind-body split that generally defines current medicine and causes much dispute in pain research and clinical practice. Chronic pain sufferers are seen as 'out-of-place' from several points of view: they need both medical and psychotherapeutic treatment, they use health care services and social and financial support without clearly belonging to one of the categories of physically ill or mentally ill, they are both people that need medication and people that are 'abusing' it in the sense that they need more of a substance that would be efficient for others in much smaller quantities. Stigma-related emotions such as shame and disgust are particularly relevant to the concept of 'liminality', as they regulate socially constructed conceptual boundaries (as physiological disgust reactions regulate the boundaries between physical body and the external world). Stigmatisation is increased by the invisibility of pain (inviting suspicion from others), the social disruption caused by pain (in terms of abusing social resources) and the esthetically displeasing nature of pain behaviour. Jackson expressed optimism regarding the new advances in neuroscience which would legitimise the experiences of chronic pain sufferers if applied in clinical practice. Until then, the current situation adds a complex array of emotions to the challenges of chronic pain, among which shame plays a particularly important regulatory role.

These two studies highlight the necessity of further exploring the role of shame in adjustment to chronic pain. On the other hand, their use of qualitative methodology points to the challenges and inherent limitations of studying shame quantitatively via self-report, as its role in regulating social interactions make it less accessible to conscious awareness via a simple and often decontextualised questionnaire item, and more accessible in an interview context.

4.4.6 Happiness/Joy

In chronic pain, research on negative emotions has been driven by the interest in understanding emotional distress associated with pain, and thus positive emotions have been rather neglected until recently. Fernandez and Milburn (1994) reported a negative relation between ratings of positive and neutral emotions (surprise, interest and joy) and the affective component of pain, but its relevance was overshadowed by the strong relation between negative emotions and pain. In the structure of the Multidimensional Pain and Affect Survey (Clark et al., 2003), positive affect (happiness) was conceptualised as one of the components of the well-being supercluster, and thus distinct from all other negative emotions in the emotional pain supercluster. Several studies of positive emotions in acute pain, predominantly from the dimensional perspective and in relation to attention, have reported mixed results for the support of the analgesic role of induced positive affective states, depending on the actual content of the emotion induction method (e.g. Bruehl et al., 1993; de Wied and Verbaten, 2001; Meagher et al., 2001; Kenntner-Mabiala et al., 2007).

Zautra et al. (2001) have applied their Dynamic Model of Affect (DMA) in chronic pain and proposed that positive emotions help diminish the impact of pain and stress on negative affect. This model suggests that the relationship between positive and negative affect changes depending on circumstances. While in everyday life individuals benefit from assessing the two types of affect independently, this differentiation is also taxing in terms of cognitive processing resources. Stressful circumstances, such as pain, increase the cognitive demands, which leads to less differentiated positive and negative affect. They also direct efforts towards attending to negative affect, thus diminishing the perception of distinct positive experiences. The model also predicts that higher levels of positive experiences during these times also lead to less negative affect, and that individual differences in the ability to differentiate emotional experiences (also known as "mood clarity"; Salovey and Mayer, 1990, as cited in Zautra et al., 2001) influence the degree of differentiation during stress.

Zautra et al. (2001) tested these predictions in a hierarchical longitudinal design in order to test both intraindividual fluctuations of negative affect as a function of pain and positive affect and interindividual differences in all respects depending on mood clarity. As expected, weekly negative affect levels were significantly related to weekly pain, mean pain, and weekly decreases in positive affect. Importantly, increased positive affect was associated with more differentiation between pain and negative affect (thus less negative affect when pain was more intense), and mood

clarity was related to more differentiation between positive and negative affect, confirming the DMA predictions. These relationships identified on a sample of arthritis patients were partly replicated on a group of fibromyalgia patients (except the mood clarity - positive affect interaction), with different measures of affect and different intervals (weekly versus 3 times a day). Zautra et al. (2005) confirmed these results, and also reported a similar relationship between stress and positive affect; in addition, increases in weekly negative affect (but not pain levels or positive affect) predicted greater pain on the subsequent week, and pain increases were more characteristic of people with higher average negative affect and lower positive affect. They interpreted these results as evidence for a sustained cycle of pain and negative affect, modulated by positive affect. Strand et al. (2006) replicated the results on a sample of Norwegian rheumatoid arthritis patients and reported the same moderating effect of positive affect on the relationship between weekly ratings of most intense pain and negative affect (but not weekly average or lowest pain, or weekly interpersonal stress reports).

The DMA approach is a comprehensive model of the role of positive emotions in chronic pain, with promising supporting evidence. Zautra et al. (2001) also discussed the neurophysiological mechanisms supporting these relationships and noted the importance of endogenous opioids and the oxytocin modulated hypothalamo-pituitary-adrenal axis in underlying down-regulation of the stress response and reinstatement of positive mood states. There are however two notable limitations that suggest further research directions.

First, the model does not focus on pain levels, but on negative affect as a dependent variable, thus interpreting the relationships in terms of the effects of pain on emotional distress and thus psychological well-being. The simultaneous (weekly or daily) reports of pain, negative affect and positive affect can be subject to different analyses, either to assess the role of pain levels in moderating the relationship between positive and negative affect, or the role of negative affect levels in moderating the pain - positive emotions link[21]. This highlights the necessity of longitudinal lagged designs to test the causal order, fact acknowledged by Zautra et al. (2005), and also the necessity of including other external variables that might impact these reports, such as characteristics of the social and physical environment that might facilitate elicitation of negative or positive experiences. Also, causal relationships

[21]These alternatives are important especially given the issues related to measurement: self-reports of one construct are inevitably contaminated with assessments of other related constructs as discussed in Section 2.7.3. Strand et al. (2007) performed all three analyses, but did not discuss their implications for the DMA approach.

would need to be supported by experimental manipulations, not only clinical designs.

Second, the dimensional approach might not be the most suitable for framing this model in relation to possible clinical interventions. Zautra et al. (2005) considered their results as support for clinical interventions aimed at "enhancing individuals' ability to process affect with greater complexity" (p. 219), mentioning mindfulness meditation among the available approaches. Thus, mechanisms described as acceptance and awareness in ACT are described as increase in "emotional complexity" within the DMA approach[22]. This theoretical link suggests the possibility of measuring progress in such interventions not by decreases in pain and negative emotions and increases in positive emotions, but by lower correlations between reports of painful, negative and positive experiences. In this context, a differentiation between positive and negative might not be the right level of complexity. As discussed in a previous section, discrete emotions labels are more suitable than valence-based labels in guiding the individual's adaptation to categories of life events. Thus, categorical models might be more appropriate to assess the individual's ability to distinguish between the most suitable course of action to a specific stimulus (be it pain, conflict, loss, danger, affiliation needs etc.). Using different labels (and appraisals) might increase one's chances to select more appropriate responses to the various situations one is confronted with.

4.4.7 Emotion regulation and interactions between emotions

The research on the impact of emotions in chronic pain reviewed in the previous subsections has already suggested the importance of emotion regulation (ER) strategies (e.g. anger regulation). Moreover, it is acknowledged that emotions often are coupled (Power and Dalgleish, 2008), and chronic pain seems to be no exception. For example, Poleshuck et al. (2009) found that chronic pain patients suffering from depression report more anxiety and psychosocial stress than non-depressed patients, and the severity of depression is associated with both these characteristics. Means-Christensen et al. (2008) reported a close relationship between symptoms of depression, anxiety and pain. Zautra et al. (2007) showed a complex pattern of interrelations in which previous episodes of depression predispose rheumatoid arthritis patients to increased pain during induced stress, while their positive emotional experiences during this induction play a protective role, decreasing pain especially

[22]Commitment to value-based action is thus corresponding to accessing resources for positive emotional experiences despite the negative affect associated to pain.

in previously depressed individuals. Smith and Zautra (2008) described a complex interplay between depression and anxiety levels influencing weekly changes in pain reports in patients with osteoarthritis or rheumatoid arthritis via changes in weekly positive and negative affect[23].

All this difficult-to-navigate emotional storm has two main sources: the negatively-valenced emotional-motivational component of pain which constantly urges escape and the competing motivations generated by the individual's goals and aspirations which may increase or counteract the effects of continuous painful stimulation (Eccleston and Crombez, 1999; Hamilton et al., 2004). Research on ER focuses on the interplay between these two major motivational forces.

ER has been operationalised in chronic pain either as an individual trait or as a dynamic process. Among the trait-like descriptions, alexithymia has been the most widely researched, mainly in cross-sectional designs. In contrast, ER as a process has been studied in longitudinal diary studies. Both approaches are reviewed next.

Alexithymia refers to a general deficit in emotion regulation, characterised mainly by difficulties in differentiating emotional states between each other and from bodily sensations, and a disinclination to daydream and externally-oriented thinking, which lead to difficulties in identifying and communicating feelings. A frequently used measure is the Toronto Alexithymia Scale (TAS; Taylor et al., 1990, as cited in Millard and Kinsler, 1992).

Early studies indicated that chronic pain patients report higher levels of alexithymia compared to healthy subjects and also to patients seeking treatment for other conditions, such as nicotine-dependency or obesity (Lumley et al., 1997). Alexithymia scores were also associated with scores of self-efficacy, depression, catastrophizing, affective pain intensity and disability, but not to sensory pain intensity; its relationship with affective pain intensity was mediated by depression scores, and it relation to disability was not significant after controlling for either self-efficacy, depression or catastrophizing (Lumley et al., 2002).

It is important to note that research on alexithymia has been focused on identifying psychopathological aspects linked with chronic pain. For example, Lumley et al. (1997) considered alexithymia "an unique constellation of personality traits that may predispose to chronic pain and other disorders" (p. 163), based on some small

[23]However, research on emotion interaction is encumbered by measurement overlap between concepts given their broad definitions, the increased symptom reporting in some respondents and the difficulty to distinguish discrete negative emotions that are part of pain-related distress.

correlations between alexithymia scores and some clinical scales of the Minnesota Multiphasic Personality Inventory (MMPI). Mehling and Krause (2005) suggested that alexithymic individuals might ignore early somatic signs and thus be at risk for pain increases and based their proposal on the fact that alexithymia is considered a stable trait and therefore unlikely to be influenced by recent changes in health status. This approach assumes that the measures of alexithymia assess the same psychological phenomenon in all contexts and for all respondents. In the light of previously reviewed research on the affective components of pain and the close relationship with sensory aspects, another possibility can be advanced: in chronic pain patients, alexithymia measures might tap into the difficulties they face in keeping emotion and sensation separate in the face of continuous painful stimulation and associated stressful events, and their increased or decreased focus on external events might be a strategy to cope with the condition. These difficulties may be apparent only in the emotion differentiation and externally-oriented thinking subscales, therefore requiring separate analyses.

Several studies raised questions regarding the utility of TAS total scores in chronic pain. Millard and Kinsler (1992) reported moderate reliability of the total score, and no or low correlations with measures of disability, pain intensity, and distress, in addition to the lack of significant positive correlations between the 3 subscales identified, which does not justify the use of a total score. Cox et al. (1994) reported differences between alexithymic and non-alexithymic chronic pain patients only in the number of words used to describe pain. Sayar et al. (2004) found differences between fibromyalgia, rheumatoid arthritis patients and healthy controls only in the subscale measuring difficulties in differentiating emotions[24]. Also, only this subscale was associated with one year prevalence of low-back pain (Mehling and Krause, 2005)[25], and with affective qualities and tolerance of acute pain, distress and illness behaviours in fibromyalgia patients (Huber et al., 2009)[26]. Glaros and Lumley (2005) found that temporomandibular disorder patients differed from no-pain controls only in their emotion differentiation and externally-oriented thinking scores, the former overlapping with depression scores, and only the latter being associated with pain severity. Lumley et al. (2005) reported that only the emotion

[24]Rather than interpreting this difference as a characteristic psychopathological feature of fibromyalgia, I would consider it as reflecting the uncertainty of the medical condition increasing the difficulty of emotion regulation, in comparison to the more clear and socially-acceptable physiological mechanism behind rheumatoid arthritis.

[25]Longitudinal relationships with health outcomes are controversial (Mehling and Krause, 2005, 2007).

[26]The relationship with sensitivity to experimentally induced pain are also controversial (Huber et al., 2009).

differentiation subscale is systematically associated to pain severity, and only in African Americans, not in Caucasians; the difference was interpreted in terms of the role of cultural norms, frequency of traumatic events and communication patterns regarding negative emotions.

These results highlight the difficulties of operationalising ER as a general trait, and suggest a more contextual and delimited approach. This is attempted by dynamic views of ER, based either on the distinction between appetitive motivation and harm avoidance motivation (Hamilton et al., 2004) or on a basic emotions approach. For example, Connelly et al. (2007) inferred ER from daily changes in intensities of negative and positive affect in patients with rheumatoid arthritis and showed that both daily negative and positive affect regulation during the prior 1-day period predict reports of current levels of pain, controlling for prior day's pain. Paquet et al. (2005) measured positive emotions, anger, anxiety and depression in a momentary assessment design in hospitalised elderly and defined ER as the maintenance or recovery of positive emotions and decrease in negative emotions over a specific episode; they also found support for the hypothesis of an association between ER (especially of anxiety) and subsequent pain reports.

Focusing on either a general trait or on short-term changes in affect means missing potentially important ER mechanisms which function at a medium-to-long term level. For this purpose, it is important to distinguish between different strategies based on their impact on emotional life. As mentioned in Subsection 4.2.2, a classification based on their potential consequences and intra- or interpersonal resources accessed (Phillips and Power, 2007) is potentially useful in studying ER also in the chronic pain context.

4.5 Conclusion

Emotion is a core component of the pain experience. The examination of its role in chronic pain has followed mainly a basic emotions approach, complemented by a focus on emotion regulation and the dynamic relations between emotional states. In addition to the distinction between sensory and affective pain perception, studies have usually focused on one basic emotion, mostly on anger, sadness (as core emotion in depression) and fear, with less emphasis on shame/guilt and positive emotions. The study of emotion is encumbered by measurement difficulties (similarly with pain and CPA), especially in self-report, due to the loose connections

between emotion components (physiological, experiential, behavioral) and to the intersubjective nature of emotional experience as communicated via language.

The research reviewed in the present chapter highlights the importance of emotion in adapting to chronic pain, but also the necessity to adopt an integrative viewpoint. Not only the distinct emotions have dynamic relationships, but emotions are also intrinsically linked to emotion regulation, cognition and behaviour. In the next chapter, I will review the research related to the role of cognitive factors in chronic pain, focusing on illness perceptions.

Chapter 5

Illness perceptions

5.1 Introduction

The previous two chapters have focused on behaviour and emotion. This chapter focuses on cognition. It is already self-evident that there are no clear distinctions between these domains, and cognition has already been discussed in relation to many theories and concepts addressing acceptance and discrete emotions. This chapter will complete this as yet fragmentary picture by reviewing research which frames the link between psychological factors and pain in terms of cognitive constructs. In doing so, the wide areas of overlap with behaviour and emotion will become even more obvious.

A construct currently acknowledged as one of the most promising in this area is that of illness perceptions (IPs; also termed illness representations, cognitions, schemata, models, prototypes or beliefs). I will first describe the theoretical foundations of this concept: the theoretical basis and characteristics of the self-regulatory model (SRM) and its measurement and application in other health areas. I will then review existing research on the role of IPs in chronic pain and situate it into the wider context of cognitive approaches to pain. I will end this chapter by presenting several critical comments regarding the interpretation and limitations of current applications of the SRM.

5.2 The self-regulatory model

5.2.1 Theoretical foundations

The SRM is a model of decision-making in health contexts based on a model of cognition and emotion: the parallel processing model. Based on earlier models

of response to threat in attitudinal and behavioral change, the model stipulates a relative independence of emotional (fear) and cognitive processing of the information considered for decision-making (Leventhal, 1970, as cited in Leventhal and Scherer, 1987), with emotional effects on behaviour being short lived without the support of cognition, i.e. an action plan (Leventhal et al., 1997). Thus, the origins of the SRM are actually closely related to research on fear responding with direct application to health contexts, which also led to the development of fear-avoidance models of chronic pain, although the two directions developed separately in chronic pain research.

The parallel processing model subsequently evolved at the level of emotion theory into the perceptual-motor model of emotion, which proposes that emotions are the result of the interaction of multiple components, structured hierarchically on three levels: sensory-motor, schematic and conceptual. The sensory-motor level includes innate expressive-motor programmes which function automatically and generate the organism's earliest emotional responses, with interpersonal communication as one of the primary functions (such as in infant-carer interactions). These automatic responses are essential for associative learning, which further develops the schematic level, also functioning automatically and consisting of schemata of emotional episodes: concrete representations of perceptual, motor and subjective components of reactions to different stimuli. These schemata of varying complexity and generality further structure the processing of subsequent events, by connecting them with expected immediate consequences. The conceptual level develops on the previous two levels as information processing becomes propositionally organised and situates emotional responding in a longer-term context related to the concept of self and the external environment; it also participates in regulating emotional experiences activated by the other two levels, by controlling activation and motor responses. The interaction between these levels of processing becomes increasingly complex as the individual develops, however a certain degree of independence remains at any age (Leventhal and Mosbach, 1983; Leventhal and Scherer, 1987).

This model is actually one of the multi-level models of emotion that built on the Zajonc-Lazarus debate regarding the automatic versus cognitive elicitation of emotion (see Chapter 4). It complements Scherer's component process model (CPM) of emotion, which focuses on the content of emotion generation in the interaction of these processing levels. More specifically, the CPM builds on previous appraisal-focused views of emotion and stipulates the continuous operation of several types of stimulus evaluation checks (SECs), which process the external stimuli on criteria of

novelty, pleasantness, goal significance (relevance, expectation, conduciveness, urgency), ability to cope (causation, control, power, adjusting) and compatibility with internal and external standards. Depending on the results of these SECs, qualitatively different emotions can emerge. This model accounts both for the distinctions between the most common emotion categories (similar to basic emotions views) and for the existence of the wide variety of non-prototypical emotional states. Ontogenetically, the model stipulates an increase in the complexity of SECs allowing for an increasingly complex emotional life (Leventhal and Scherer, 1987). The authors propose that all SECs operate at all three levels of processing and refer to different but similar contents at each level. This appraisal-focused aspect of the theory is essential in understanding the self-regulatory model, as the content of the illness representations (the most developed element of SRM) is actually very similar to the content of emotion appraisals, and the description of the "Prototype Assembly and Appraisal Checks" in the complex interplay between emotion and cognition in health decision-making is clearly a development of the SECs.

In addition to its complementarity with Scherer's component process model and its solid grounding in cognitive and social psychology, the SRM also has wide areas of overlap with other health behaviour theories such as the Transactional Stress and Coping Model (TSCM), the Health Belief Model (HBM), the Theory of Reasoned Action (TRA) or the Theory of Planned Behaviour (TPB). In essence, these models attempt to explain a wide range of behaviours from prevention to self-management of chronic illness with the help of various cognitive variables, among which cognitive appraisals play a central role. While HBM, TRA and TBP are considered intrapersonal theories, the TSCM and SRM are interpersonal, as they include also the external social influences as explanatory variables. A brief description of these models will clarify the common elements and thus place SRM in the wider context of health psychology.

The Health Belief Model (HBM) stipulates that performing a certain behaviour is a function of perceived threat (perceived susceptibility/vulnerability to the related health risk and perceived severity of the potential consequences) and response selection based on perceived costs/barriers and benefits of alternative actions. Other important factors are the presence of certain cues for action, and the belief in the personal ability to perform that action (self-efficacy). Decision-making is influenced by other sociopsychological factors indirectly, via the above-mentioned parameters (Janz et al., 2002). The Theory of Reasoned Action (TRA) describes the intention to perform a behaviour as a result of attitudes (beliefs that a behaviour is

associated with specific outcomes, and evaluations of behavioural outcomes) and subjective norms (beliefs about whether people approve of a certain behaviour and motivation to comply with these norms). The Theory of Planned Behaviour (TPB), actually an extension of TRA, adds to these determinants the perceived behavioural control (control beliefs and perceived power, i.e. whether control is likely and effective) (Montao and Kasprzyk, 2002). TRA and TPB differ from HBM by the accent they place on norms and attitudes and by the attention given to the behaviour as such, as opposed to the related threat; they all have in common with SRM the focus on cognitive assessments of the situation and possibilities of action, and the important place given to control. The SRM however is distinct by the equal accent given to emotion and importance of situational stimuli.

The Transactional Stress and Coping Model (TSCM) describes stressful experiences as person-environment transactions. An external event (such as a health threat) generates initially evaluations of its significance in terms of susceptibility and severity, relevance for the person's goals, and possible causes (primary appraisals), and also evaluations of the person's ability to change the situation, to manage one's emotions and of the possible effectiveness of these efforts (secondary appraisals). These initial assessments determine the attempts to manage the situation both as problem management (problem-focused coping) and as emotional regulation (emotion-focused coping)[1], which are subsequently assessed based on their effects compared to expected effects. For changeable stressors, problem-focused coping would be more adaptive, while emotion-focused coping would be more suitable for unchangeable stressors, although coping flexibility has been found to be important in many health situations (Wenzel et al., 2002). Part of the TSCM appraisals, such as susceptibility, severity and personal abilities to control are also present in the other models mentioned above. But while in the other models the final end is performing the targeted behaviour, in the TSCM a wide variety of behaviours can be performed and are assessed on the basis of their effectiveness in reducing the influence of the stressor/threat. Also, the stressor itself influences the outcome by interacting with the person's perception and action, thus adding a new layer of complexity. Among these other models, the SRM is most similar with the TSCM, but adds to it several other levels of complexity.

[1]This aspect of the TSCM stimulated the body of research which further developed into the now blooming field of emotion regulation (Gross, 1999).

5.2.2 The model

Drawing from these multiple influences and existing research on treatment adherence, the SRM represents an elaboration the initial parallel processing model of health threats. In essence, it focuses on common sense representations of such threats and their relevance in selecting strategies to cope with chronic illness (e.g. treatment adherence behaviours). It describes a continuous, self-regulating process with three stages: interpretations of the current situation (i.e. symptoms), leading to selection of coping procedures, followed by assessment of the coping outcomes. Consistent with the parallel processing model, the SRM stipulates the existence of two components: an emotion/distress-focused and an 'objective' assessment.

Both emotional and cognitive interpretations happen at two parallel levels, a concrete (schematic) level of symptom-based schemata, and an abstract (propositional) level where the illness is labelled and concrete appraisals combine to form more complex representations. Specific targets for coping are set at both levels, which can function independently and often dissociate, with the concrete appraisals being more salient for ongoing coping. Assessment of the coping outcomes and decision-making for further coping procedures are also performed at both cognitive and emotional levels, with various heuristics employed, some specific to health-treat information, such as the symmetry rule (linking experienced symptoms with an illness label), the stress-illness and age-illness rules (categorising symptoms as stress related or age related), or the affect heuristic (believing negative emotions increase disease vulnerability). Establishing a coherence between these levels is essential for adequate management of chronic illnesses and individual efforts of integration may be impeded by biased evaluation of the outcomes of the coping procedures used (Leventhal et al., 1992; Leventhal, 1993; Diefenbach et al., 2008).

Based on research on a previous similar model (the common sense model of illness representations; Leventhal, Meyer and Nerenz, 1980, as cited in Leventhal et al., 1992), five attributes of the potentially threatening stimuli are distinguished at the cognitive level: identity, causes, timeline, consequences and controllability[2]. Identity refers to the label attributed to the stimulus, and its perceived sensory properties, the causes refer to antecedents of illness onset, the timeline is described in terms of imminence and rate of change, the consequences can be expected at many levels, from physical to socio-economic, controllability refers to both personal

[2]Some other structures have been reported for specific conditions, such as Chronic Fatigue Syndrome, Addison's disease (Heijmans and de Ridder, 1998), and Coronary Artery Disease (Hirani et al., 2006), suggesting that the structure of IPs might be partly disease-specific.

abilities to control or cure and others' potential influences. These attributes are not independent, and three distinct patterns have been identified, acute, cyclic and chronic, characterised by specific sets of attributes (Leventhal et al., 1992; Leventhal, 1993; Weinman et al., 1996). The emotional responses can be elicited by the pain and discomfort of the symptoms as such, by the identity and consequences of the illness as it is interpreted by the individual, by appraisals of the coping outcomes, or by external events (Cameron et al., 1993).

Leventhal (2008) describes a complex process of interaction between cognitive and emotional elements, guided by Prototype Assembly and Appraisal Checks (PCs) which constantly assess the flow of perceived somatic changes. Primary PCs (assessing sensory pattern, location, duration, intensity, trajectory, and control, in addition to PCs eliciting emotions) generate an initial illness prototype, which might be labelled as acute and non-threatening, or as signs of normal aging, or of a life-threatening condition. Secondary PCs update the illness prototype and related emotional experience both intrapersonally (in checking results of coping procedures, or in relation to the self-image, for example) and interpersonally (in interactions with medical professionals, or by social comparison).

The continuous, cyclic process of interpreting the health threat, coping with it and assessing the outcomes has been described in terms of four phases in the context of health care use: self-evaluation (assessment of a somatic change), illness (individual attempts at controlling the symptom), utilisation (from the decision to seek help to the medical encounter) and diagnostic (the doctor's label). The authors acknowledge the difficulty to describe the complex temporal dynamics of the above mentioned attributes, but also underline the SRM's potential for informing effective interventions by focusing on both the schematic and conceptual levels and on reaching a coherence between the levels of representation (Leventhal et al., 1992).

The SRM explicitly distinguishes itself from other health behaviour models (briefly described in the previous subsection), which are considered incomplete (Leventhal et al., 1997). Its proclaimed theoretical advantages are its comprehensiveness, the focus on process rather than on preconditions of adequate behaviour, the equal weight given to both emotional and cognitive processing of health threats, and the efforts to integrate these levels. Moreover, the SRM situates the health-related decision-making in the context of wider intrapersonal and social factors and underlines the necessity of a coherent relationship at these levels also (Leventhal et al.,

1992). However the practical applications of the SRM have focused mainly on beliefs and representations than on emotional components, and the integrative efforts are still in their infancy (Diefenbach et al., 2008).

5.2.3 Measurement

Research on illness perceptions has used predominantly qualitative methodology and purpose-built questionnaires. While these methods have the advantage of identifying the content relevant for the specific condition or patient, the drawbacks are the resources required, the subjectivity of interpretation and difficulties in generalisation. Weinman et al. (1996) developed the Illness Perceptions Questionnaire (IPQ), which focuses on the five attribute categories, and includes the possibility to add content relevant for specific conditions. In IPQ, identity scores represent the number of symptoms associated with the illness, timeline scores focus on beliefs regarding the illness duration, consequences scores indicate the extent to which the person feels affected by the illness on multiple levels (personal, social, economic), cure/control scores mark the degree of perceived chances for improvement and perceived influence on the illness outcome, cause-related items can be treated separately or combined in categories (such as external versus internal). The validation of IPQ was performed on populations with various diagnoses (myocardial infarction, chronic fatigue syndrome, rheumatoid arthritis, pain, diabetes, etc.) and showed good reliability and acceptable concurrent, discriminant and predictive validity.

A recent revised version of IPQ (IPQ-R; Moss-Morris et al., 2002) split the control subscale into personal control and treatment control and included three additional subscales addressing cyclical timeline perceptions, illness coherence and emotional representations. The cyclical timeline scores reflect the perceived variability of illness-related symptoms. Illness coherence measures the perceived degree of understanding of the illness. Emotional representations focus on six affective responses to illness: depressed, upset, angry, worried, anxious and afraid. A short form recently developed (BIPQ; Broadbent et al., 2006b) offers the possibility of a rapid assessment suitable to multi-measure studies. BIPQ consists of 9 questions, each assessing a single attribute of the illness perception: consequences, timeline, personal control, treatment control, identity, concern, understanding, emotional response and causation.

5.2.4 Research in other health conditions

The SRM, especially since the development of the psychometric instruments, has been applied to various conditions, such as heart disease, rheumatoid arthritis, cancer, psoriasis, diabetes, and research has confirmed relationships between IPs and several measures of patient functioning, such as coping, mood, functional adaptation and treatment adherence (Moss-Morris et al., 2002). A comprehensive review of this substantial literature is beyond the scope of this chapter. I will only outline the diversity of research designs and health conditions studied with several examples.

The wide majority of studies have focused on the IP component of the SRM, measured by either interview, purpose-built questionnaires, or a variant of IPQ, usually with a cross-sectional design. For example, Scharloo et al. (1998) investigated the relations between IPs, coping and subjective and objective measures of health status in patients with chronic obstructive pulmonary disease (COPD), rheumatoid arthritis (RA), and psoriasis. They report a complex pattern in which subjective health is related to different objective health indicators, IPs and coping, depending on the condition and health domain (e.g. physical functioning is related to medical indices and passive coping in patients with COPD, but only with identity IPs in psoriasis patients, while social functioning was related to identity IPs in all groups, but also with timeline IPs in psoriasis patients, and with control IPs and passive coping in RA patients). Beliefs about the necessity of medicines and concerns about long-term effects of taking them showed significant associations to reported adherence in various chronic illness groups (asthma, renal, cardiac, oncology, HIV), indicating that patients generally perform a cost-benefit analysis for treatment decisions (Horne and Weinman, 1999; Horne et al., 2004). However, IPs had only a minor contribution in adherence to cholesterol-lowering medication in patients with hypercholesterolaemia when controlling for other health-related variables, and might be rather a consequence of effective treatment enhanced by adherence (Senior et al., 2004). Also, IPs are weak predictors of health behaviours in secondary prevention of coronary heart disease and only medication beliefs are comparatively more related to adherence (Byrne et al., 2005).

Longitudinal studies have shown mixed results depending on the health condition studied. For example, a substantial body of research addressed the predictive role of IPs in rehabilitation following MI. There are significant associations between IPs at admission to hospital and ulterior performance in various rehabilitation

domains, controlling for other relevant demographic and illness severity variables (Petrie et al., 1996; French et al., 2005). The role of IPs in attendance to cardiac rehabilitation programmes seems to be moderated by the time interval between hospitalisation and IP assessment and any interventions addressing IPs during this interval; generally, attendance is associated with perceiving more symptoms, higher control, more consequences and higher coherence of the condition (French et al., 2006). In patients with psoriasis, identity IPs and coping strategies predicted significant (albeit small) percentages of the variance in some patient functioning variables at 1-year follow-up, while control and identity IPs predicted use of medical services, controlling for patient functioning at initial assessment and other relevant variables (Scharloo et al., 2000).

Several studies have designed interventions targeting IPs. For example, Petrie et al. (2002) and Broadbent et al. (2009a,b) reported positive results of randomized controlled trials of brief in-hospital interventions for changing MI-related IPs with significant positive impact on return to work and reported symptoms at 3 or 6-month follow-up, and also on spouses' IPs and distress.

Even if the majority of studies focused on IPs after medical diagnosis, the SRM was also applied to various other research questions. For example, it has lead to important advances in understanding the process of decision-making from initial symptom perception to accessing health care services. Cameron et al. (1993) studied middle-aged and older adults (with a mixed qualitative and quantitative design), and described a process of decision-making starting with identification of atypical symptoms and leading to the development of a representation of the condition as threatening and beyond the personal control, in parallel with an emotion-focused process of assessing the associated distress and procedures of coping with it, which together with external advice to seek help determine the decision to use health care services. Horne et al. (2000) interviewed patients admitted to hospital for MI and described the delay to seek medical help as a function of the mismatch between experienced symptoms and symptom expectations for a MI, with the majority of respondents experiencing at least a symptom they did not previously associated with MI.

Based on this increasing body of research, the SRM has been acknowledged as a very promising paradigm for research in psychosomatics, outperforming coping approaches[3] in the study of adherence, emotional distress and illness-related disability

[3]Even if research has consistently confirmed a lack of statistical mediation via coping of the relation between IPs and various health outcomes, it is important to note that this statistical

(Weinman and Petrie, 1997). Most importantly, its comprehensiveness and solid grounding on observation of self-management processes in clinical and community settings make it a suitable framework for developing sound theory-based interventions for self-management, which might prove more cost-effective than current practitioner-delivered behavioural interventions (Leventhal et al., 2008). Nevertheless, especially due to its complexity, the SRM is difficult to test in its entirety. The authors consider it rather an open, flexible framework for the development of more specific models (Leventhal et al., 1992).

5.3 Illness perceptions and pain

Leventhal (1993) considered pain research as a suitable application for the study of parallel processing, as the distinction between the emotional/motivational network and the sensory network is similar to parallel processing of perceptual-cognitive and emotional information, characterised by both complex interaction and possible dissociation. In an early formulation of the parallel processing model of pain distress, Leventhal and Everhart (1979) underlined the automatic nature of both sensory (stimulus location, duration, intensity, attributes) and emotional (distress) processing of pain and the role of attention and context in the separate or blended conscious awareness of sensory and emotional components of pain[4]. Consistent with the perceptual-motor model of emotion and with the appraisal literature, meaning is considered central to the emotional component of pain: pain schemas and beliefs related to the threat significance of a painful stimulus (situated at a schematic and conceptual level, respectively) may amplify or diminish the emotional response to pain and the subjective perception of its intensity, which happen primarily at the perceptual-motor level.

According to this model, pain schemas influence pain perception via action on attention mechanisms which influence the conscious awareness of stimuli, and via schematic integration of various inputs (sensory and contextual information) and

relation between questionnaire measures does not actually test the temporal sequence between illness representations, coping procedures and assessment of coping outcomes stipulated by the SRM. IPs and outcome measures are unsurprisingly more associated than coping, as both are appraisals of the impact of the condition, albeit from different perspectives, while coping refers to the frequency of performing a series of related behaviours. This is one of the inherent difficulties of testing the SRM with quantitative data not tailored to the dynamics of individual situations.

[4]As detailed in Chapter 2, the parallel yet interacting processing of sensory and emotional components is supported by recent findings regarding the neurophysiological mechanisms of pain perception.

outputs (motor responses). Importantly, they also determine which discrete emotions (distress, fear, guilt, anger, even pleasure) can be associated with the sensory component of pain, possibly also depending on the sensory characteristics of the stimuli (intensity, duration) and on the social context (p. 282). At the conceptual level, specific pain-distress rules may function: the magnitude rule, the pain-injury rule, the belief in the effectiveness of distraction or the belief that pain-distress is visible in external behaviour. The model has been successfully applied to clinical interventions for reduction of acute pain associated to medical procedures.

Given its comprehensiveness, its solid basis on emotion theory, and its focus on emotion-cognition interactions, it is surprising that the parallel processing model has not lead to an equal focus on cognition and emotion in chronic pain research. The emotion component has been mostly neglected, while "illness perception" has been considered a representative concept for cognitive approaches to health psychology (Weinman and Petrie, 1997). In chronic pain, illness perceptions follow a tradition of concepts aimed at measuring the 'maladaptive thinking' associated with increased pain reporting. I will therefore review first a few similar perspectives in order to situate IPs in the wider context of cognitive approaches to pain.

5.3.1 Cognitive-behavioral theory applied to chronic pain

Cognitive approaches to chronic pain are essentially based on behavioral models of the relationship between external contingencies and behaviour, and add to it the focus on the role of information processing variables. In their seminal book, Turk et al. (1983) organised the common elements of the various CBT approaches into three stages of the therapeutic process - problem assessment (from the patient's perspective) and reconceptualisation, acquiring and consolidating skills, and the application and follow-through phase - and proposed several applications of CBT to chronic pain based on these common elements, situated in the wider context of health psychology (then labelled behavioral medicine).

The first stage is fundamentally related to IPs and the SRM, as it focuses on the patient's current cognitive model of his/her condition (and treatment), and thus on issues of treatment adherence and resistance. It also involves a transformation of the often undifferentiated and overwhelming problem formulation into a series of specific, manageable problems, supported by two main activities: adopting new terms and frameworks for analysis, and collecting data (by narrative or record-keeping) to guide the analysis process. From a SRM perspective, this stage can

be described as a cyclic process of reconstructing the patient's IPs, applying the corresponding coping procedures and evaluating coping outcomes, aiming for coherence between the patient's and therapist's IPs about the condition[5]. The CBT approach is however necessarily wider, as it addresses other areas of the patient's life (profession, family, etc.).

The second stage, naturally overlapping with the first, consists in gradually learning and applying new coping procedures in accord with the new problem formulations. In contrast to the SRM, in which coping procedures seem to emerge directly from cognitive and emotional IPs (probably also as a consequence of the underdevelopment of this aspect of the model), the CBT approach highlights the necessity of active training of skills such as problem-solving, relaxation, etc. On the other hand, the automatic and parallel generation of coping procedures from emotional IPs is an important aspect that limits the flexibility of the behavioral response to threat and that has been generally neglected in CBT, which views affect as a consequence of cognitive appraisals. This element of SRM brings it closer to emotion approaches to psychotherapy and chronic pain, but, as already mentioned, it is often neglected in practical applications of the SRM, too.

The third stage consists in gradually generalising the newly learnt strategies to situations where they are likely to lead to successful outcomes, which leads to consolidation of the new behaviour. An important focus of this stage is encouraging an awareness of the patient's new abilities to apply these strategies in various situations outside the therapeutic relation, and dealing with occasional relapses. In SRM terms, this stage aims for enhancing coherence of the patient's IPs also at intraindividual and interindividual levels (with self-image of efficacy, personality traits, significant other's IPs, environmental constraints over coping procedures, etc.), to ensure enduring adherence to self-management.

This brief overview of the CBT approach as presented in Turk et al. (1983) highlights the cognitivist essence of the SRM, but also the differences between these approaches. First, the application of CBT to health behaviour via SRM (or other theories such as the Transactional Stress and Coping Model, the Health Belief Model, the Theory of Reasoned Action and the Theory of Planned Behaviour) brings out the processes described above from the area of psychopathology to the realm of normal behaviour. Second, the SRM's focus on emotion is theoretically

[5]In SRM terms, the development of the cognitive approach to chronic pain and related research can also be seen as a parallel and ongoing process of reconstruction at the level of the therapists' IPs.

distinct, and certainly worth a more extended application. Third, the behavioral training elements of CBT are more developed than in the SRM, and point towards the necessity of further developments of this aspect of the SRM. Fourth, CBT has a wider focus than the SRM, as it addresses other life domains in addition to health.

According to the CBT approach (Turk et al., 1983, p. 5–6), therapeutic change can take place at several levels: the content of thought (beliefs, schemas), the process of thought (automatic thoughts, problem-solving, cognitive coping), or the behaviour (coping skills). The literature concerning the various theories, concepts and instruments in this domain is too broad to review here. Suffice it to say that the SRM has wide areas of overlap with many of them, and the search for the most useful conceptualisation is far from over (e.g. Damme et al., 2008). Actually, a notable feature of this research domain is represented by the significant overlaps between concepts and also the overlaps between thought content, thought process, behaviour and even emotion within the same concept.

Concept distinctions are particularly unclear. For example, in a review of the key developments regarding pain beliefs and coping, DeGood and Tait (2001) tried to differentiate between beliefs as referring to the understanding of events, as opposed to attitudes, which refer to feelings. However, one of the most common measures of pain beliefs, the Survey of Pain Attitudes (SOPA; Jensen et al., 1994) does not perform this distinction and also include beliefs about pain causing harm, which other authors consider as part of the fear of pain (see Subsection 4.4.4). DeGood and Tait (2001) also distinguished between pain beliefs and self-efficacy expectancies, which are future-oriented beliefs, and between outcome expectancies and self-efficacy beliefs, which are actually beliefs of personal control. However Jensen et al. (1991) considered beliefs, appraisals and expectancies as synonyms describing cognitions about pain, and showed the similarities between various instruments measuring concepts such as general locus of control, perceived control over pain, attributional styles, cognitive errors, self-efficacy beliefs, outcome expectancies and various other disability-related pain beliefs.

A representative example of conceptual overlap within a construct (also noted by Jensen et al., 1991) is pain catastrophizing, in which emotions (worry), negative cognitions and behaviour (avoidance-based coping) are mixed. It is included among the eight coping strategies initially developed based on theory for the Coping Strategies Questionnaire (Rosenstiel and Keefe, 1983), and in this context is measured by six items that seem to refer to feelings rather than cognitions. One refers to worry ("I worry all the time about whether it will end"), while the rest are expressing feelings

of hopelessness, despair and intense distress, such as 'I can't go on', or 'I can't stand it anymore' (Geisser et al., 1994).

They have been interpreted as negative, dysfunctional cognitions, and Vlaeyen and Crombez (1999) considered catastrophizing as an essential link between painful experiences and fear of pain. In their extension to Lethem et al.'s (1983) model of pain-related fear, painful movement can either generate adaptive cognitions, leading to confrontation and improvement, or catastrophizing thinking (depending on the doctor-patient interaction, personality traits such as negative affectivity and anxiety sensitivity, and interactions between these two), leading to pain-related fear and avoidance behaviours, and thus to disuse, depression and disability, intensifying the pain experience.

An alternative interpretation is that pain catastrophizing is an expression of profound emotional distress (McCracken and Gross, 1993), which explains the high correlations with measures of depression found in the literature (Lawson et al., 1990), although it may appear as distinct from depression measures (Geisser et al., 1994) due to the pain focus of the scale. Also, Aldrich et al. (2000) proposed to reframe catastrophizing as a manifestation of worrying about pain (a natural consequence of the interruptive function of pain in the context of chronic painful stimulation), instead of considering it a patient characteristic, equal with negative thinking and poor coping strategy[6].

This tendency of theoretical overlap between cognitive-behavioral approaches to chronic pain is partially a result of the difficulty to operationalise distinctly the emotional, cognitive and behavioral aspects of adjustment. The direct result of this overlap is the difficulty of testing any hypotheses related to the causal relationships within the whole process. Calls for concept clarity have occasionally been expressed. Jensen et al. (1991) drew attention to the frequent methodological problem in existing instruments of confounding coping, beliefs and adjustment in the same measure. In another review of the literature, Keefe et al. (1992) noted the existing confusion due to overlapping concepts studied using correlation, and the necessity to use adequate designs to reveal causal processes. Also, DeGood and Tait (2001, p. 326) pointed to the conceptual confusion between pain beliefs and coping and suggested attention to conceptual issues in designing further research. But these overlaps are

[6]As described in Subsection 4.4.4, fear-avoidance models are actually a mixture of emotion, cognition and behaviour. Measures of fear/anxiety have conceptualised emotion in terms of beliefs/appraisals of the negative consequences of pain and of the usefulness of avoidance behaviours in preventing further pain increases.

still a predominant feature of the field. The research within the SRM framework described in the next section builds upon this rich yet confusing inheritance.

5.3.2 SRM research

A growing body of research has focused on the role of IPs in various health conditions involving chronic pain: rheumatic diseases such as osteoarthritis (OA), rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), multiple sclerosis (MS), chronic fatigue syndrome (CFS), migraine, back pain, as well as on heterogeneous patient samples.

Several studies have focused on the role of IPs in rheumatic diseases. Pimm and Weinman (1998), in a review of the prior literature on illness beliefs in rheumatic diseases, highlighted the central role of pain in patients' representations of their condition, which is perceived as chronic, incurable, unpredictable, and relatively controllable via treatment, but with low personal control. They also reported research confirming associations between individual differences in beliefs and differences in distress, self-care, and health outcomes. Their review placed SRM applications in rheumatic diseases on a solid foundation of prior research in related frameworks and highlighted the similarities between them. For example, a cross-sectional study (Murphy et al., 1999) reported significant associations between IPs of consequences and personal control and depression, controlling for disability. In OA patients, IPs of identity, consequences, timeline and emotional impact were related to limitation in activities beyond the expected values based on medical evaluation (Botha-Scheepers et al., 2006).

Longitudinal studies have only partially confirmed these results. Schiaffino et al. (1998) compared RA with MS sufferers and investigated the interactions between IPs and later health status in predicting changes in depression. They reported that, while in MS only perceived symptom variability was related to increased depression at 4-month follow-up, in RA depression was associated with more aspects: IPs of greater treatment and personal control and interactions between IPs of consequences and later health status. A two-year study on a sample of female RA patients (Groarke et al., 2005) found that IPs (especially regarding illness identity and consequences) explained 17-33% of the variance in various measures of concomitant physical and psychological functioning, including pain, controlling for medical indicators of disease status; however IPs had no predictive power for adjustment measures in subsequent stages.

In other conditions, similar results have been found. Heijmans (1998) reported that IPs were associated with physical and social functioning, psychological adjustment and well-being in a sample of CFS patients, controlling for age, gender and illness duration. Coping strategies did not mediate between IPs and these outcomes, and CFS patients tended to consider their condition as serious, mainly biologically caused, with multiple symptoms and significant impact on their lives, although they tended to be optimistic regarding possibilities of control and treatment. In a 6-month longitudinal study of low back pain patients attending primary care (Foster et al., 2008), IPs of consequences, timeline, personal control and treatment control were significantly associated with poor outcome, as measured by reduction in self-reported disability and global self-ratings of change at follow-up stage, adjusting for demographics and illness duration. Radat et al. (2009) reported differences in IPs of identity, treatment control and consequences between episodic migraine and chronic daily headache sufferers, controlling for other measures of headache impact, psychological distress, coping and locus of control.

Several recent studies have investigated heterogeneous samples of chronic pain sufferers aiming to identify common characteristics. Petrie et al. (2005) used both interviews and questionnaires to investigate patients' expectations from their first visit to a pain clinic. Obtaining an explanation or increased understanding of their condition was the most frequent expectation reported, followed by the search for relief or a cure. However, patients with high depression scores were more prone to report they expect relief or cure of pain rather than understanding, while high disability patients expected more control, and less understanding, and even less a cure. The importance of understanding their condition highlights the central role of IPs in the patients' efforts to manage pain. Also, the study points to a possible influence of depression or disability on the coherence between patient's IPs and doctor's IPs.

For patients attending a multidisciplinary pain program (Moss-Morris et al., 2007), more improvement in physical health was associated with reductions in IPs of serious consequences, while improvement in mental health was related to reductions in IPs of emotional distress, but unexpectedly also with reductions in perceptions of coherence. Chronic timeline IPs were not affected, and control IPs significantly decreased, probably showing the adoption of an accepting approach to pain management. As a possible first step in customising treatment packages that include IPs, two major clusters of chronic pain patients have been identified based on IPs: adaptors and non-adaptors (Hobro et al., 2004). The two groups differed on a

variety of aspects such as perceived general health, pain, energy/vitality, mental health, general physical functioning, depression, beliefs about medication (and naturally most IPs), except IPs of cyclical timeline, self-reported anxiety, physical and emotional role limitations, social functioning, demographics and medical history.

Research is still in its initial stages, and the authors recommended replications of these findings, and the application of more powerful designs (Hobro et al., 2004). The clinical utility of IPs needs further investigation, whether as a criterion for patient classification (for which their stability and predictive utility across time would need to be proven), or in relation to other related concepts.

5.4 Interpretation caveats regarding illness perceptions

The SRM has already been applied successfully in health contexts, and it has the potential to lead to useful interventions in chronic pain as well. However, despite these research developments, the proclaimed advantages of SRM are not yet taken advantage of, and this limits the significance of research addressing IPs without the whole context of the model.

The major difficulty in interpreting research on IPs is assessing to what extent they reflect differences in ways of perceiving events that are intrinsic to the individual or differences in concrete characteristics of the health condition and other relevant factors. The SRM was designed to change the focus on describing the process rather than identifying the preconditions of adequate behaviour (as detailed in Section 5.2) and thus IPs were not viewed as explanans, i.e. as determinants of the behaviours for which an explanation was sought, but rather as part of a process which in itself constituted the phenomenon to study. However, correlational studies of IPs and health status are often interpreted in terms of the impact of a subjective perception on the objective outcome or measure of functioning, and some authors describe IPs in terms of a positive-negative dichotomy (Petrie and Weinman, 2006). These frequent interpretations actually fit in the 'preconditions' framework: identifying the "patient cognitions that can act as either a help or a hindrance to illness adjustment" (e.g. Weinman and Petrie, 1997, p. 115), and, in my opinion, not enough attention is paid to the role of context and system coherence.

As an example, in their study of patients with episodic migraine and chronic daily headache, Radat et al. (2009) interpreted differences in responses to BIPQ items as psychological differences between the two groups, when they might actually reflect

differences in illness severity (as reflected in self-report). They noted the strong associations between these items and the measure of headache impact (and correctly regarded these associations as being uninformative relative to causal direction and clinical significance), but failed to consider the alternative interpretation of meaning overlap between the two instruments. The BIPQ items might simply be self-report proxy measures for the headache impact in this context, and the associations would not indicate a connection between psychological factors and health status, but both instruments being alternate measures of the same aspect[7]. Radat et al. (2009) also used BIPQ total scores, which assumes that the illness perceptions are correlated and reflect an underlying common characteristic, but did not specify its theoretical meaning. One could argue that perceptions of consequences, concern, symptoms and emotional impact (perhaps even timeline) reflect aspects of a common 'illness impact perception'. But perceptions of control and understanding do not easily fit on such a continuum. In fact, illness perceptions are not uniformly intercorrelated (see Moss-Morris et al., 2002 for correlations between IPQ-R subscales, and Section 8.11 for a statistical analysis of these relationships in the present study).

It is important to note that the SRM does not imply that, irrespective of the condition, one is supposed to feel unaffected, think it won't last long, feel in control, consider the treatment effective, experience less symptoms, feel less concerned, more knowledgeable and less affected emotionally. On the contrary, the adaptive approach of SRM implies that self-regulation depends on the coherence between the cognitive and emotional, schematic and conceptual, intrapersonal and interpersonal levels, each influenced by different aspects of the current situation (including the physical symptoms, other external events and sources of information and previous coping strategies and their outcomes). Studies that claim to apply the SRM should analyse their data and interpret their results in accord to this theoretical stance.

One possibility is controlling for medical status when assessing differences in IPs, and most studies do (e.g. Petrie et al., 1996; Scharloo et al., 2000; Foster et al., 2008), although it is often difficult to identify the most suitable measure[8]. If the medical condition has an equivalent manifestation in all subjects (or its influence is

[7]This is a typical example of overlooking basic measurement principles regarding specificity and sensitivity. A questionnaire, experimental task or psychophysiological measure is not invariably measuring a construct. Its psychometric properties are to a great extent determined by the assessment context and experimental design. If essential confounding variables are not controlled for, the instrument loses specificity. If context is changed, it does not automatically keep its sensitivity (Cacioppo et al., 2000).

[8]For example, illness duration (employed as control by Foster et al., 2008) is obviously only a limited aspect of the clinical characteristics of low back pain.

partialled out), one can justifiably assume that the differences in illness perceptions are due to interindividual differences in other domains (psychological, social, etc.) and might influence the outcomes by differences in illness behaviours resulting from perceiving their situation more or less adaptively. In some health conditions, it might be argued that the objective aspects of the health status can easily be assessed or considered equivalent, such as for diabetes (Weinman and Petrie, 1997)[9], but this argument is difficult to support in the case of chronic pain.

As detailed in Chapter 2, chronic pain is not among the easiest conditions to assess objectively, and measures of health status in chronic pain are inevitably overlapping. Thus, the associations between subjective assessments of pain intensity or disability and subjective assessments of perceptions of concern, timeline, consequences, etc. are unsurprising and to a large extent due to the fact that respondents access similar information to address the questions. For example, as Macfarlane (2008) explained in relation to musculo-skeletal pain, IPs of greater consequences might reflect illness severity, while timeline IPs might result from prior experience. The differences in individual scores between essentially similar measures might reflect slightly different question wording, the potential of one measure to capture elements of the physical pathology that other methods do not access (e.g. as a global assessment of the physical state of the organism, and not of a localised pathology), or other elements of the wider personal and social context of the health condition (e.g. the individual's abilities to cope with the condition given the personal history or social support available). In this case, changes the IPs (a frequent suggestion following interpretation of cross-sectional studies; e.g. Heijmans, 1998) cannot be triggered by focusing on the perception as such, but on identifying and addressing its determinants beyond illness severity.

The consequences of disconsidering the bidirectional relation between illness severity and IPs in the context of chronic pain are significant, given the long-lasting contentious issue of psychogenic pain. The danger to classify IPs as 'positive' or 'negative' without considering the context lies in reviving the old psychiatric approach to pain, with highly likely negative consequences for the doctor-patient relationship and treatment efficiency. From a less judgemental approach to psychosomatics which assumes that IPs are not restricted to pathological responses (Weinman and Petrie, 1997), the SRM risks a transformation of this promising idea into a 'bad' IPs hunt.

[9]Even in these situations, the remaining differences in IPs can be due to both individual characteristics and other aspects of the medical situation that the respondents consider when developing their IPs, but were not included as separate variables in the study.

Another possibility of correctly interpreting IPs in the context of the SRM is by comparing the dynamic relations between the various representations, which the SRM describes as coherence: a match between schematic and propositional levels, between emotion and cognition, between patient and significant others, or between perceptions and personality, which leads to better self-management of the chronic condition (Leventhal et al., 1992, 2008). Unfortunately, this concept does not prove easy to measure or study. The coherence subscale recently included in the IPQ-R (Moss-Morris et al., 2002) does not directly address it, it rather measures the respondents' impression of how much they understand their illness, which can reflect an authentic coherence between some levels or a false feeling of comprehension. In fact, a major difficulty is that very few levels stipulated by the SRM are actually accessible by current measures, and efforts to extend the applications of the model beyond the self-reported IPs are only a recent development.

Self-report measures access the propositional level, but not the schematic level, which is by definition automatic and not accessible via language. The authors actually warned that theoretically-derived measures might not even assess the propositional level adequately (Leventhal et al., 1997), and recommended using concrete examples of representations and coping strategies, rather than general experimenter-generated categories, as these might obscure the wide diversity of illness thoughts and behaviours individuals use to manage chronic illness, which may prove essential for clinical practice (Leventhal et al., 1992). The authors of IPQ also recommended the complementary use of qualitative methodology and the development of questionnaire versions adapted for specific needs (Moss-Morris et al., 2002).

Newer research methods have started tapping into the schematic level by means of drawings (Broadbent et al., 2004, 2006a, 2009c; Reynolds et al., 2007) and Stroop tasks (Henderson et al., 2007). For example, the size and presence of damage on heart drawings were related to illness-specific anxiety, depression, IPs and clinical measures of illness severity (Reynolds et al., 2007), the extent of damage represented in the patients' drawings of their hearts after MI predicted slower return to work and IPs regarding recovery after 3 months (Broadbent et al., 2004), while increases in the size of the heart drawn at subsequent stages were related to increased cardiac anxiety and poorer recovery (Broadbent et al., 2006a). Properties of the patients' drawings of their headaches (content, size and darkness) were also associated with IPs of identity and consequences and also with physical and emotional functioning (Broadbent et al., 2009c). Priming IPs for an illness schema of common cold (by asking subjects to narrate a prior experience) resulted in response bias in a Stroop

task for common cold-related stimuli, but not for stimuli related to cardiovascular disease, and the bias was associated to the respondents' explicit IPs of consequences and emotional distress (as measured by the IPQ-R), confirming the SRM claims regarding the existence of illness-specific schemata (Henderson et al., 2007).

The perceptual-motor level described in Leventhal's model of emotion is not even included in the SRM, even if the perceptual-motor theory of emotion underlines its importance in emotion generation, and the SRM acknowledges the role of emotion in health-related decision-making[10]. Even if Leventhal et al. (1992) discussed the role of physical symptoms in describing specific examples, only schematic appraisals were proposed. But as discussed in Chapter 2, pain has an immediate, automatic impact on emotion, which can be described as activating at the perceptual-motor level, and thus influencing the sufferer's emotional reactions which may interfere with schematic or conceptual-level decisions regarding coping strategies.

While propositional cognition is the main focus at present in SRM applications, emotion representations are a recent addition to IPQ-R. However, the emotional representations subscale items of the IPQ-R (and the correspondent item in the BIPQ) do not assess emotional appraisals, in the sense described by the perceptual-motor model of emotion, only emotional responses. The emotional processing is barely developed in SRM in terms of content. If content would follow the distinctions in appraisal models, one would be able to characterise emotional appraisals of symptoms in terms of novelty, pleasantness, etc., which would enable a distinction between qualitatively different emotional consequences of the representations (i.e. discrete emotions), not only the increased illness threat, and also clarification of the internal structure of the emotional content of representations. Emotional responses are described as 'blended' and 'embedded' in the cognitive-affective-behavioral framework, with prototype checks leading to both update of IPs and emotional reactions (Leventhal, 2008). Indeed, some cognitive representations seem to overlap original emotional appraisals, as interpretations of causation and control are also among the types of stimulus evaluation checks described in Scherer's CPM, and perception of consequences can be compared to goal significance appraisals. Unsurprisingly, these cognitive representations are correlated with the emotional subscale in IPQ-R (Moss-Morris et al., 2002), and with other measures of emotional distress (Fortune et al., 2000). The emotional component of the SRM is the focus of newer theoretical developments (Diefenbach, 2008).

[10]No suggestion is given regarding a perceptual-motor level of cognitive processing of illness symptoms.

The SRM situates self-regulation in chronic illness in the bigger context of intrapersonal (such as self-efficacy, or optimism) and social (such as community-shared and health practitioner-reinforced illness beliefs) phenomena (Leventhal et al., 2008). Comparatively fewer studies have addressed these relations. For example, the relation between IPs and self-efficacy proves complex in patients with CHD: general self-efficacy is related to concurrent IPs regarding consequences and expectations regarding outcomes of dieting and exercising, but only outcome expectations are associated with self-efficacy levels at 9-month follow-up (Lau-Walker, 2006), possibly suggesting a mediating role of outcome expectations in the relation between IPs and self-efficacy.

The coherence between the patients' IPs and their carers' IPs about their illness has recently started to be addressed. A significant other IPQ version is available (Weinman et al., 1996). Studies regarding the similarity of patient and carer IPs reported mixed results, depending on the health condition. For example, Law (2002) found that mothers of adolescents with type 1 diabetes perceive their condition as having more consequences and emotional impact, but the differences in dissimilarity are not related to the adolescents' well-being. In a sample of adolescents with atopic eczema, the similarity between the parents' IPs was related to the patients' well-being (Salewski, 2003). The similarity between the IPs of males after their first MI and their spouses' IPs regarding their condition was related to better long-term physical, psychological and social functioning, but only when both considered the condition as having less impact, shorter duration, fewer symptoms and more controllability (Figueiras and Weinman, 2003). Creating coherence the between the patient's and the medical professional's IPs becomes essential to an efficient provision of reassurance following medical testing, and seems to be dependent on a series of patient-related and doctor-related characteristics, but also on features of the medical situation itself, such as the time delay before the test is taken (Petrie et al., 2007).

Moreover, IPs are only a component of the SRM, which also includes treatment beliefs and outcome expectations; these additional components are rarely considered together with IPs in studies attempting to test the SRM (Leventhal et al., 2008). Coherence between IPs and treatment beliefs is considered central to SRM, and leading to perceptions of treatment effectiveness and to treatment adherence, a prediction confirmed in studies of hypertension, asthma, and myocardial infaction. For communication intervention purposes, achieving coherence is understood as a conversion of illness and treatment representations into behavioral scripts, as

behaviours motivated by the representations meet expectations of outcomes (Leventhal et al., 2008).

All these studies addressing the complex issues of coherence, although at the beginning, are promising research directions that adequately apply the theoretical claims of the SRM. If multiple levels are not assessed, efforts to control for objective characteristics of the health condition are necessary, if the condition is amenable to a relatively objective diagnostic. This does not mean that the use of a single self-report measure of IPs in the chronic pain context cannot provide information relevant to the SRM. The interpretations of the results needs however to take into consideration the multiple determination of the self-reported IPs and possible overlaps with other measures of functioning.

5.5 Conclusion

This chapter has reviewed cognitive aspects of adjustment to chronic pain, with particular focus on the concept of illness perceptions and the self-regulatory model. A few important conclusions should be highlighted. First, IPs as measured by validated questionnaires (IPQ and BIPQ) focus on a limited range of pain-related appraisals, and many other appraisals can be identified depending on the health condition, individual, or research interest. Second, IPs are only one element of the SRM; coping procedures and outcome evaluations, emotional states, the schematic level, plus intra- and interpersonal levels are equally important, although less addressed aspects. Third, IPs are evaluative in nature, and thus obviously overlap with self-reports of health status. IPs are important predictive factors as they express the fact that there are significant differences in patients with the same medical condition beyond the known and measurable medical indicators. But they are not to be taken as representing internal characteristics of the patient, easily changeable by persuasion (i.e. should not be targeted directly as maladaptive cognitions). They are the product of a global assessment of various sources of information regarding the physical symptoms, the personal and social context, the various illness entities as described by media, community and medical professionals, etc. Influencing IPs would have to be driven by investigations of the personal, social and contextual determinants of IPs.

This review has highlighted yet again the interrelations between cognition, emotion and behaviour. IPs are both cognitive and affective appraisals, and directly result

in action to overcome the perceived health threat. Therefore a comprehensive description of adjustment to chronic pain would have to consider all three domains. This is the topic of the next chapter.

Chapter 6

Studying interrelations within an integrative framework

6.1 Introduction

In a review of the current state of the art in chronic pain research, Keefe et al. (2004) highlighted the need to develop models that integrate the knowledge related to separate (but often overlapping) concepts into a more comprehensive theory that would consider their relative importance and temporal relationships. They also noted the necessity to clarify these concepts. The present thesis is in a sense an attempt to address these two important issues.

The previous chapters have treated pain, acceptance, emotions and illness perceptions separately. This chapter attempts to build an integrative account. It starts with clarifying the similarities and differences between these separate domains by discussing both theory and empirical evidence. It then clarifies the necessary characteristics of a theoretical model, and the requirements of model testing via different statistical models. The rest of the chapter focuses on the conceptual analysis of the current models of pain experience from the perspective of integrative efforts, which further guides the justification and formulation of the hypotheses for the present study.

6.2 Interrelations between emotions, acceptance and illness perceptions

6.2.1 Emotions and acceptance

The relationship between acceptance and emotion (and its regulation) has been directly addressed in ACT/RFT literature. Hayes et al. (2001, p. 171) considered the dispute between componential theories and basic emotions theories regarding the genetic origins of emotions as unimportant. They gave more attention to the social implications of how emotion is regulated by language and cognition. In ACT, emotions are seen as socially constructed to a large extent: via training, relational frames develop between sets of bodily sensations, behavioural predispositions, thoughts, situations and emotion labels (Hayes et al., 2001, p. 127). The similarity of an individual's relational frames to those of the social community and their flexible application depending on context to meet personal needs is taken to represent healthy emotional development.

Among the current emotion theories, this view of emotion as socially constructed and of language as relevant for emotion regulation is most similar to Barrett's (2006b) conceptual act model, which sees discrete emotional experiences as the result of psychological categorisation of core affect (characterised by valence and arousal), and the acquiring of emotion categories as a result mainly of social construction. Emotion categories are described as heterogeneous sets of situated conceptualisations (i.e. perceptual symbols) which participate in the construction of perceptual experiences and influence subsequent behaviour on multiple modalities; they have a functional role in selecting communicative and instrumental behaviours that likely to ensure self-regulation or goal achievement in the particular social context[1]. Learning the emotion categories that are socially functional in one's culture participates in a healthy psychological and social development. Barrett also identifies interindividual differences in the precision of verbally representing emotional experiences ("emotion granularity"), highlighting the possibility of training the functional use of emotion conceptualisation as a skill.

RFT/ACT also states that emotions, like all internal events, are not causes of behaviour, but could be "controlling variables that participate in an overall causal

[1]The theoretical distinction between relational classes and situated representations, although it is controversial and important in differentiating RFT from cognitive accounts of human behaviour, is less important at this level of analysis, as both serve the same function of influencing behaviour.

relation" (Hayes et al., 2001, p. 176)[2]. This view of emotion as unrelated causally to behaviour can be identified as a Platonian/Cartesian/network theory view on emotion (see Subsection 4.2.1), which is unsurprising given the skinnerian foundation of ACT. This is another standpoint that RFT/ACT partially shares with the conceptual act model. In her critique of basic emotion views, Barrett (2006a) noted how compelling the view that emotions are natural kinds (i.e. nonarbitrary clusters of instances existing in nature and not created by the human mind) is for both laypersons and researchers despite the difficulty of researchers to reliably measure them. The fact that we explain our behaviours via the subjective experience that accompanies them compels us to see them as causal entities (p. 47), although in emotion research this view is in her opinion counterproductive. As acceptance theory states, giving internal events as reasons for behaviour is not necessarily beneficial in personal life either[3].

However, Barrett (2006b) granted an important role to emotion categorisation in guiding adaptive behaviour and thus described the use of emotion categories as important elements in causal networks of human behaviour. Along similar lines, Frijda (2008) stated that even if emotion might not represent "a natural class of phenomena", it "fills a need in pointing to particular phenomena of feeling and behaviour" which "tend to intrude upon ongoing thought and behaviour" and "seek to assume control, tend to persist over time, and may do so even when prevailing conditions make it advisable for them not to do so" (p. 68)[4]. Thus, even if emotion labels have not always been taken to correspond to neural programs which reliably lead to coordinated multimodal adaptive responses, a minimal consensus in the emotion literature refers to emotion as a useful concept in the sense of an association between use of emotion labels/categories and relatively coherent types of goal-oriented and self-regulating behaviours. This view also concurs with the RFT/ACT view regarding the role of language in emotion.

Emotion categories, or schemas, also include "meta-emotional skills and representations" (Power and Dalgleish, 2008), which guide the perception, understanding and regulation of emotion experience. An analysis of the RFT/ACT description of

[2]Although not clearly stated, this theoretical statement might be equivalent to a statistical moderation relationship. Causality is discussed in more detail in Section 6.3.

[3]Barrett (2006a) also noted that research methodology that stipulates the existence of latent traits is consistent with such natural kind views of emotions (p. 47). Nevertheless, in my opinion, emotion variables can be included in causal chains (e.g. in SE models) without adopting a 'natural kinds' approach, for example when considering the subject's endorsement of emotion labels in characterising their recent experiences as indicators of the frequency of using the corresponding emotion categories in interpreting and guiding their experience.

[4]See also Johnson-Laird and Oatley (1992) for a defense of folk theories of emotion.

psychological flexibility and acceptance from the perspective of emotion regulation is a promising avenue for integrating these two approaches, although it is rather controversial and unclear at present.

In RFT terms,

> people are taught to categorise a loose set of situational cues, bodily sensations, behavioural predispositions, and so on as "anxiety" and to evaluate it as "bad." This "emotion" can then be recalled or predicted via language (e.g., "I felt anxious at school last week" or "I am afraid I will get anxious when I get on the place"). Because aversive states of this kind can be brought into a situation via language itself, psychological pain cannot be avoided purely by avoiding external situations. Humans thus begin to target negatively evaluated private events per se as the focus of avoidance. For example, thoughts linked to "anxiety" can be actively avoided or suppressed. (Hayes et al., 2004a)

In this statement, the authors seem to suggest that only the negative and inflexible meta-emotional representations (e.g. anxiety is bad) lead to avoidance of inner emotional events and thus to psychopathology, not language as a whole. Their focus on the negative role of language could be due to considering anxiety as the blueprint for all other emotion categories. Friman et al. (1998) explained anxiety by the process of inner (verbal) events becoming associated to avoidance behaviours via relational framing so that these behaviours are stimulated in the absence of the real (original) danger. This property of relational framing of randomly carrying over behavioural functions from one event to another via transformation of stimulus functions and forming relational classes is described as a characteristically human process and the source of increased suffering.

Similarly, Blackledge and Hayes (2001) proposed that attempts at regulating emotion can cause psychopathology (in a social context that encourages emotion regulation as a way to diminish negative affect) and considered this statement as opposing current views that failure of emotion regulation is potentially pathological. In fact, the two statements are not contradictory, instead they refer to different acceptions of the term. Blackledge and Hayes's (2001) is a limited definition of emotion regulation as equivalent to getting rid of negative emotions (part of experiential avoidance of inner events), while current views in emotion theory describe emotion regulation as both reduction and enhancement of both positive and negative emotional states, which can take place in relation to any of the distinct emotion components

(experience, physiology, behavioral expression), as discussed in Chapter 4. Thus, acceptance (or "feeling feelings as feelings", as described by Blackledge and Hayes, 2001, p. 247) can also be considered a form of emotion regulation, since orienting attention towards its constituents changes the emotion experienced.

If experiential avoidance (EA) is a form of emotion regulation, what are its distinctive features, and it is invariably dysfunctional? In a comparative analysis of ACT and CBT methods, Hofmann and Asmundson (2008) considered ACT techniques as counteracting maladaptive response-focused emotion regulation, and CBT as targeting primarily antecedent-focused emotion regulation. However, in Eccleston and Crombez's (1999) cognitive-affective model of the interruptive function of pain, acceptance reduces pain-associated worry by redefining the problem in terms of reducing disability and distress rather than pain reduction and thus stopping the misdirected problem-solving efforts. A recent opinion in this controversy (Kollman et al., 2009) stated that even if direct control efforts are absent, "acceptance can be conceptualized as an emotional regulation strategy that combines aspects of "antecedent-focused" and "response-focused" emotion regulation, such that it entails both the appraisal of emotion acceptability and the allowing of emotional experience after its generation in the absence of control efforts" (p. 206). Moreover, Kashdan et al. (2006) noted that EA becomes disordered when it interferes with valued living via its inflexible application, and not as a short-term, context-related emotion regulation strategy. Even if these versions of acceptance are different from the ACT definition, they are important for our present integrative effort as they reveal the similarities and differences between acceptance and other concepts, in this case emotion.

Indeed, emotion theory generally asserts that language is essentially functional in relation to emotion, and can also encode adaptive meta-emotional representations. For example, Barrett's (2006b) model focuses on the functional role of emotion categories. As situated conceptualisations, they have the property of stimulating similar behaviours in similar situations, thus guiding adaptation from a higher level of abstraction (the anxiety category would lead to avoidance in some situations, while anger would lead more frequently to aggressive behaviours, and so on). In this view, an important characteristic of emotion categories is their flexible and context dependent connection to instrumental and communicative behaviours, and thus language in itself is viewed as fundamentally adaptive; it is not the emotion category itself that is dysfunctional, but the meta-emotional representations that guide its application. Moreover, the flexible application of both enhancement and

suppression of emotion expression is a better predictor of long-term psychological adjustment than the ability to either suppress or enhance (Bonanno et al., 2004).

The dynamic models of emotion regulation are also in accord with the acceptance approach. For example, Zautra et al. (2001) criticised unidimensional models of adaptation to pain, like stress and coping models, which view adjustment outcomes on a single continuum. Their model insists on the differentiation between positive and negative affective states in times of stress as an important outcome which enables the individual to formulate a more nuanced definition of his/her well-being. This emotion approach to adaptation to chronic pain is conceptually similar to the acceptance view, where differentiation between the present pain levels and the readiness to engage in activities is an important therapeutic outcome.

Strand et al. (2007) further developed the DMA model by introducing a cognitive characteristic: the individual's readiness to self-manage pain ("pain readiness to change"). In their study, the higher weekly pain reports were related to lower positive affect reports especially for patients that reported more commitment to an active coping approach, as resulted from their scores in the three related subscales: precontemplation (seeking for a cure for pain), contemplation (starting to consider active coping), and action/maintenance (using coping strategies on a daily basis). The authors explained this finding in terms of perceptions of control: pain increases might be seen as failure of coping strategies and felt as a disappointment.

Several studies so far have empirically investigated the relation between acceptance and emotional responses. For example, Sloan (2004) described EA as an emotion regulation strategy that leads to self-reports of increased emotion when exposed to fear, disgust and happiness-inducing film clips (but not to contentment and sadness-inducing films), and lower heart rate reactivity only to fear and disgust clips (no differences were observed for electromyographic recordings of facial expressions of frowning and smiling). In Kashdan et al.'s (2006) cross-sectional study, EA mediated the relation between anxiety-related distress (anxiety sensitivity, trait anxiety, suffocation fears and bodily sensations fears) and several other maladaptive regulatory strategies (maladaptive coping, emotional response styles and perceptions of uncontrollability). Also, EA mediated the relation between two emotion regulation strategies (suppression and reappraisal) and several indicators of psychological distress and meaningful living during a 3-week monitoring period. The authors considered these results as supporting a view of EA as a broader construct (a "core toxic diathesis", p. 1302) which includes avoidant and detached coping, emotional suppression and uncontrollability, in addition to its own theorised components such

as inflexibility and cognitive entanglement[5]. It is important to mention that this shifting image of EA (and PF) in relation to emotion regulation is partly due to the difficulty of defining the concepts clearly within a theory that highlights the contextual variation of the phenomena studied, as detailed in Chapter 3.

In chronic pain, few studies have addressed this issue. Pain-related anxiety (as measured by PASS) and acceptance (CPAQ) were considered related but distinct dimensions of chronic pain adjustment based on a study by McCracken et al. (1999). Both were characteristic of dysfunctional patients (according to the WHYMPI classification), and contributed to a discriminant function analysis which classified correctly 72.5% of dysfunctional patients and 91% of adaptive copers (but none of the interpersonally distressed patients). The pooled within-group correlation between CPAQ and PASS was $r = -.51$. In two samples of female osteoarthritis and fibromyalgia patients, pain acceptance (selected items from CPAQ) was associated with higher levels of weekly positive affect, but unrelated to negative affect reports; moreover, accepting patients reported less negative affect simultaneous with higher pain severity, but this relation is possibly mediated by the increases in positive affect (Kratz et al., 2007). In a heterogeneous sample of patients referred to pain management, CPA, mindfulness and values-based action partly mediated (cross-sectionally) the relation between anxiety sensitivity (AS) components and measures of patient functioning such as depression, pain-related anxiety, psychosocial disability and number of visits to GP (McCracken and Keogh, 2009). The authors interpreted these results as indicating that AS is part of the wider concept of experiential avoidance, which also includes CPA, mindfulness and values-based action[6].

The theoretical and empirical work reviewed above suggests that CPA can be also seen as an emotion regulation strategy which impacts on the frequency and intensity of experiencing various negative and positive affective states. A comparative empirical analysis of the relations between CPA and specific discrete emotions has not been attempted until now.

[5]As discussed in relation to CPA and disability, these mediation effects can also be due to the fact that EA is conceptually closer to outcome measures such as distress, in comparison to more distinct concepts such as emotional suppression, or various coping strategies.

[6]An alternative interpretation would be a conceptual overlap between AS and ACT measures. Issues of measurement and structural relations are discussed in detail in Section 6.3.

6.2.2 Emotions and illness perceptions

As detailed in Chapter 5, the SRM is in essence a theory of the interactions between emotion and cognition in guiding behaviour in the context of a health threat. Emotions can be elicited by the symptoms, by initial illness representations (especially identity and consequences), by specific emotional appraisals, by assessment of coping outcomes or by interpersonal factors (not detailed in the model). They have an equally important role in the iterative selection of coping procedures and assessment of their results.

Despite the equal focus on emotion and cognition in the SRM, the role of emotion in adjustment to chronic illness from a self-regulation perspective has only started to be explored. Research has usually focused only on the cognitive component (mostly at the propositional level), with only a recent addition of an emotional impact subscale in the IPQ-R (and two corresponding items in the BIPQ). This shift in research focus is beginning to unveil the affective component of risk assessment, the affective heuristics that influence decision making in health settings, and the role of affect in directing attention on specific information, in motivating behaviour and in acting as common currency for comparing complex and qualitatively different situations in decision-making (Diefenbach, 2008).

In chronic pain however, no study has yet explored the relations between illness perceptions and discrete emotions. Despite an early formulation of the parallel processing model of pain distress similar to the SRM which specifically stipulated the possibility of pain schemas determining associations between sensory pain characteristics and specific discrete emotions (Leventhal and Everhart, 1979), most studies of the relation between cognition and emotion have focused on the relation between various cognitive factors and chronic pain associated depression, anxiety or anger, formulated outside the SRM (Arnstein et al., 1999; Maxwell et al., 1998; Materazzo et al., 2000; Turk and Okifuji, 1997; Woby et al., 2004; Newth and DeLongis, 2004; Page et al., 2004; Cordova et al., 2005; Heath et al., 2008; Tan et al., 2008; Karoly et al., 2008). As discussed in Chapter 4, a discrete emotions approach is potentially more rewarding.

6.2.3 Acceptance and illness perceptions

The SRM and ACT approaches to chronic pain have not been compared in the literature. Given the strong cognitive-behavioral tradition behind the SRM and the critical views of ACT proponents regarding CBT, illness perceptions might be

considered as having less in common with acceptance. There are however many common aspects. As Hofmann and Asmundson (2008) pointed out, ACT and CBT share many techniques and theoretical views. For example, in relation to valued living, ACT advises the functional analysis of the person's beliefs based on their impact on the selected goals, and this analysis is similar to the SRM cycles of situation assessment - action - outcome assessment. Thus, psychological flexibility also entails adopting the beliefs that are most adaptive in relation to the person's value structure and present context. Moreover, ACT and SRM have in common the emotional components reviewed in the previous subsections. To my knowledge, no empirical exploration of the relationships between CPA and illness perceptions has been reported yet.

6.2.4 The role of demographic and other contextual factors

It is important to underline that psychological factors in chronic pain are active within a network of various other contextual variables characterising the sufferers (gender, age, ethnicity, socio-economic status, marital status, etc.), their condition (duration, body area affected, the presence of a clear diagnosis and of comorbid conditions) or the their social environment and situation (the presence of stressful life events, social and financial support, conflict situations such as marital problems or litigation). These factors can act as moderators, mediators, or alternative causal factors for the relation between psychological aspects and health status in chronic pain, and can be in turn differently affected by changes in the condition. Some of their implications are briefly reviewed next.

As a general example, patient demographic and clinical characteristics were significantly associated to various dimensions of quality of life (QOL) in a heterogeneous sample of patients admitted to a pain management programme: women reported lower physical role-related QOL; higher educated people reported better general, emotional and mental health, physical function, and vitality; marriage, employment and shorter pain duration were associated with better general health; involvement in compensation claims or litigation was related to reports of lower mental health; reports of more than 3 pain locations were associated to lower physical function (Kerr et al., 2004). Beyond such general associations, the relation of each of these characteristics with various health outcomes is multifaceted; the specific influences in different contexts and their causal mechanisms are a topic of continuing research.

Gender is connected to chronic pain adjustment via both biology and social roles. Most studies indicate slightly higher pain sensitivity for acute pain in women, but results vary depending on type of stimuli and experimental design. Biological bases might consist in differences in pain modulation mechanisms, and in the neuroendocrine and reproductive systems. Social factors might relate to the willingness to report pain. For chronic pain conditions, most studies report higher prevalence in women, although results are not consistent for all conditions and age intervals, and most studies focus on Western cultures. Women are also more likely to seek care for their pain condition, possibly due to both pain sensitivity and differences in the socialisation of pain, and use more pain management strategies (Berkley, 1997; LeResche, 2001).

Psychological factors may impact on chronic pain adjustment differently depending on gender. For example, Jones and Zachariae (2002) reviewed the limited evidence on the moderating effects of gender on the relation between anxiety and pain sensitivity, and concluded that, although women's pain responses seem to be more sensitive to context-related anxiety, dispositional anxiety seems to be associated to chronic pain severity only in men. In a recent study on chronic pain patients, Keogh et al. (2006) reported that gender mediated the relation between depression (and not pain-related anxiety) and disability, as women with higher depression scores also reported significantly more disability. As these controversial and limited results suggest, the role of gender in this relationship is yet unclear, therefore separate analyses are recommended for future research (Jones and Zachariae, 2002).

Age-related differences in pain experience and expression can also be due to both biological and psychosocial factors. While childhood is a period of substantial changes in biological, cognitive and emotional maturation and in the socialisation of pain behaviours, adulthood (18+ years) is comparatively more stable. Nevertheless, increasing age is associated with higher prevalence of persistent pain and a plateau or decline in the old population (75+). Age also impacts differently on specific anatomical pain sites and reports of associated symptoms, or reporting of sensory pain qualities (but not intensity ratings) and on pain-related anxiety (but not depression), cognitive beliefs, coping mechanisms and disability (Gagliese and Melzack, 2003; Gibson and Chambers, 2004).

The influence of ethnicity on pain reporting has been encumbered by stereotyping and issues of classification, but it is certainly relevant in the context of identifying cultural differences in attitudes towards pain and pain behaviours, with direct implication for diagnosis and treatment in multiethnic contexts. Numerous studies

have identified differences between various ethnic groups in terms of sensitivity, behaviours, or attitudes to both acute and chronic pain (Rollman, 2004). Although differences tend to be small or nonexistent when confounding variables such as education, work status, pain duration are controlled for, ethnicity can also act as a moderator in the relation between some psychosocial factors and pain severity (Edwards et al., 2005a).

Other characteristics of the medical condition, such as pain location, have been shown to be associated with pain quality and pain related disability. For example, a recent study by Porter-Moffitt et al. (2006) reported significant differences between 7 pain groups based on pain location (e.g. headache, lumbar, thoracic, etc.) on limitation of daily activities, perceived pain and disability, and psychosocial functioning. These associations however have not been consistently reported in the literature (Jensen and Karoly, 2001).

External contextual factors such as social support, life events, or conflictual situations also contribute to chronic pain adjustment. The role of social support in chronic pain has been researched as both an aggravating and therapeutic influence. Skevington (1995, p. 92) reviewed evidence for pain modelling in families, for the role of partners in positively reinforcing pain behaviours such as pain expression, but also for their role in reinforcing the use of pain management skills and the increase in activity levels. The relationship is apparently moderated by various other factors, such as the type of interaction, the information exchanged, the characteristics of the individual involved, etc. (p. 195). There has been an increasing refinement in modelling this relation, from the simple observation of the partner/carer's role in shaping pain behaviour in operant-behavioural therapy, to cognitive-behavioural models focusing on the interaction between the patients' beliefs and expectations and those of their social environment, to later contextual-interactional models in which patterns of interaction lead to different individual and group outcomes (Romano and Schmaling, 2001). This research suggests that marital status can either facilitate or hinder chronic pain adjustment, in interaction with other psychological variables.

Both major life events and daily hassles have been shown to be related in various ways to the pain experience: as reported antecedents of pain increases or initial treatment seeking, and as correlates of decreased use of adaptive coping strategies and increased frequency of pain behaviours (Bradley and McKendree-Smith, 2001). A particularly stressful situation is claiming for compensation benefits, which significantly affects emotional and physical functioning (Guest and Drummond, 1992).

The examples above highlight the need to include such contextual variables in studies that address the relation between psychological factors and chronic pain adjustment, either to control for their influence on both types of variables or to identify their potential moderating effects on the relationship. However, their inclusion certainly adds a new layer of complexity, and leads away from parsimony in model development.

6.2.5 Towards an integrative approach

The complexity of the interactions between different psychological aspects of living with chronic pain and between these aspects and various facets of health status and context characteristics intimidates any attempt to develop integrative models, as recommended by Keefe et al. (2004). It concurs with the FC and ACT statements (detailed in Chapter 3) that there isn't an absolute truth and a stable structure of the reality independent of context and intention, that all efforts to describe it are inevitably limited, situated, and that the application of these descriptions to specific situations is only successful to the extent that it is not taken as a invariable law. In fact, in this respect ACT is not in opposition to 'mainstream' psychological science, as the dependence on context is acknowledged as one of the main difficulties of psychological theorising by many authors, including proponents of the positivist approach, (e.g. Meehl, 1978).

Thus, it becomes essential to clarify what is the purpose of a model, what is the role of quantitative research and how does it help clinical practice in chronic pain. Is it useful and feasible to condense the information described so far, to clarify concepts and identify areas of overlap, so that we describe the phenomenon of pain more clearly and parsimoniously? The next section attempts to answer these philosophical and in the same time methodological questions. Although this topic might seem rather a detour from the main aim of the present thesis, I would argue that it is indispensable to the effort to integrate the different domains in pain research, especially given that one of them, acceptance, is built on a distinct philosophy of science described in opposition with the rest of the domains (functional contextualism versus mechanism, as described in Section 3.3).

6.3 Why do we need an integrative model?

6.3.1 Theoretical models

The general goal of all pain management efforts can be described as the preservation of health from a biopsychosocial perspective, which includes both the maintenance of an optimal functioning of the physical organism in the given conditions and the provision of the adequate support to the sufferers for living a rewarding life in their social environment. This goal may be reached however by different approaches with different theoretical assumptions, which may dictate the development of different types of models. Why would an integrative model be necessary, and what would be its place in the wider landscape of chronic pain research?

In order to clarify the use of models in pain management, I propose a distinction between three main contexts of model use, which reflect three meanings of the term. First, as the Self-Regulatory Model describes, the individuals confronted with a health threat develop their own models of the condition, which guide their search for better understanding, more effective coping skills and treatment methods. Second, the health professionals, including clinical psychologists, develop via professional training and experience their own individual models of chronic pain, which guide their work in assessing the patients' individual situations and supporting therapeutic change. Third, theoretical models are developed to guide research efforts (data collection, analysis and interpretation), finally aimed at informing and improving intervention (both self-help and therapy). Our search for an integrative model refers to the latter type of models. The use of the term 'theoretical' for research-related models does not intend to imply that individual models are not theoretical in nature (although they necessarily have a higher degree of contextualisation), or to denote superiority. The three types of models need to answer specific requirements related to their purpose, as it is detailed next. Instead, the term refers to the intersubjectivity and generalizability of a theory, in its common acception.

The three types of models serve different purposes, and thus have different characteristics. The individual models of illness are limited and continuously fluctuating depending on the specific circumstances; rigour is not required, since effectiveness of the models is strictly related to the outcomes of the coping procedures they initiate. Nevertheless, as the SRM describes, the assessment of outcomes can be biased by the use of inadequate heuristics, and a coherence between the health care provider's and the patient's models of illness is necessary for an effective management of the

medical condition. The effectiveness of therapeutic models is ensured by the flexible application of rules identified via research and professional experience to the patient's particular situation and resources, and by their seamless integration with experiential therapeutic tools, such as the therapist's communication skills and use of experiential methods, in the actual interaction. From this perspective, I would argue that functional contextualism is more suited to guide models operating at these two levels, as it focuses on the act-in-context as an analytical whole, and on pragmatic criteria for analysis, i.e. judging a model based on its practical usefulness in specific situations (Hayes et al., 1999b, pp. 18-21). Thus, FC is only apparently opposing the "mechanicist" approach, each underlying different types of models[7].

In contrast, theoretical models, while having the same ultimate goal, need to reach different intermediary objectives essentially related to their application to a wider range of similar situations, in the sense of being informative to a larger number of both sufferers and therapists. This wider applicability requires a higher degree of generalizability which implies several key assumptions: theoretical entities exist (i.e. there are common aspects between the various observed situations), they are independent of any single set of observations and have a causal role in the generation of such observed phenomena, and their predictive value resides in the accuracy with which the causal relations theorised describe the observable reality, past and future. These assumptions correspond to a realist philosophy of science and underlie one of the methodologies employed by all models examined in the present thesis: the latent variables theory (Borsboom, 2005, p. 60).

By situating the present integrative efforts at the level of theoretical models and by identifying the latent variables methodology as an important common element in the models examined, this thesis adopts realist ontological and epistemological assumptions, and chooses to examine the theories considered and the empirical data based on realist criteria, as they apply in the methodological literature (e.g. clarity of construct operationalization, unidimensionality of measurement, statistical fit with observed data)[8]. Of course, this approach should be seen as complementary to individual and therapeutic models, and also to the further development of the more specific models examined. Hayes et al. (2004c) used the metaphor of two journeys with different start and end points to highlight the fact that philosophical

[7]These two philosophical perspectives parallel the enduring controversy between positivism and hermeneutics in social sciences (and especially in clinical psychology), which have been considered as referring to two levels of investigation: nomothetic, i.e. pursuing general knowledge, and idiographic, i.e. focusing on understanding unique events (Dooremalen et al., 2007, Ch. 5).

[8]While these criteria are different from functional contexualist ones, ultimately they are also in the service of the general goal of predicting and influencing chronic pain adjustment.

assumptions are pre-analytical, "they can't really be justified, only owned" (p. 18). They suggest that their single criterion of validity is to have coherent assumptions regarding goals, methods, measures, etc. By choosing to be guided by methodology as a common element, the present approach aims to satisfy this criterion.

However, an integrative model involves distilling the richness of information in these models to extract common elements which describe the phenomenon at a more general level. Thus, information is inevitably lost and the concepts integrated, while maintaining their core elements, receive relatively different interpretations within the new model. Moreover, models articulated at different levels or guided by different philosophical approaches are subject to the Procrustean bed of the realist criteria[9]. For these reasons, the present thesis should not be seen as suggesting the abandonment of research within the specific models, but rather as an invitation to dialogue at this higher level of generalisation, which many lead to more focused research efforts, more effective training, more reliable health policies, etc. These practical applications justify research at this level, based on realist assumptions. To further clarify the criteria for theory assessment adopted in the present thesis, the common methods used in empirical testing by the models examined will be discussed next, with particular emphasis on structural equation modeling.

6.3.2 Statistical models

While individual and therapeutic models are validated by the subjective assessment of their outcomes in a particular context, testing theoretical models requires objective scientific criteria. Most studies reviewed in the previous chapters used quantitative methodology, which was also applied in the present study, therefore this subsection is dedicated to statistical model testing.

The linear model: from correlation to SEM

While theoretical models are usually developed in verbal form and describe a phenomenon on a more abstract and general level, deriving statistical models from theory requires a translation of these verbal statements in mathematical form by

[9]According to Borsboom (2005, pp. 63-68), applying the latent variable methodology means implicitly adopting a realist philosophy of science. More specifically, the realist stance can be identified formally in the application of probability theory in statistical testing at the level of individual scores, parameter estimates and model fit.

deriving predictions that apply to the specific conditions of the data collection context and expressing them in terms of relationships between variables. This translation process is essential for theory development, although it is not usually given adequate attention. Without going into a detailed methodological digression, my aim is to highlight the essential differences and requirements of the various methods in relation to testing complex integrative theoretical models, which is the main goal of this thesis. Particular emphasis is given to structural equation models, testing causal relations and longitudinal models.

Model testing may involve various levels of complexity, from deriving predictions about associations between only two variables in a specific (experimental or observational) context, to efforts of specifying complex networks of directional relationships. Correlational hypotheses can be considered a basic level of model testing, but they are minimally informative for theoretical models with multiple variables beyond the limited support for a relationship characterising a specific part of the model. A second level of testing are multiple-regression models (MRMs), which estimate simultaneously a series of equations and can test more complex theoretical predictions, such as the relationship between two concepts when controlling for possible spurious variables, the selection of the best predictors of a certain outcome from a series of candidate variables, or mediation and moderation effects (Tabachnick and Fidell, 2001, Ch. 5). MRMs focus on the optimal prediction of a single outcome, while many theoretical models stipulate sequences of relationships where a certain outcome can become a predictor in relation to a different concept. For such theories, path models (PMs) can provide a more adequate statistical test.

However both MRMs and PMs have several limitations. First, they assume that the variables are measured without error, while in many practical applications, especially in social sciences, research frequently faces intractable measurement problems, and the chronic pain area is no exception, as discussed in the previous chapters. Second, they assume that measurement errors of the included variables are not correlated, while in many substantive research areas, including chronic pain, psychometric instruments of distinct concepts are subject to similar sources of variance. Third, they assume that the psychometric instruments used to measure the included variables are unidimensional, i.e. the items used relate only to their relevant concepts and are not associated with other constructs in the model; as detailed in the previous chapters, conceptual overlap is one of the major problems of chronic pain research. These limitations lower the accuracy of estimated parameters if these

assumptions are not met and thus impact on the accuracy of the statistical test. Structural Equation Models (SEM) can provide a solution to these problems.

Developed four decades ago, SEM resulted from the combination of path modeling and confirmatory factor analysis (Schumacker and Lomax, 2004). As defined by MacCallum and Austin (2000), a SEM is a pattern of linear relationships between measured variables and latent variables; the relationships can be either directional (implying that one variable influences another) or nondirectional (where only correlation is implied). Thus, SEM opens a variety of possibilities for the development and testing of more complex models, including complex analyses such as multiple-group and hierarchical models and more integrated ways of modeling moderation and mediation (Schumacker and Lomax, 2004). In addition, SEM offers the possibility to go beyond null-hypothesis testing, which is known to subject psychological theories to only limited and often inconclusive testing (Meehl, 1978), to the comparative analysis of alternative plausible models.

The additional levels of complexity and testing possibilities don't come without limitations and methodological requirements, often neglected in SEM applications. In relation to the issue of measurement error, Bedeian et al. (1997) highlighted the fact that the appropriateness of adjusting for measurement error (both the classical "correction for attenuation" method and the SEM method) is controversial, as does not automatically lead to 'true' parameter values. In their opinion, "models are only as good as their measurement components" (p. 798), as latent reliability affects both model goodness-of-fit and parameter estimates, and the use of SEM methodology does not exempt the researcher from examining and addressing issues of latent reliability and correlated measurement errors for indicators, ensuring sample representativeness, adequate sample size, and measurement quality[10].

Measurement error is central to the use of SEM and the methodological dispute regarding the distinction between measurement and structural components of SEMs. It is also essential to the main goal of the present thesis (i.e. clarifying concepts and progressing towards an integrative model of chronic pain adjustment). Therefore a brief methodological explanation is necessary[11].

The measurement versus structure distinction in SEM parallels the distinction between induction/analysis and deduction/synthesis in theoretical models. While

[10]More arguments supporting the necessity of examining psychometric properties of measurement tools are presented in Chapter 8.

[11]The technical aspects and decisions in data analysis are detailed in Appendix B.

MRM and PM focus on the structural relationships and leave measurement issues for psychometricians (employing either classical test theory, item response theory or latent trait theory), SEM offers the possibility to integrate both components in a single model, although this potential is still rarely used in chronic pain research, as detailed in Appendix B. A first barrier is that most existing psychological instruments fail the stringent criteria of SEM models (in this case, confirmatory factor analyses). Some authors argue that these criteria are too stringent for measures with more than 3 indicators, given that more indicators are necessary for good construct validity (e.g. Marsh et al., 2004), while others encourage the efforts of improving measures according to SEM criteria, as they potentially lead to new progress in the substantive areas (McIntosh, 2007). A second potential barrier to integrating measurement and structure in the same model is the increased number of parameters to be estimated, which leads to decreased power to detect model misspecifications and requirements for increased sample size; a recommended remedy for this problem is the inclusion of selected items from existing psychometric instruments (Hayduk, 1996, p. 25–30), with the drawback of lower comparability between studies. A third potential barrier is the lack of unidimensionality of most existing questionnaires, especially when similar concepts are included in SEMs, which become apparent in model fit diagnostics and bias parameter estimates (Fornell and Yi, 1992, as cited in Hayduk, 1996; Bedeian et al., 1997). All these barriers can be seen either as recommendations for not integrating measurement and structure in SEMs, or as an opportunity for more powerful model testing and development. If failure of fit for the integrated statistical model is not interpreted as direct proof for the failure of the theoretical models (since misspecification might also occur in the translation of the theoretical model to the statistical one), I would argue that such integrated analyses can provide useful information for model development at both the measurement and structural levels.

As described in the previous chapters, chronic pain research has been focusing on both identifying reliable concepts and testing their utility based on their relationships with relevant clinical outcomes and other validated concepts. Thus, measurement and structure are closely interconnected. There is as much uncertainty about how to measure and name the various related factors as there is uncertainty about the structure of the global phenomenon of psychological adjustment. In essence, the research problem in chronic pain management is to identify aspects of the overall phenomenon that can influence other aspects considered outcomes, such as perception of pain intensity and duration, physical disability, and that can be modified via

therapeutic intervention. But the challenge for research models (as opposed to individual or therapeutic models) and their statistical counterparts is how to cut the whole phenomenon into distinct elements (amenable to self-report or other measurement methods), so as not to overlap measurement and be able to identify accurately the substantive links between these elements of the bigger phenomenon. From this perspective, I consider that the integrative use of measurement and structure in SEM can offer some answers to this problem of clarifying concepts and developing an integrative model, and will therefore be used in the present study, together with other methods.

Another major controversy in the SEM literature refers to the causal inferences that the method allows. Some authors assert that, although SEM allows modeling of causal processes with both non-experimental and experimental data, modeling non-experimental and especially cross-sectional data cannot support causal (unidirectional) relations; however in situations where experimental manipulation or even data collection at multiple time points is not possible, SEM can provide a complementary methodology for examining plausibility of models, if several alternative models are examined comparatively (Maruyama, 1998, Ch. 1). Other authors, while acknowledging the role of experimental control in testing causation, highlight that causation exists independent of human manipulation, which can also lead to the illusion of causation if other important criteria are not satisfied (Mulaik, 2009).

Bollen (1989) offers a detailed and balanced account of these criteria. He describes causality in SEM by three requirements: isolation, association, direction of influence. Isolation is rather an ideal rather than an achievable condition, and it is approximated in research designs (observational or experimental) by control or randomization. In quantitative terms, perfect isolation is replaced by pseudo-isolation, the assumption that the disturbance of the dependent variable is uncorrelated with the independent (exogenous) variable(s). If this assumption does not hold, the estimated association between the dependent and independent variables is biased; hence, the necessity of including all the factors known to be relevant to the specific relationship (common causes, additional related causes, intervening variables) and specifying adequate relationships (reciprocal causation, non-linear associations, covarying errors or disturbances) in order to increase the plausibility of this assumption. Association, although it is apparently simple to test, might be biased by heteroscedasticity or collinearity, measurement error or sampling fluctuations; hence, the importance of considering these aspects carefully. The direction of causation, brings forth the issue of temporal precedence, and of the time interval between

the actual events and its relation to the interval of measurement; hence, the relevance of elaborating the theoretical basis for the specified relations in model testing, including the possibility of simultaneous or reciprocal causality (Bollen, 1989)[12].

The above considerations show that the adequate use of the SEM methodology requires considering multiple issues, such as addressing reliability issues, the simultaneous testing of both measurement and structural levels (preferably without using total scores), the causal inferences allowed by the study design (e.g. cross-sectional). The methodological literature also recommends the comparative analysis of alternative and equivalent models, considering multiple diagnostics in addition to global model fit for model interpretation, the comprehensive reporting of the results, etc., which are rarely considered in practice. A more detailed methodological discussion of SEM is presented in Appendix B, together with several examples of studies applying SEM in chronic pain research.

In modeling longitudinal data, an important issue is represented by the multiple measurements for each case, which allow, or indeed make necessary, the separation intraindividual variance from interindividual differences, and the examination of time-related variability in the form of growth trends. Hierarchical longitudinal models (HLM) and latent growth models within SEM address these issues and are therefore more adequate for longitudinal theory testing than correlation, MLMs, or PA (Singer and Willett, 2003). Although temporal precedence is necessary for testing causal models, an additional requirement is equally important: eliminating the alternative hypothesis of reciprocal causality. Singer and Willett (2003) distinguished between defined (predetermined, e.g. gender), ancillary (determined by an external stochastic process, e.g. weather), contextual (determined by a proximal stochastic process, e.g. peers' behaviour) and internal/endogenuous (measuring the individual's status, e.g. psychological, physical or social characteristics) predictors. When data are measured simultaneously and are either contextually determined or targeting similar internal processes, causal interpretations have a weak support due to the possibility of reciprocal causality. They recommended the use of time-lagged designs, where prior assessments of predictors relate to subsequent outcomes.

The advantages of SEM and HLM do not imply that methods such as correlation and multiple regression are less valuable for testing theoretical models. The methods provide complementary information and can be used together in both exploratory data analysis and theory testing.

[12]To these criteria, Mulaik (2009) briefly adds the necessity to assume causal homogeneity, to specify a relevant context, and to identify points of equilibrium in change processes.

In summary, the use of linear modeling, in particular SEM, for theory evaluation imposes several related criteria regarding measurement quality (reliability, validity, unidimensionality) and the relationships between variables (isolation, association, direction of influence), which translate the realist philosophical assumptions at a methodological level and will further guide our data analysis.

6.4 Selecting an existing model versus developing a new one

In order to gain an integrative understanding, it is useful to identify a common ground for the theories involved and adopt a consistent approach. Thus, in addition to the criteria discussed above, an integrative model be characterised by an ability to accommodate findings from different models and by external consonance with related research. Chronic pain research already has a variety of models, some of them presented in the previous chapters. These may be more general models applied to chronic pain (e.g. the ACT, the DMA or the SRM), specific to chronic pain (e.g. the cognitive-affective model of the interruptive function of pain; CAM, Eccleston and Crombez, 1999), or detailing a particular aspect (e.g. the cognitive-behavioral mediation model of depression in chronic pain of Rudy et al., 1988, or the fear-avoidance model of exaggerated pain perception of Lethem et al., 1983). Therefore, before deciding on building a new integrative model as recommended by Keefe et al. (2004), it is necessary to explore the existing models in chronic pain research and assess their adequacy in explaining the relevant behavioural, emotional and cognitive aspects discussed in the previous chapters. This conceptual analysis attempts to follow the advice of Diefenbach et al. (2008, p. 656–7) in model development:

> model building should not consist of including an increasing number of variables in ever more complex models. In contrast, we suggest that researchers rigorously evaluate variables and their hypothesized relationships to other factors in a given model. These tests should be conducted both experimentally in laboratory settings that simulate the appropriate health contexts, as well as naturalistically in the field, employing both quantitative and qualitative methods. Only then can we be confident that our theoretical models are valid for predicting health-related behaviour.

In my opinion, among the theoretical models reviewed so far, two can be considered to have sufficient breadth to potentially accommodate research findings from the

rest of the models while adhering to the realist assumptions set by our approach: Leventhal et al.'s (1992) SRM and Eccleston and Crombez's (1999) CAM. Importantly, they share many characteristics. A central common aspect is the focus on the interaction between cognition and emotion in guiding behaviour, which places them in a good position to integrate findings from more specific models. In addition, they both build on the strengths of previous mainstream psychological theories in emotion research, cognitive psychology and operant-behavioral theory (in health psychology and chronic pain research respectively) and have therefore the resources to maintain relatively accurate interpretations of the concepts integrated. They also both have the potential to accommodate the shift in chronic pain research from choosing between opposing unidirectional relationships (e.g. pain causes depression versus depression causes pain) to the description of the dynamic interdependence between the psychosocial context and pain itself, which is considered a much needed perspective change (Jacob and Kerns, 2001, p. 363).

These two models are also complementary in several ways, as they are both in early stages of testing and development, and each describes conceptual aspects of the psychosocial adjustment to chronic pain that the other has developed to a lesser extent. For example, as described in Chapter 5, in the SRM the emotion component is not extensively developed, although it is central to its theoretical construction. The SRM is also a more general model of response to health threat, and thus it does not consider the specific effects that pain itself (as a symptom) has on behaviour. Its focus on health threats leads to a limited consideration of other motivations in the person's life and of the possible conflicts with coping with the illness threat (although it acknowledges the necessity of studying health behaviour in the wider intrapersonal and interpersonal contexts).

To all these limitations, the CAM has partial answers. Although the emotional responses to pain are not extensively developed in this model either, it stipulates an influence of emotion at both the level of the pain stimulus selection (via habitual ways of interpreting threat, or emotional response to other stimuli in the environment) and the level of the chronic problem-solving efforts. Its focus on attention processes, based on the specific function of pain to interrupt ongoing activity and motivate escape behaviours, takes into consideration the specific characteristics of the main symptom in chronic pain. Moreover, the interpretation of the sufferer's focus on pain as essentially an adaptive behaviour reduces the stigma usually associated with efforts to reduce pain perception which usually comes with seeing

avoidance as an essentially maladaptive process leading to suffering. CAM specifically describes the conflict between pain-motivated escape and ongoing activities motivated by the other life goals, thus introducing an essential element for therapeutic intervention.

On the other hand, the CAM has a series of limitations to which the SRM can bring welcomed additions. The factors that influence the perception of threat and thus lead to the selective attention to pain signals are only briefly mentioned in the CAM, while the most developed aspect of the SRM refers to the perceptions of the health threat, admittedly mostly at the cognitive propositional level, as reviewed in Chapter 5. The cognitive evaluation of outcomes of the problem-solving process is also underdeveloped in the CAM; the process leading from misdirected problem-solving to reframing the pain problem adaptively needs a more detailed account of these assessment components. The SRM's focus on assessing health threats and coping outcomes via cognitive and affective heuristics that might bias the evaluation and thus lead to perseverance in incongruent illness perceptions and ineffective coping procedures offers a way to study this process.

The other models reviewed, while having a more limited scope, can be reinterpreted within the two broader models and bring complementary details to the general outlines. Within ACT, mainly due to its behavioural lineage and its unique RFT foundations, emotion and cognition tend to be grouped as functional classes of behaviour and labelled 'private events'. Consequently, ACT does not view emotional and cognitive concepts in the way that other emotion and cognition researchers do, but emotional and cognitive elements can be identified in many components of its distinct theoretical construction. Thus, ACT may bring a valuable contribution. Acceptance may be interpreted within the SRM in terms of affective heuristics (e.g. anxiety is bad), coping procedures (avoidance versus engagement in activities), and illness perceptions (e.g. of controllability). The CAM specifically redefines acceptance as reformulating of the pain problem in terms of reducing disability and distress (i.e. an antecendent-focused emotion regulation strategy), but as detailed in Section 6.2.1, it can also be assimilated to response-focused emotion regulation strategies[13].

The existing models of pain processing, such as the gate control theory, the neuromatrix model, Price and Harkins's (1992) sequential processing model or Leventhal

[13]A yet unexplored aspect of acceptance can be related to the third category of emotion regulation in Gross's (1999) classification, i.e. selecting and modifying the antecedent events via attentional processes, which has obvious connections to the CAM's focus on attention.

and Everhart's (1979) parallel processing model of pain distress (PPM), do not directly address the phenomenon of adjusting to chronic pain. However these models need to be consonant with and complement such a model of chronic pain adjustment. The PPM offers precisely such a link between neurophysiological models of online pain processing, especially in acute pain, and psychological models of pain response that extend to chronic pain adjustment, such as the CAM and SRM.

The models that focus on specific affective responses are inevitably limited to these particular aspects, but can be also integrated in the two broader models. The research on anger expression and suppression offers an excellent example of schematic affective processing of health threats for the SRM, and can be considered as related in a cyclical manner to both perceptions of stimulus threat directing attention to pain stimuli and effect of the misdirected problem-solving efforts in CAM. The fear-avoidance models (Lethem et al., 1983; Vlaeyen et al., 1995) were fundamental for the development of the CAM and have already been integrated and reinterpreted in more functional terms in this model, as catastrophizing and avoidance behaviours are viewed as consequences of problem-solving efforts in the context of chronic pain. The perception of health threat and the related coping procedures in the SRM also include cognitive and emotional appraisals of danger and avoidant coping, among others. Depression accounts such as the cognitive-behavioral mediation model (Rudy et al., 1988) refer to another limited category of perceptions of the health threat (i.e. of reduced control and self-efficiency) and behaviours (i.e. reduced instrumental activities) as leading to lower adjustment, and thus can be integrated as part of the problem-solving efforts in the CAM, or the illness perceptions and coping procedures in SRM. The limited account of shame could also be considered as part of the affective content of the perseverance loop involved in solving the problem of pain in the social context. The SRM proposal that pain can become associated to specific emotions including shame is an open invitation to further explore its role in chronic pain. The DMA account on positive emotions, while limited to the interplay between positive and negative affect, describes a dynamic interplay between stressful stimuli (particularly pain), the individual characteristics and the momentary circumstances. This dynamic, although it refers only to the affective aspects of chronic pain from a dimensional perspective, overlaps conceptually with the continuous competition between the pain-motivated escape behaviours and approach behaviours motivated by other personal goals described in CAM, and with the cyclical updating of illness models in the SRM. Thus, it represents a good

example of the study of interactions that can be applied to other areas of the wider models, including the cognitive components[14].

This brief conceptual analysis has attempted to answer the question of whether a new integrative model is necessary, given the variety of existing models. It suggests that further development of a model of chronic pain adjustment starting from the preliminary outline of the CAM within the broader framework of the SRM has the potential to offer the integrative approach needed. Certainly, a general model can be complemented by the formulation and testing of detailed hypotheses regarding specific components, which would enrich and update the broader model and provide sufficient detail for its application to specific contexts. It is within this framework that the hypotheses of the present study were formulated, tested and interpreted.

6.5 Research aims and hypotheses

A first empirical step towards an integrative model, as Diefenbach et al. (2008) suggested, consists in thorough examinations of current concepts and their relationships. To this end, concepts such as psychological flexibility, chronic pain acceptance, discrete emotions (anger, sadness, fear, shame, happiness), emotion regulation strategies, illness perceptions, together with indicators of health status and demographic and context factors, were measured via self-report in a heterogeneous sample of chronic pain sufferers at three time points, in order to assess their dynamic interrelations. Specific predictions were formulated based on results of previous studies and theoretical implications derived from the current models[15]. The four research areas (acceptance, emotions, illness perceptions and health status) were first explored via correlational analyses at a cross-sectional level, and the stability of the concepts is examined at a longitudinal level. Their interrelations were examined subsequently, both between each of the three psychological domains and their separate and combined interactions with health status. The hypotheses

[14]Other models not described so far address either specific mechanisms or general considerations that may be also integrated in the two models but will not be discusses here for reasons of brevity. Examples include: the Glasgow model (Waddell and colleagues, as described in Asmundson and Wright, 2004), the biobehavioral model (Turk and Flor, 1999, as described in Asmundson and Wright, 2004), the diathesis-stress model for chronic pain (Asmundson and Wright, 2004), the diathesis-stress model of depression in chronic pain (Banks and Kerns, 1996), the avoidance-endurance model (Hasenbring et al., 2009).

[15]Multiple hypotheses were formulated where existing theory and research allowed, in accordance to long-standing guidelines (Chamberlin, 1890) particularly suitable to current statistical methods (Elliott and Brook, 2007). The advantages of using multiple working hypotheses (whether alternative, sequential or simultaneous) would deserve a more detailed treatment. However for reasons of brevity I will only note the revived interest in using these guidelines in the context of testing multiple causal models of complex systems, as discussed in Elliott and Brook (2007).

and their justification are described in the next subsections. The next two chapters detail the data collection and analysis. The results are discussed in Chapter 9.

6.5.1 Separate examination of the four areas: acceptance, emotions, illness perceptions, health status

At the cross-sectional level, correlational analyses were used to explore each of the substantive areas. For reasons of brevity, separate hypotheses regarding each of the correlations between the relevant concepts are not explicitly stated here. The analysis and interpretation of the results was based on the expected associations according to existing theoretical and empirical literature. For example, CPA and PF scores were expected to correlate significantly (although the discriminant validity of the two measures was considered an exploratory issue), negative emotions were expected to be associated to dysfunctional regulation strategies, health status variables were expected to correlate, but measure distinct aspects.

A special attention was given to illness perceptions, as the question of how illness perceptions are interconnected has been only partially answered in the SRM literature. As discussed in Chapter 5, BIPQ has been used by computing total scores (Radat et al., 2009), which would suggest unidimensionality, but also by identifying distinct patient groups (Hobro et al., 2004), which would suggest that a categorical approach is preferable to a dimensional one. To clarify this controversy, the data were analysed via exploratory factor analysis and cluster analysis. It was hypothesised that, given the previous empirical results, BIPQ subscales do not belong to a unidimensional construct. It was equally relevant to replicate the results of Hobro et al. (2004) in the present study, as a first step towards identifying the relevance of these groups on health status and the relationships with the other concepts, and to examine the adequacy of this solution in terms of distinctiveness and, longitudinally, stability. A similar analysis was performed in relation to CPAQ scores, based on Vowles et al.'s (2008b) cluster analysis.

The role of socio-demographic and medical characteristics was also explored in relation to each domain. At the longitudinal level, the stability of each measure was explored, as a preliminary step for integrative analyses.

6.5.2 Interrelations

Specific hypotheses were formulated for the examination of the relationships between acceptance, emotions and illness perceptions, in light of the literature reviewed in the previous chapters and in Section 6.2.

Emotions and acceptance

As detailed in Subsection 6.2.1, acceptance can be described as an adaptive emotion regulation strategy related to the frequency of experiencing various positive and negative emotions. Also, Phillips and Power (2007) described dysfunctional ERSs in terms of rejecting and ignoring the informational relevance of emotional content, and functional ERSs as 'holding' and processing the emotional content; this distinction is similar to the acceptance-avoidance contrast. Therefore it can be hypothesised that:

Hypothesis 1: Both CPA of PF are related to more frequent positive emotions and functional ERSs, and less frequent negative emotions and dysfunctional ERSs.

Illness perceptions and acceptance

Although Hobro et al. (2004) did not specifically address the differences between their 'adaptors' and 'non-adaptors' groups in terms of acceptance[16], the theoretical similarities between illness perceptions and acceptance are substantial, as described in Subsection 6.2.3. Therefore another hypothesis is that:

Hypothesis 2: Non-adaptors (as identified by cluster analysis of illness perceptions) have significantly lower scores of acceptance (CPA and PF) compared with adaptors.

The relationships between specific IPs and acceptance were further analysed in an exploratory manner.

Illness perceptions and emotion

The differences between 'adaptors' and 'non-adaptors' in terms of their emotional life was not a main focus in Hobro et al. (2004), which only addressed general levels of anxiety and depression as indicators of psychological distress. However the SRM stipulates that emotions can be generated by IPs, and the individual's response to

[16]They only used measures of patients' beliefs about their medication, pain intensity, anxiety and depression and subjective perceptions of general health.

health threats is the results of an interplay between distinct yet related affective and cognitive factors, suggesting that:

Hypothesis 3: Non-adaptors report significantly more frequent negative emotions and use of dysfunctional ERSs and less frequent positive emotions and use of functional ERSs compared with adaptors.

The relationships between specific IPs and specific emotions are more difficult to hypothesise based on the SRM, as this aspect of the model is underdeveloped. They were therefore analysed in an exploratory manner.

6.5.3 Exploratory analysis

Three types of additional exploratory analyses were conducted. First, the relationships between all three types of psychosocial factors and health status were explored comparatively at a cross-sectional level, and the possibility of developing longitudinal models based on the results of these analyses was further explored. Second, the variance in individual growth trends of chronic pain adjustment was explored with a focus on identifying explanatory variables for existing trends. Third, the concept of *discrimination ability* was defined based on existing literature and explored in the present data set in relation to sensory-affective distinctions in pain perception and discrete emotion distinctions.

Health status and psychosocial variables

A first step towards distinguishing the unique contribution of psychosocial variables to health status is to identify the contextual variables which might explain part of the variance. Thus, for each health indicator, exploratory multiple regression models were performed with the following potential predictors: gender, age, education, annual income, marital status, pain location, pain spread, pain duration, age at pain onset, previous and current pain treatment (surgery, physiotherapy, complementary therapies), comorbidity (and separately presence of osteoarthritis), negative and positive life events (total scores and selected events, such as recent injuries or financial problems). The significant predictors were used as control variables in the subsequent analyses.

Previous research on CPA (reviewed in Chapter 3; McCracken and Eccleston, 2003; Viane et al., 2003; McCracken and Eccleston, 2005, 2006; Vowles et al., 2007a; McCracken and Vowles, 2008) showed that participants who report more acceptance

of chronic pain also report better health status in terms of physical and psychosocial disability, vocational status, healthcare utilisation and pain intensity, controlling for the relevant context variables. Therefore, it can be hypothesised that, also in this sample:

Hypothesis 4: CPA and PF are associated with better health status (health care utilisation, pain intensity, pain-related disability, work status), controlling for contextual factors.

As detailed in Chapter 4, the relationship between discrete emotions, emotion regulation strategies and health status in chronic pain patients is reflected in multiple ways in pain perception, health care use and disability. Emotion determines the way the sufferers attend to and assess pain stimuli and their choice of coping procedures, and is in turn influenced by the intensity of the symptoms and degree of limitation on the daily activities and personal goals. A general hypothesis can be formulated:

Hypothesis 5: Lower distress and less frequent use of dysfunctional ERSs are associated with better health status (health care utilisation, pain intensity, pain-related disability, work status), controlling for contextual factors.

Discrete emotions were also introduced separately in MRMs to explore their unique associations with specific health status indicators, following Fernandez and Milburn's (1994) stepwise regression of 10 discrete emotions in relation to the A-PRI index.

As described in Section 5.3.2, Hobro et al. (2004) described the two patient groups they have identified based on their illness perceptions ('adaptors' and 'non-adaptors'), as different in terms of health status indicators. Therefore it was hypothesised that:

Hypothesis 6: Non-adaptors report significantly better health status compared with adaptors.

Distinct illness perceptions were also introduced separately in MRMs to explore their unique associations with specific health status indicators.

The results of the separate analyses were introduced in integrative MRMs in order to compare the contributions of all relevant factors to predicting health status indicators. The relations between these factors were further explored in an integrative measurement SE model.

Individual growth trends

The above exploratory and confirmatory analyses have focused on the relationships between concepts at group level and in a cross-sectional format. However longitudinal study design allows a different, 2-level approach of the potential intra- and interindividual change processes. Therefore a separate exploratory analysis will attempt to characterise the between and within-subject variance of the participants' scores on the psychological and health status indicators during the study period via hierarchical longitudinal modeling (HLM).

The issue of discrimination ability

Several theoretical and empirical efforts in the chronic pain literature, some already reviewed in the previous chapters, share an interesting point regarding the individual's ability to distinguish between different aspects of the chronic pain experience, such as between sensory and affective components of pain perception, or between different emotional reactions, or between pain and emotion. This ability has potential applications in pain management and is worth exploring separately in the present study. I will briefly summarise next the description of this idea in the works of the different authors, and then formulate some starting points for this exploratory analysis.

A first account of this ability to discriminate between separate aspects of chronic pain was presented in Fordyce's (1976) description of the "vicious cycle effect" in chronic pain, where the frequent association between distress and pain makes discrimination between these states increasingly difficult, which he termed "discrimination error". He used this explanation as an argument against interpreting present distress as an indicator of a psychological causation of chronic pain (i.e. psychogenic pain). His concept of "operant pain" was thus related to the increase of this association with time, and he viewed treatment as an attempt to disentangle distress (and pain behaviours) from the pain itself.

This important aspect of the pain-distress relationship has been apparently lost in early CBT accounts of chronic pain, which refer instead to a desynchrony of subjective, physiological and behavioural aspects of pain as influenced by personality, attitudes, expectations (Phillips, 1977, as cited in Lethem et al., 1983). This idea was further developed in the fear-avoidance model of exaggerated pain perception (Lethem et al., 1983), which stipulated that stressful life events, personal pain history, coping strategies and behaviour patterns increase the probability of avoidance

responses and thus lead to a dysfunctional desynchrony, when affective responses are more intense than sensory responses. Desynchrony was also described between affective and sensory components of pain (Phillips and Hunter, 1981, as cited in Lethem et al., 1983). Avoidance behaviours were associated only with the affective component, not the sensory component of pain, pointing to the specific properties of the affective components in stimulating escape, as detailed also in the CAM (Eccleston and Crombez, 1999). The mechanisms by which pain motivates avoidance more in certain contexts were not detailed in this model, and only recent research has started to shed some light on the discrimination mechanism, as described below.

The ability to discriminate between various aspects of the pain experience can actually be considered one of the main targets of the CBT approach (Turk et al., 1983), which starts with the assessment and reconceptualisation of the sufferer's situation. In essence this stage targets the transformation of an undifferentiated, overwhelming problem into distinct, manageable problems. ACT follows on similar lines, as chronic pain acceptance involves the discrimination between the presence of pain and the availability for value-based activities, which resembles the distinction between the sensory-informational aspect of pain and its motivational-emotional component which competes with other current motivations.

The SRM and especially its related parallel processing model of pain distress (Leventhal and Everhart, 1979) also highlight the necessity of a distinction between the sensory-cognitive aspects of pain (or any other health symptom) and its emotional aspects. The clinical application to diminishing acute pain related to medical interventions via conscious exposure to sensory information prior to medical procedures is a powerful argument for the value of this discrimination ability. Eccleston and Crombez's (1999) elaboration of the CAM also includes a discussion on the dissociation between pain and threat. According to CAM, the threat value of the pain stimulus moderates its selection over competing stimuli/demands, thus enhancing its interruptive function. Other moderators are intensity, novelty, predictability. Structural relations between moderators are not known, but the authors state that clarifying these relationships would need a clarification of current concepts, such as somatic awareness, negative affectivity, anxiety sensitivity, fear of pain, catastrophizing.

As described in Chapter 4, appraisals of threat characterise the affective component of pain, and related affective responses. It follows that operating a distinction between the pain stimulus and its affective value may enable a reinterpretation of the signal and thus a potential decrease not in the sensory properties, but in its

ability to motivate the interruption of ongoing activities and initiation of escape behaviours. Also, Eccleston and Crombez (1999) proposed that the presence of competing environmental demands might also mediate pain's interruptive function: relevant events can have priority above pain. Relevance implies motivational value, therefore emotional value. Thus, a dissociation between pain and emotion may also lead to decreasing the motivational properties of pain, and replacement of escape behaviour with approach behaviours motivated by competing goals, thus leading to a decreased association between pain and escape behaviour. This phenomenon is similar to a certain extent to the counterconditioning procedure in animal research on fear conditioning, largely unexplored in humans, according to Eccleston and Crombez (1999).

The dispute regarding catastrophizing as a coping procedure or an expression of emotional distress (see Chapter 5) can also be formulated in terms of discriminating between pain and emotion. Adhering to expressions of intense distress related to pain perceptions is potentially a reflection of the difficulties the individual faces when confronted with intense pain, particularly in distinguishing between pain and affect. Elaborating this difficulty in terms of maladaptive cognition might be less appropriate than a formulation in terms of an affective ability. In diagnosis and intervention, an affective approach would lead to acknowledging the enhanced distress and seeing it as an effect of different contextual factors and identifying together areas of change in the environment and learning emotion regulation strategies (including acceptance), instead of correcting the cognitions which, as Vlieger et al. (2006) stated, might be only a by-product of the efforts to solve the problem of pain.

Discriminating between pain and emotion is also reflected in the ACT concept of relational framing and in its therapeutic goal of changing not the content, but the function of mental events by enhancing the flexibility of the relational frames in which the events participate. In emotion research, concepts such as emotion granularity (Barrett, 2006b) or mood clarity (Salovey and Mayer, 1990, as cited in Zautra et al., 2001) tap into similar issues of distinguishing between different aspects of the experience in order to generate more adequate behaviours, and the Dynamic Model of Affect also stipulates that "the degree of complexity in individuals' awareness of their own emotions is significantly reduced during times of stress" (Zautra et al., 2005, p. 212).

Limited empirical research has been conducted in this respect in the chronic pain area, mainly via moderation analyses. Affleck et al. (1992) reported that the relation between daily pain and mood is moderated by neuroticism (increased neuroticism leads to lower correlations) and by illness duration, disability, disease activity and average daily pain (all leading to higher correlations). Zautra et al.'s (2001) study described in Subsection 4.4.6 reported a moderating effect of positive emotion on the relation between pain and negative affect, the latter decreasing in the presence of the former. Mood clarity, defined as the ability to differentiate emotional experiences, also increased the differentiation between negative and positive affect. The results were confirmed and extended by Zautra et al. (2005) and Strand et al. (2006). Kratz et al. (2007) linked acceptance with these moderating mechanisms, suggesting that the differentiation ability might be an essential component of chronic pain acceptance. Also, Conner et al. (2006) identified a hidden moderating role of depression history on the strength of contingencies between daily pain and emotion-related experiences, despite a lack of correlation with mean daily ratings; depression status, although associated with interpersonal differences in daily ratings, did not have this moderating effect. Importantly, these studies show that the discrimination ability is both context- and person-related and therefore might be manipulated by both changes in the individual's environment and by skills training.

This discrimination ability can be tested in the present study via moderation analyses. A first aspect is the distinction between affective and sensory pain qualities. As discussed in Chapter 2), the distinctiveness between the S-PRI and A-PRI indices of McGill Pain Questionnaire is controversial, some studies indicating a lack of distinction, while others supporting the different sensitivity of the two indices in certain situations. Therefore, exploratory moderation analyses were performed for acceptance, discrete emotions and ERSs, illness perceptions, other health status indicators and various contextual variables. A second possible distinction is between pain and emotional distress, which is likely to be influenced by positive emotion as suggested by the studies mentioned-above; other moderators could also be explored. A third aspect is the distinction between discrete emotions. The concept of discrimination ability suggests that correlations between emotions might be moderated by various personal and contextual characteristics, therefore exploratory moderation analyses were also performed for acceptance, ERSs, illness perceptions, health status and control variables.

6.6 Conclusion

This chapter explored the research focusing on the interactions between acceptance, emotions and illness perceptions and on the role of contextual variables in chronic pain adjustment. Given the complexity of the various aspects examined so far, the utility and feasibility of developing an integrative model was briefly addressed at a philosophical and methodological level. It was next proposed that the CAM (Eccleston and Crombez, 1999) and the SRM (Leventhal et al., 1997) could offer sufficient support for further integrative efforts, and other existing models could be considered as corresponding to, or complementing, various elements of the two models.

Based on the theoretical analyses, specific hypotheses were formulated for the separate research domains and the possible interactions between them. Also, three additional exploratory aims were set. The first refers to the relation of the three domains to health status indicators, the second addresses intra- and interindividual variance and time-related trends and fluctuations, while the third sets to explore various moderation mechanism in relation to the proposed concept of 'discrimination ability'.

Chapter 7

Basic emotions, emotion regulation strategies, acceptance, illness perceptions and health related outcomes in a sample of chronic pain sufferers - a longitudinal study

7.1 Introduction

As detailed in the previous chapters, research on the role of psychological factors in living with chronic pain has usually been channelled on three separate lines of research: acceptance, emotions and illness perceptions. Few attempts have been made to compare these three areas and integrate them into more comprehensive models. The goal of the present study was to investigate the relationships between basic emotions, emotion regulation strategies, acceptance, illness perceptions and health status indicators in chronic pain. Perceived pain intensity, healthcare utilisation, and perceived physical and psychosocial disability were considered health status indicators.

7.2 Methodology and procedure

The study was conducted with the approval of the Lothian Research Ethics Committee and the Research and Development Office of NHS Lothian. As one of the main objectives was to study the relationships in time, the design of the study was longitudinal. It included 3 stages, at 4 month intervals. The duration of the intervals was selected for both theoretical and practical reasons. Theoretically, the effectiveness of an intervention is usually considered within similar intervals (e.g. 3 months in McCracken et al., 2005, 3.5 and 6.5 months in Wicksell et al., 2009, 6

months in Dahl et al., 2004). Also, the retrospective recall bias was shown to be lower for intervals around 3 months (Von Korff, 2001). Logistically, it was estimated that the necessary number of participants will be contacted in this interval, so that the data collection waves do not overlap.

The data were collected via a questionnaire including several validated instruments. The questionnaire method was selected mainly for logistic reasons: it is the least demanding form of measurement for participants in these contexts, while its reliability and validity are not considerably lower compared to other methods of pain measurement, as discussed in Chapter 2.

Participants were recruited mainly from the NHS Lothian Chronic Pain Service users, but also from the users of three chronic pain support organisations. Three inclusion criteria were applied for participant selection: adult age (18 years and above), knowledge of English language and ability to understand the information presented in the Patient Information Sheet, consent form and questionnaire. The exclusion criterion was the presence of malignant chronic pain. For the NHS sample, the selection was done via analysis of information available in the patients' files regarding date of birth, previous history of medical conditions and any additional relevant notes (e.g. the request of a translator in previous consultations). For participants from chronic pain support organisations, the inclusion criteria were presented in the patient information document and verification was based on self-report of age (completing the questionnaire was considered as an indicator of meeting the other two inclusion criteria), while the exclusion criterion was verified based on self-report of comorbid conditions.

7.2.1 Questionnaire description

The main variables included and their corresponding measures are described in Table 7.1. Other variables included were ethnicity, nationality, pain duration, previous treatment, current treatment[1]. The rationale for including these variables is detailed in Chapter 6.

The first stage postal questionnaire for NHS participants had the following structure: an introduction, questions regarding demographics, questionnaires related to general concepts, questions related to the medical history, questionnaires related to chronic pain related health outcomes and psychosocial factors. This structure

[1]Part of the participants also responded to questions regarding diagnosis, benefits, litigation, social support.

Variable type	Variable name	Measurement
Health related outcome	vocational (employment) status	single question
	healthcare utilisation	(Andersson et al., 1999)
	medication use	(Andersson et al., 1999)
	self-help	(Andersson et al., 1999)
	pain intensity	SF-MPQ
	pain-related disability	RM-SIP
Psychosocial factor	emotions	BES
	emotion regulation	REQ
	acceptance	CPAQ, AAQ
	illness perceptions	BIPQ
Control variable	gender	single question
	age	single question
	education	single question
	marital status	single question
	annual income (perceived)	single question
	positive and negative events	LTE
	pain duration	single question
	age at pain onset	age - pain duration
	pain location	open-ended question or questionnaire online
	comorbid conditions	SACQ

Table 7.1: Variables and measurement

was intended to ease response by guiding the participant from general to specific questions. The introduction consisted in a brief presentation of the study, details on confidentiality and contact details for the study investigator and organising institution (as a summary of the patient information sheet). Several validated instruments were included: the Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004b), the Basic Emotions Scale (BES; Power, 2006), the Regulation of Emotion Questionnaire (REQ; Phillips and Power, 2007), the Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004a), the List of Threatening Experiences (LTE; Brugha and Cragg, 1990), the Brief Illness Perception Questionnaire (BIPQ; Broadbent et al., 2006b), the Sickness Impact Profile Roland Scale (RM-SIP; Roland and Morris, 1983), the Short Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987), the Self-Administered Comorbidity Questionnaire (SACQ; Sangha et al., 2003). Illness history (pain duration, previous treatments, how the pain started), pain location, current treatment, healthcare utilisation (adapted from Andersson et al., 1999) were assessed via single items or sets of similar items. Demographics included gender, date of birth, education, marital status, vocational status, annual income, ethnicity, nationality. The questionnaire ended with details regarding the participation in the next two stages, a request for the participant's and their GP's contact info, questions regarding whether they would like to receive a report of

the study by post, whether they received help to fill the form, the duration of survey completion, and a space for comments. The first stage questionnaire for NHS participants is presented in Appendix A. In the second and third stage, only the questionnaires and questions that measure time-varying predictors and outcomes were included in the survey[2].

The validated instruments used are described next. As Chapter 8 is dedicated to the analysis of their psychometric properties in the present study and includes detailed information about their published reliability and validity support, it will not be reiterated here for reasons of brevity. The next paragraphs describe only their purpose and format, while the results of the psychometric analysis on the present sample are summarised in Section 7.5.

The Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004a) is a 9-item single-factor scale which measures experiential avoidance, defined as unwillingness to experience particular private events (memories, emotions, thoughts etc.) and efforts to "alter the form or frequency of these experiences or the contexts that occasion them, even when these forms of avoidance cause behavioral harm" (p. 554). The theoretical relationships between experiential avoidance, acceptance and psychological flexibility in Acceptance and Commitment Therapy are reviewed in Chapter 3. The 9 items focus on issues such as cognitive entanglement, need for emotional and cognitive control, behaviour inhibition due to emotional or cognitive private events, negative evaluation and fear of feelings, etc. The response scale is a 7-point Likert scale with labels ranging from 'never true' to 'always true'.

The Basic Emotions Scale (BES; Power, 2006) assesses self-reports of the frequency of experiencing 5 basic emotions: anger, sadness, disgust, fear and happiness. Each of the 5 emotions is represented by 4 emotion terms (e.g. anger by anger, frustration, irritation and aggression). The underlying theoretical assumption is that "the 'true' basic emotion can best be represented as an underlying latent variable for which the terms such as 'disgust', 'anger', 'anxiety', and so on, provide observed or manifest variables which are indicators of the theoretical latent variable" (Power, 2006, p. 697). The respondents were instructed to answer how much they experienced each

[2]The questionnaires presented both to the online participants and the members of Pain Association Scotland (PAS) via post included several feed-back questions related to the activity of PAS (which will not be discussed here), and a few additional questions considered at the time potentially useful as control variables: knowledge about the diagnosis of their medical condition, whether they received benefits for it, whether they were involved in litigation, and the perceived social support from family, friends, doctors, etc. However due to the small size of this sample these responses were not included in the main analysis.

emotion via a 7-point Likert scale, from 'never' to 'very often'. The scale is based on previous research of semantic analysis of emotion terms (Oatley and Johnson-Laird, 1987). Two versions of the scale exist: a trait-like scale (how often they experienced the emotions 'in general') and a state-like scale, ('during the past week'). The trait-like scale was selected for this study for all three stages, as the one-week interval of the state version was considered too short in comparison to the 4-month intervals structure of the data collection protocol, and specifying longer intervals would have resulted in an increased response difficulty.

The Regulation of Emotion Questionnaire (REQ; Phillips and Power, 2007) assesses emotion regulation strategies based on two dichotomies: external/ internal and functional/ dysfunctional. Dysfunctional strategies are characterised by rejection of the emotion, via the use of internal resources and inhibition (internal) or via using other people, objects or emotional expression (external). Functional strategies are defined by acceptance of emotion via use of internal resources and mechanisms such as positive reappraisal, concentration and learning (internal) or via using external resources such as social support and acting to modify the initial situation (external). Items were theoretically derived and categorised based on existing literature on emotion regulation. They refer to emotion regulation strategies that are employed by the general population irrespective of age (although the questionnaire was validated on an adolescent sample). Each of the 4 resulting subscales is represented by 5 items, with the exception of the 6-item external functional scale. [3]. The respondents were required to report on how often they responded to their emotions in general by using each of the 21 strategies; the answers were given via a 5-point Likert scale, from 'never' to 'always'.

The List of Threatening Experiences (LTE; Brugha and Cragg, 1990; Brugha et al., 1985) is a set of 12 life event categories such as serious illness or injury, death of a close person, relationship difficulties, work and financial difficulties, etc., considered as having an "aetiologically significant rating of marked or moderate long-term threat" (Brugha et al., 1985, p. 189). Although the method was initially designed to identify events that are highly relevant to depression, the LTE is also recommended by the authors for use as a measure of external stress (adversity, or contextual threat) in studies that simultaneously assess variables such as social support, coping, and cognitive aspect in various psychiatric, psychological or social settings (Brugha and Cragg, 1990). A similar but briefer list of positive events was provided in

[3]Two additional items were included for this subscale for measure improvement purposes, given the problems encountered previously in questionnaire development which resulted in a shorter 4-item subscale.

order to assess the possible protective effect of individual external context. The respondents had the possibility to add 3 additional events of each type in case they considered important to mention events that did not belong to any of the categories provided.

The Self-Administered Comorbidity Questionnaire (SACQ; Sangha et al., 2003) assesses the extent and severity of comorbid conditions by patient self-report. For various practical reasons it is a more suitable measure to use in research in clinical and health services settings in comparison to collecting information from medical records or administrative data. The instrument consists of a list of 12 frequent medical conditions described in simplified language plus the option to add 3 other conditions (5 options were provided in the present study). The respondents were asked if they had each of the problems, and, given a positive response, if they received treatment (to assess disease severity) and if it limited their activities (to assess the associated burden). A total score was obtained by adding 1 point for each affirmative answer.

The Short Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) is one of the most widely used instruments for measuring pain intensity. It was developed based on the original MPQ and consists of a list of 15 most commonly used descriptors that assess the sensory and affective properties of pain rated on an 4-point intensity scale from 'none' to 'severe', a Present Pain Intensity (PPI) index with 5 descriptors representing increasing levels of pain intensity from mild to excruciating and a Visual Analog Scale (VAS) with 'no pain' and 'worst possible pain' as anchors. In addition to the PPI and VAS scores, SF-MPQ obtains 2 scores of sensory and affective pain intensity by adding up the intensities of the pain descriptors in each subclass and a total score by adding the sensory and affective scores.

The Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004b) is an instrument for assessing acceptance in chronic pain sufferers. It consists of 20 statements to which the respondents are asked to rate the truth as it applies to them, with a 7-point Likert response format (from 'never true' to 'always true'). The recently revised version of CPAQ identifies two facets of acceptance for which it computes two separate scores: pain willingness (recognition of the ineffectiveness of efforts to control pain), and activities engagement (the pursuit of activities even if pain is present).

The Sickness Impact Profile Roland Scale (RM-SIP; Roland and Morris, 1983; Jensen et al., 1992; Stroud et al., 2004) is a 24-item measure of self-rated disability

first developed for use in low-back pain, based on items from the Sickness Impact Profile (SIP; a measure of dysfunction in chronic pain). It has been subsequently shown to be useful in the assessment of patients with pain in other sites, as presence or absence of low back pain did not influence the stability of the scale relative to SIP (Jensen et al., 1992). The items refer to various difficulties experienced in daily activities due to pain. The respondents were asked to assess whether the items describe their condition over the past few days via a dichotomous response format. The total score sums up the affirmative answers.

The Brief Illness Perception Questionnaire (BIPQ; Broadbent et al., 2006b) is a nine-item instrument for assessing cognitive and emotional representations of illness (term replaced with chronic pain in this study). Eight questions with 11-point scale response format assess the consequences, timeline, personal control, treatment control, identity, concern, understanding, and emotional response related to chronic pain and an open-ended question addresses the respondent's beliefs regarding the 3 most important causes of the condition. Each item generates a score for the specific illness representation. The open-ended responses of the causality item can be grouped in categories relevant to the illness studied, and subject to categorical analysis. A supplementary question was included regarding whether the participant considered the causes of his/her condition are rather medical, psychological or social.

7.2.2 Recruitment protocol: first stage

The NHS Lothian Chronic Pain Service is located in 3 main centers in Edinburgh, UK: the Astley Ainslie Hospital (AAH), the Western General Hospital (WGH) and the Royal Infirmary of Edinburgh (RIE). Two recruitment strategies were applied here: face-to-face meeting at the time of appointment and postal questionnaire.

For the face-to-face meetings, chronic pain sufferers that attended appointments within this service were sent a letter of invitation from their consultant physician, a Participant Information Sheet (PIS) and a consent form (Appendix A), on average one week prior to their appointment at the clinic. Permission was obtained from the consultant physicians to access patient files and send invitations. The letters invited the potential participants to study the PIS and bring the PIS and consent form with them at the time of the appointment if they were interested in participating. The researcher was available in the waiting room when they arrived at the clinic and approached them to briefly ask if they had received the letter, if they were interested in participating and if they have any questions. If they expressed interest,

the researcher asked them to sign the consent form and gave them the first stage questionnaire and a prepaid envelope. They were advised to fill in the questionnaire during the following week and send it back by post in the prepaid envelope. No reminders were sent to participants at this stage.

For chronic pain sufferers that were not attending the Chronic Pain Service at the moment of the data collection and for those whose appointments were at times when the researcher was not available in the waiting room, only one letter was sent that included a modified letter of invitation, a modified PIS, two consent forms, already signed by the researcher, the first stage questionnaire, and a prepaid return envelope. The changes in the letter of invitation and patient information sheet consisted of adapting the content to the mailing procedure. If the patients were interested, they were advised to fill in the questionnaire, sign a consent form and send them to the researcher in the prepaid return envelope provided.

For chronic pain sufferers that preferred to respond via online completion of the questionnaire, an online version was provided which included the patient information sheet, the consent form and the first questionnaire. The differences between the online and pen-and-paper versions of the questionnaire were mainly due to the format options available in the online questionnaire development tool (www.survey.bris.ac.uk). For example, the visual analog scale for assessing pain intensity (SF-MPQ) was transformed into a 1 to 10 Likert format, while the open questions regarding the three main pain locations were transformed into a questionnaire format (Appendix A) based on Parsons et al. (2006). Other minor changes were included in order to increase response rate, such as inserting intermediary screens for encouraging completion and splitting questionnaires in two sections for easing access to response options.

Three chronic pain support organisations (Pain Support, BackCare, Pain Association) were invited and accepted to participate in the study. The online version of the study was advertised on the pain Support website (www.painsupport.co.uk) and via a brief article in the BackCare magazine, TalkBack (www.backcare.org.uk). Also, the researcher's contact details were provided in case a participant would prefer to ask for a pen-and-paper copy of the study participation pack. Several participation packs were prepared for Pain Association and the staff who organise local pain support group meetings throughout Scotland offered to invite group members to respond.

7.2.3 Recruitment protocol: second and third stages

The first questionnaire included a question on whether the participant agreed to be contacted in several months for the second stage of the study. Participants who agreed to be contacted in the future were asked to fill in their contact details and details of their GP. The GP details were used to inform them regarding the patients' participation in the longitudinal study. After around 3-4 months from the date of questionnaire reception, the researcher contacted the majority of the participants by phone to update their details accordingly. If they were still interested in participating, they were given the second questionnaire to fill in and return by post within a week. If no letter was received within 2 weeks, the researcher either contacted the participant by phone to check reception of the first letter, or sent a reminder together with another copy of the questionnaire and a prepaid return envelope. If no letter was received at this time, the researcher assumed that they were not interested in further participating in the study and did not contact them again. After another interval of around 3-4 months, the same procedure as in stage 2 was repeated, as the stage 2 and stage 3 questionnaires were identical[4].

The participants that answered the online version of the questionnaires, if they were interested in participating to the next 2 stages of the research, were requested to contact the researcher by e-mail and their e-mail addresses were used to send them the links to the second and third questionnaires after the above mentioned intervals. Reminders were sent by e-mail in this case. The information gathered online was anonymous. No contact details were requested in the online questionnaire. Participants' answers for all three stages were matched via their answers to questions about date of birth, height and colour of eyes.

The management of contact data and tasks was performed using a personal information manager (www.essentialpim.com). Dates of questionnaire reception were recorded for each participant and used to compute approximate dates for sending the next questionnaire, to verify reception and to send reminders. Other relevant comments related to each participant were recorded if necessary, such as change of address, requests, availability for phone contact.

After the data were analysed, the researcher sent a brief report of the general results of the study by post to all participants (Appendix A). The stages of the

[4]The only exception was skipping the initial phone contact, as many participants expressed their opinion that a phone conversation was not necessary, given that they once agreed with the whole procedure.

Stage	Time period	The researcher's responsibilities	The participant's responsibilities
1	January -March 2007	*a*) give the participant a patient information sheet and 2 consent forms; *b*) answer all participant's questions about the study; *c*) give the participant a questionnaire and a prepaid return envelope	*a*) answer the questionnaire as soon as they have the necessary time, best within a week; *b*) send back the completed questionnaire and the consent form by post in the prepaid return envelope
2	May - July 2007	*a*) contact the participant by phone to update their interest in the study and their contact details; *b*) send out a questionnaire and a prepaid return envelope; *c*) send out a reminder if no letter is received within 2 weeks	*a*) contact the researcher in case their interest in the study or their contact details change; *b*) answer the questionnaire as soon as they have the necessary time, best within a week; *c*) send back the completed questionnaire by post in the prepaid return envelope
3	September -November 2007	similar to stage 2	similar to stage 2
end of study	June - July 2008	send out a brief report of the results	

Table 7.2: Stages of data collection

data collection process are summarised in Table 7.2 (time intervals are the ones initially estimated, actual completion times were slightly delayed).

7.2.4 The recruitment process in figures

A total of 687 letters were sent to NHS users in an interval of 7 ½ months. The response rate was 28%; 193 users have sent the first stage questionnaire back (116 from WGH, 19 from RIE, 56 from AAH, 2 unknown; the difference between sites reflects the usual patient and staff numbers in the pain clinic sites). Several reasons can be hypothesised for this low response rate. First, both the postal method and the face-to-face method have drawbacks. The postal method can be considered unwanted mail, while approaching patients during waiting times can be perceived as an interference with their preparation for the medical consultation. Therefore both methods cannot be expected to lead to high response rates, although they were practically the most convenient for both participants and medical staff. Second, the length of the questionnaire was substantial especially for the first stage. Third, chronic pain is a very demanding condition, and sufferers often find very few resources to deal with the daily tasks; answering a questionnaire would not be among their top priorities and they might not have enough energy to do it. Fourth, the relationship between the chronic pain sufferer and the medical establishment is

rarely smooth despite efforts from both sides, as detailed in Chapter 2. The survey could have been perceived by many as yet another set of questions that offer them very little in return, and many patients approached in the waiting room expressed these concerns. Fifth, several Royal Mail strikes happened during data collection and some questionnaires might have been lost; as there was no possibility of sending reminders for the first stage, this situation could not be prevented[5].

Response rate did not vary between recruitment sites (29% WGH, 29% RIE, 25% AAH; $\chi^2(2) = 1.8$, $p = .4$). The slightly lower rate at AAH could be due to the fact that not only the people that had upcoming appointments have been contacted, but all people under treatment or investigations, for which the relevance of the letter might have been reduced. Response rate was slightly higher when participants have been approached face-to-face (31%, including missed appointments) compared to postal questionnaires (26%), although the difference is not significant ($\chi^2(1) = 1.33$, $p = .2$). Considering the substantial effort involved in meeting participants during the waiting times and the possible disruption to the consultation process, these data suggest that sending questionnaires by post is perhaps more suitable in this context.

In addition to the participants recruited via NHS, the advertisements via support organisations led to 29 responses by post and 47 responses via the online survey. Of these respondents, 30 have said they found out about the study from Pain Association Scotland, 27 from PainSupport, 5 from BackCare and 14 from other sources (friends, internet, their chiropractor). The response rate cannot be calculated for this recruitment method, as the sample was self-selected. The use of this method also recommends cautious generalisation.

Out of the initial 269 participants, 228 responded to the second stage (15% attrition rate), and 213 responded to all three stages (21% total attrition rate, 7% between the last two stages). Among the non-respondents, only two have expressed their wish not to be contacted in the next stages on the first stage questionnaire. There was no difference in attrition rate between the face-to-face method and the postal method in the NHS in both stage two and three ($\chi^2(1) = .42$, $p = .5$ and $\chi^2(1) = .92$, $p = .3$), further supporting the use of the postal method as most appropriate given the drawbacks of the face-to-face method. Attrition rates were comparable with other studies. For example, 26% respondents completed less than 3 of the total of 4 assessments in Mossey and Gallagher's (2004) 2-year longitudinal study of US

[5]In the second and third stages, contacting participants whose questionnaires did not arrive in the two-week interval planned has helped track down these situations and reduce attrition.

retirement community residents (although the respondents were older and the main cause of attrition was death, 11%). Covic et al. (2003) reported an attrition rate of 15% with a similar design (3 time points within a 12-month interval) on a sample of rheumatoid arthritis patients of similar age distribution, recruited from Australian rheumatology practices.

Recruitment lasted in total approximately 1½ years (from the date when the first letter was sent, 21st March 2007, to the date when the last questionnaire was posted, 8th September 2008). The duration of each stage overlapped substantially with the others (11 ½ months the first stage, 12 months the second stage, 9 months the third stage). This length was mainly due to a few late responses, as most participants responded within a short period of time. For example, 75% of the participants recruited from the NHS responded within 2 weeks in the first stage of data collection, 90% within 4 weeks and 99% within 4 ½ weeks, while the maximum response time was 7 months. The delays were due to various reasons: forgetting, holidays, lost letters in the post or delays in post services, family or personal priorities, not necessarily related to the health condition. The actual time interval between assessments varied depending on the logistics of postal contact, the mean time being approximately 4 ½ months.

The time it took to complete the first stage survey (the longer of the three) was approximately 45 minutes. Several participants have stated that they have split the task in two or three consecutive days or made a few pauses between sections of the questionnaire. Pauses were also recommended explicitly in the online version, as sitting for long periods in front of the computer may increase the probability of flare-ups in some groups of patients. The majority of the participants (84%) stated they received no help to fill the form (according to responses to the first stage questionnaire from NHS participants).

7.3 Data analysis

The data analysis was performed using SPSS 14.0, EQS 6.1 and R(lmer package). Preliminary data screening operations were performed in SPSS according to Tabachnick and Fidell (2001, Ch. 4) in order to check for accuracy of data input (identifying and correcting any out-of-range values), diagnosing missing values patterns and fit with assumptions of multivariate analysis. Justifications for each of the data preparation decisions are detailed where necessary in the relevant section.

Descriptive statistics as well as correlation, regression and cluster analyses necessary for testing the initial hypotheses were performed in SPSS. All analyses included checks of normality, linearity, homoscedasticity, independence of residuals, multivariate outliers, multicollinearity and singularity as advised by Tabachnick and Fidell (2001). These properties were considered adequate in all cases unless otherwise stated[6]. In all tables presented, * represents significance at $\alpha = .05$ and ** significance at $\alpha = .01$ (2-tailed). The multiple regression analyses followed guidelines described by Tabachnick and Fidell (2001, p. 163). Power analyses were performed using GPower3 (Faul et al., 2007). The cluster analyses were executed according to guidelines by Everitt et al. (2001) and Clatworthy et al. (2005). The confirmatory factor analyses (CFAs) necessary for questionnaire analysis and the structural equation models (SEMs) in the analyses of interrelations were performed in EQS 6.1. As model testing and reporting is a controversial topic in the SEM literature, the strategy applied is discussed in detail in Appendix B. The data preparation and missing data analyses for all variables and questionnaires are presented in Appendix C.

7.4 Sample characteristics

The demographic characteristics for the present sample are presented in Tables 7.3 and 7.4 with the descriptive statistics applicable. They describe a group of mostly white, British adults, the majority of whom were female, with medium or high education, more than half unable to work because of pain or other reasons (housewife, retired), most of them married or living as married, reporting an average annual income. Their chronic pain condition had usually started in middle age, following an accident, surgery or with a gradual aggravation of the condition. However the sample characteristics were rather varied: several of the other categories were well represented, while the age and pain onset ranges were substantial.

As most of the participants were recruited from a pain clinic, they reported following multiple treatments in the past. Pain relief medication was the most reported treatment, followed by physiotherapy, pain management and antidepressant medication. The percentages of the treatments are presented comparatively in Figure 7.1. Notably, the use of complementary therapies (acupuncture, homeopathy, herbalism) was reported by more than half of the participants. Surgery, psychological therapies

[6]When multivariate outliers were identified in the analyses, these were usually eliminated. However no significant differences in parameters were identified between analyses with and without outliers.

Variable name	Values	Counts	Percentages
Gender	male	78	29.4
	female	186	70.2
	missing	1	0.4
Education	none	6	2.3
	secondary school	116	43.8
	college/university	143	54
	missing	0	0
Vocational status	ft working	52	19.6
	pt working	32	12.1
	ft training	11	4.2
	unable to work - pain	144	43.0
	unable to work - other reason	52	19.6
	missing	4	1.5
Marital status	single	55	20.8
	married	145	54.7
	living as married	20	7.5
	separated	7	2.6
	divorced	24	9.1
	widowed	13	4.9
	missing	1	0.4
Annual income	below average	89	33.6
	average	126	47.5
	above average	37	14.0
	missing	13	4.9
Ethnicity	white	259	97.7
	other	5	1.9
	missing	1	0.4
Nationality	British	256	95
	other	7	3
	missing	5	2

Table 7.3: Descriptive statistics - demographics

Variable	Mean (yrs)	SD (yrs)	Min (yrs)	Max (yrs)
age	50	12	18	80
time from pain onset	10 1/4	8 1/2	1/4	54
age at pain onset	39	12	2	73 1/2

	Values	Counts	Percentages
pain started	gradually	64	24.2
	suddenly	9	3.4
	suddenly and gradually worse	53	20.0
	as a result of an accident	60	22.6
	as a result of an illness	26	9.8
	following surgery	40	15.1
	Other (e.g. post pregnancy)	7	2.6
	missing	6	2.3

Table 7.4: Descriptive statistics - pain related

Current treatment	stage 1	stage 2	stage 3
Pain relief medication	89	89	90
Antidepressants	38	33	33
Sleeping tablets	15	18	19
Surgery	6	8	9
Physiotherapy	21	20	18
Psychological therapies	13	12	10
Pain management programmes	34	37	29
Complementary therapies	26	24	29
Other	25	22	18

Table 7.5: Current treatment percentages for the three stages

and sleep medication were relatively rarely used compared to medication and pain management. Some participants reported the use of several other treatments (chiropractice, osteopathy, massage, TENS, music therapy, hypnotherapy, nutritional therapy, reiki, relaxation, rolfing - deep tissue massage, tai-chi, self-help) which reflects not only the diversity of treatments available on the complementary medicine market but also issues related to the complexity of chronic pain. It is remarkable that despite the richness of treatments little improvement was reported in the 1 year interval of the study, as reported later in this chapter. During the course of the study, participants reported they followed a relatively stable treatment schedule, as shown in Table 7.5. Very few participants reported they did not follow any treatment (3.4–5.2%), they usually reported following 2 treatment types simultaneously, the majority reported between 1 and 4 treatments (86–92%).

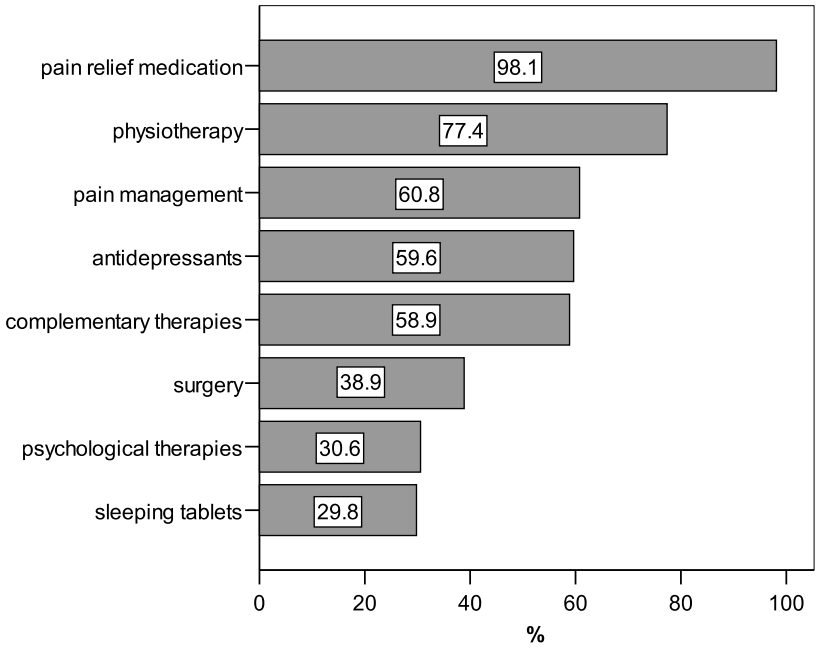


Figure 7.1: Previous treatments

Some of the participants from the chronic pain support organisations also responded to questions regarding medical diagnosis, benefits, litigation and perceived social support. Due to the low number of respondents that have answered this type of questionnaire (introduced later in the data collection stage), this information was only used descriptively. Out of 50 people that responded to these questions, only 33 (66%) reported they had received a diagnosis for their condition. They expressed their diagnosis either in medical technical terms (such as fibromyalgia, ankylosing spondylitis, osteopaenia, osteoarthritis, CRPS, MS, ME), lay terms (for example nerve damage, out of line hips, disc bulge, trapped nerves), or even events considered causally related to the pain ("fell down stairs", "complications after spinal surgery"). The majority of patients that reported receiving a diagnosis felt they understand their diagnosis rather well (76–78% scored above 5 on a 0–10 scale on all three stages). Approximately half (54%) of these participants stated they currently received financial benefits related to their chronic pain (disability living allowance, incapacity benefit, industrial injury benefit). None of the participants reported being involved in litigation. The participants reported feeling supported mostly by family, friends and support groups in coping with their health condition, while colleagues and medical professionals were perceived as less supporting. A few participants reported other sources of support: carers, church, complementary medicine practitioners.

The majority of the participants considered that the causes of their chronic pain were rather medical (between 95 and 97%, according to responses from the three stages), while very few reported their belief that the causes were psychological (7.7-11%) or social (6.1-8.2%). These responses reflect the rather delicate issue of causation in chronic pain (and the social aspects of the patient-doctor relationship).

Only 18% of respondents reported no other associated conditions, according to the Self-Administered Comorbidity Questionnaire (see Figure 8.4). The rest of the respondents reported one or more conditions, among which the most prevalent was depression (43%), followed by osteoarthritis (35%), high blood pressure (23%) and ulcer/ stomach disease (20%). Other health problems (such as hearth disease, diabetes, lung disease etc.) had a much lower prevalence (less than 10%). Some respondents reported other medical conditions such as asthma, irritable bowel syndrome or various other immune, circulatory, urinary or dermatological conditions of low prevalence in this sample. The high prevalence of depression in this chronic pain sample is in agreement with other similar reports in the literature, as reviewed in Chapter 4.

The most frequent pain location reported was lower back pain (see Figure 7.2), but most participants reported several pain locations: only 25% reported pain in one broad area (head and neck, limbs, back or visceral), 39% in two areas, 19% in three and 16% generalised pain. Only 17% reported pain in a single location.

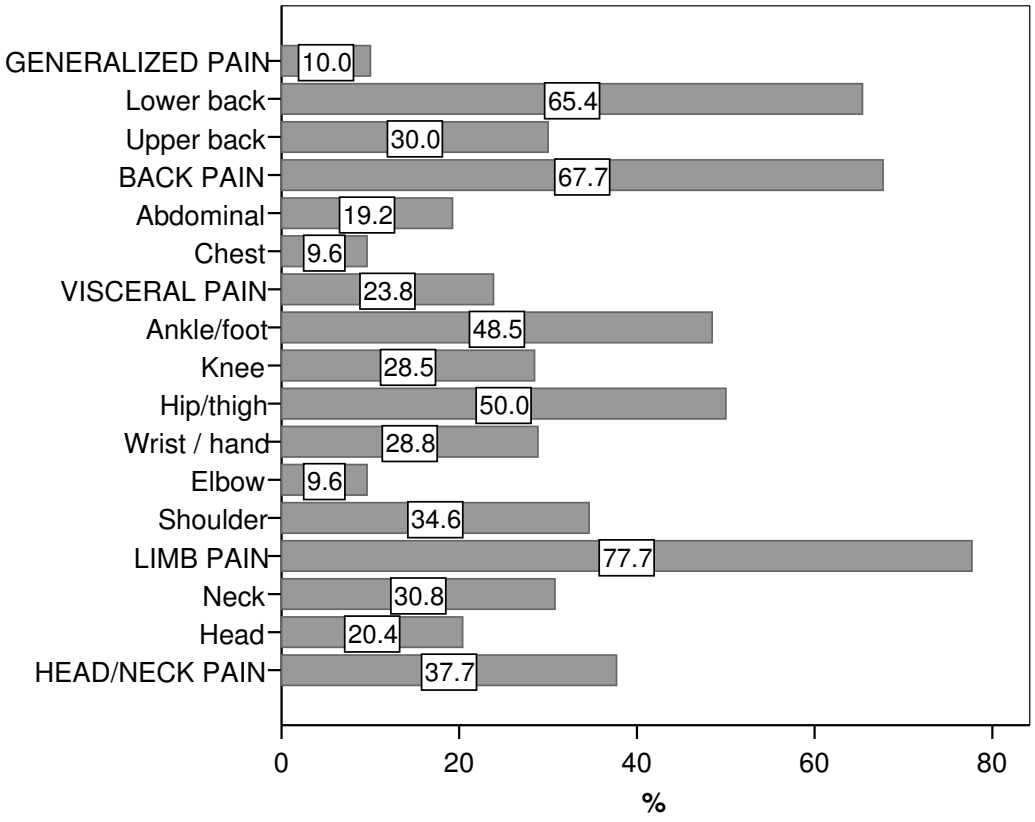


Figure 7.2: Pain location percentages (for specific and broader areas)

7.5 Questionnaires analysis

A detailed analysis of the psychometric properties of the questionnaires used in the present study can be found in Chapter 8, together with a rationale for the necessity of this analysis. The missing data analysis is reported in Appendix C. The descriptive statistics and psychometric decisions are summarised below (data presented in Table 7.6).

AAQ was characterised by good distributions for items and total scores, acceptable internal consistency and stability. A one-factor CFA with method bias specifications reached χ^2 non-significance only in one stage, indicating that although AAQ is a

	Possible range	Mean (SD)	Cronbach's α	Stability
AAQ	1-7	3.8-3.9 (.9)	.76-.78	.76-.72
BES - Anger	1-7	4.2-4.4 (1.2-1.3)	.79-.82	.73-.80
BES - Sadness	1-7	3.4-3.6 (1.5)	.87-.89	.76-.80
BES - Shame	1-7	2.8-2.9 (1.4-1.5)	.88-.91	.75-.78
BES - Anxiety	1-7	4.6 (1.2-1.3)	.85-.89	.71-.75
BES - Happiness	1-7	4.7-4.9 (1.2-1.3)	.90-.92	.76-.81
REQ - ID	1-5	2.3 (.7)	.75-.77	.74-.76
REQ - IF	1-5	2.9-3.0 (.6)	.70-.72	.65-.71
REQ - ED	1-5	1.5 (.5)	.73-.77	.72-.77
REQ - ED	1-5	2.6-2.7 (.7)	.75-.78	.76-.84
LTE - negative*	0-12(+)	1 (1.5-1.6)	-	-
LTE - positive*	0-5(+)	0-1 (.9)	-	-
SACQ*	0-45(+)	4 (3.96)	-	-
MPQ - S-PRI	0-33	14.0-14.5 (7.5-7.6)	.81-.85	.56-.63
MPQ - A-PRI	0-12	4.5-4.8 (3.3-3.5)	.78-.80	.45-.66
MPQ - VAS	0-144	93.0-96.0 (31.0-33.3)	-	.54-.71
MPQ - PPI	1-5	3.0-3.2 (1.1-1.2)	-	.49-.64
MPQ - T-PRI	0-45	19.0-19.4 (10.2-10.3)	.87-.89	.56-.71
CPAQ - PW	1-7	2.3-2.6 (1.1)	.83-.85	.71-.81
CPAQ - AE	1-7	2.9-3.1 (1.1-1.2)	.88-.90	.75-.82
CPAQ - total	1-7	2.6-2.9 (.9-1.0)	.90-.91	.79-.87
SIP	0-24	12-13 (6)	.89-.90	.83-.86
BIPQ - consequences	0-10	7.0-7.6 (2.0-2.2)	-	.61-.63
BIPQ - timeline	0-10	8.9-9.0 (1.6-1.8)	-	.49-.56
BIPQ - personal control	0-10	4.4-5.1 (2.5-2.6)	-	.29-.35
BIPQ - treatment control	0-10	5.2-5.5 (2.6-2.7)	-	.22-.38
BIPQ - identity	0-10	7.4-7.6 (2.0)	-	.43-.47
BIPQ - concern	0-10	6.8-7.5 (2.4-2.6)	-	.52-.57
BIPQ - understanding	0-10	7.0-7.4 (2.4-2.6)	-	.43-.48
BIPQ - emotional response	0-10	6.8-7.2 (2.4-2.5)	-	.52-.57
BIPQ - causes	-	-	-	-
HCU - visits GP	-	-	-	.37-.46
HCU - other visits	-	-	-	.38-.44
HCU - medication	-	-	-	.55-.60
HCU - self-help	-	-	-	.50-.51

Table 7.6: Descriptive and psychometric data for the questionnaires (*=median scores reported; +="other" options may increase the maximum total score)

good approximation of levels of psychological flexibility, further improvements are necessary[7].

BES showed good item and subscale scores distributions, with the exception of the shame/disgust subscale (and part of its items) which was underreported. The subscales also showed good internal consistency and stability. CFA and EFA analyses revealed a more complex structure of the questionnaire responses than a correlated 5-factor model, however this model can be considered an acceptable and parsimonious approximation of emotional life reports as measured by BES.

REQ proved to be less adequate in a chronic pain context (and adult sample). Several emotion regulation strategies were notably underreported, and the 4-factor CFA model had a poor fit. EFA analyses revealed several other items that were related to other subscales in addition to their own. Only a limited number of items showed good properties. However, the internal consistency and stability of the subscales were above the minimal reliability standards (.70; Nunnally and Bernstein, 1994, p. 265)[8]. Therefore, both subscale scores and selected best items were used in further analyses involving REQ.

LTE reports indicated that illness, injury or assault to the person or close relatives, death of a close friend or relative, serious problems with a close friend and financial problems were the most frequent negative events, while improved financial status and birth of a child were the most frequent positive events. Since the questions enquire about distinct and time limited situations, LTE does not require test-retest reliability and internal consistency. Both the total number of events and the occurrence of most frequent events were subsequently analysed.

The responses to SACQ indicated that depression and osteoarthritis were the most frequent comorbid conditions (42.6% and 34.7% respectively). The distribution of total scores was expectedly skewed and the variable was dichotomised for further analyses. The presence of depression and osteoarthritis was analysed separately.

The analysis of SF-MPQ focused on two main issues. First, there was a substantial proportion of missing data (betwen 3.0–9.5% for PPI and 16.6–31.7% for 'splitting') and therefore subject to a detailed analysis and computed based of two patterns identified (by EM and replacement with 0). Second, a 2-factor CFA model for the pain descriptors showed lack of fit and high correlation estimates for the two

[7]A second improved version is currently available (Bond et al., submitted).

[8]With only one exception concerning the stability of the internal functional subscale in one of the stages.

subscales. However, given the possibility that the correlations between S-PRI and A-PRI might depend on other patient caracteristics, they were kept for further analyses together with the T-PRI, VAS and PPI scores. The subscales showed however good internal consistency, and moderate stability (which is to be expected for momentary pain ratings, despite the lack of overall change in pain severity characteristic of the chronic pain population).

The CPAQ items and subscales had good distributions in general (excepting only 2 items), and good internal consistency and stability. However the analysis also revealed that the two subscales can be due to a method artifact (the PW subscale contains only reverse scored items, while AE is formed only by normally scored items). Therefore both subscale and total scores were analysed further, with particular interest on the differentiation between the two subscales.

The RM-SIP proved to cover a wide range of disability levels, with normal distribution of total scores, good internal consistency and stability. As no other requirements for validity are stipulated by theory, the measure was considered adequate, although limited testing of the questionnaire structure suggested lack of unidimensionality and selected items were also used in further analyses.

Most BIPQ items were negatively skewed, suggesting a lower ability to discriminate between chronic pain respondents. Due to the heterogeneous pattern of correlations among subscales, total scores could not be computed[9]. Therefore, the subscale scores were used separately. The items also showed lower stability, to a certain degree unsurprising for single item scales, but also lower than initial 3-6 weeks estimates, which indicates either real fluctuations of illness perceptions at such intervals, or the lower test-retest reliability of BIPQ. Causality responses focused on attributions related to medical treatment (39.4%), the respondent's own behaviour (35.7%) and a specific external event (34.4%).

The distributions of health care utilisation responses were analysed to produce meaningful total scores: number of GP visits, number of different specialist treatments used, number of different types of medication used, and number of different self-help methods used. These scores showed medium stability, which is to be expected to a certain degree given treatment patterns, but also probably due to the lower reliability of the measures. As medication categories also had lower face validity (technical, unclear and incomplete), results of analyses based on these scores need to be treated with caution and replicated with more adequate measures.

[9]The authors of BIPQ recommended the use of single-item subscales.

As discussed in Chapter 6, a more powerful test of the hypotheses is using a full SEM of the stated relationships between concepts specified as latent variables measured indirectly by the specific items. As few questionnaires have passed such a test themselves, part of the hypotheses were tested by using both total scores and selected items.

The next steps in data analysis focus on testing the expected relationships between the constructs measured following the sequence presented in Section 6.5.

7.6 Confirmatory data analysis

7.6.1 Separate examination of the four areas

Chronic pain acceptance and psychological flexibility

A first question regarding CPA which can be examined in the present data set is whether the responses would be better characterised by a categorical approach (distinct patient groups) rather than a dimensional one (scores on continuous variables). Therefore, a first step in the examination of CPA in this sample is the replication and extension of the cluster analysis reported in Vowles et al. (2008b).

In replicating results of cluster analyses, it is recommended to apply the same statistical method. In a review of the use of cluster analysis in health psychology, Clatworthy et al. (2005) underlined the usefulness of this method to identify relatively homogeneous groups regarding the characteristics relevant for a specific service, as a first step towards tailoring interventions. They also noted its misuse in the literature and the low quality of reporting. As cluster analysis includes numerous methods and no clear guidelines, in order to facilitate replication they recommended reporting minimum five types of information: the computer program, the similarity measure, the cluster method, the procedures used to determine the number of groups in the data and the validation procedures (Aldenderfer and Blashfield, 1984, as cited by Clatworthy et al., 2005). Although the procedure is not described in detail in the original study[10], the present analysis attempted to follow similar steps, performed separately on the 20 items and the 2 subscale scores. Hierarchical analyses (agglomerative scheduling, between groups, squared Euclidean distance) were

[10]The authors stated they performed a series of hierarchical and k-means cluster analyses using the two subscales. Even if the clustering method is also used for unidimensional or bidimensional data, a simpler method for revealing the presence of clusters in a bidimensional space (AE and PW scores) would be the inspection of a scatterplot rather than any classification algorithm. Thus, it cannot be ascertained whether the subscale items or scores were used.

No. of	20 items analysis			2 subscales analysis		
clusters	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
15	106.636	107.879	96.371	1.045	1.109	.895
14	108.000	108.936	99.000	1.138	1.416	1.034
13	110.347	111.417	103.935	1.520	1.466	1.229
12	115.927	113.518	104.152	1.663	1.629	1.456
11	117.000	115.000	109.000	1.731	1.685	1.536
10	117.580	120.406	110.241	2.062	1.707	1.708
9	122.865	129.000	114.990	2.221	1.878	1.726
8	123.000	131.038	118.809	2.398	2.015	1.903
7	128.723	134.433	121.909	2.883	2.542	1.985
6	134.902	137.321	123.778	3.179	2.596	3.042
5	149.500	142.333	133.700	3.253	2.801	3.465
4	167.934	147.523	136.317	5.342	5.411	3.583
3	182.119	175.676	146.853	7.563	6.713	6.517
2	188.999	181.922	148.417	8.076	8.341	7.926
1	199.856	227.844	178.424	8.289	8.838	14.364

Table 7.7: Distance coefficients for the last 10 steps of the agglomeration schedules - cluster analysis of CPAQ

first performed to search for a suitable solution based on distance coefficients. K-means analyses were performed to generate 3-cluster solutions. Cluster differences relative to the CPA subscales were explored to identify the characteristics of the groups. Differences in demographics, medical history and patient functioning measures were next explored and compared to the original study. In addition to Vowles et al. (2008b), the stability of clusters between the 3 stages and the distinctiveness of the clusters were also examined.

The examination of the cluster solutions of the last 15 steps of the agglomeration schedules (Tables 7.7 and 7.8) did not indicate a clear 3 cluster solution (as it can be seen, most solutions selected based on the largest increases in distance coefficients include either 1 or 2 main groups, and only 2 of the 18 solutions include 3 groups). Exploring previous steps of the agglomeration schedules revealed no other notable gaps. These results raised doubts regarding the suitability of a 3 group classification in the present sample.

The k-means cluster analyses generated groups with comparable numbers of cases (Table 7.9). The classification of cases based on items and subscale scores was significantly related ($\chi^2(4) = 264$ to 378, $p < .001$), which is to be expected as the subscale scores are sums of items[11]. The cluster means on PW and AE were similar to the solution reported in Vowles et al. (2008b): a group with high scores on

[11]As the three clusters have increasing AE and PW mean values, the similarity between the equivalent item and subscale-based solutions can be expressed in terms of correlations: $r = .77$, .85 and .96 for the three stages.

		Stage					
		1st		2nd		3rd	
Analysis	Solution	No.	% cases	No.	% cases	No.	% cases
20 items	1	5	97%	2	99.6%	2	92%
	2	6 or 4	97%	4	98%	4	91%
	3	2	99.6%	10	73%, 23%	6	89%
2 subscales	1	4	77%, 20%	5	57%, 22%, 19%	2	98%
	2	5	57%, 22%, 19%	3	78%, 22%	4	61%, 28%
	3	3	78%, 22%	4	76%, 22%	3	69%, 30%

Table 7.8: Total number of clusters and percentages of cases classified within the most represented groups in the solutions - cluster analysis of CPAQ

	20 items			2 subscales		
Group	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
Low acceptance	80	56	66	69	65	61
'Discordant'	85	100	81	141	117	83
High acceptance	100	70	64	55	44	67
Missing	5	44	59	5	44	59

Table 7.9: Number of participants allocated to the different groups - 3-cluster solutions, CPAQ

		Analysis					
		20 items			2 subscales		
Score	Group	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
AE	L	1.6 (.6)	1.8 (.9)	1.9 (.9)	1.5 (.6)	1.9 (1.0)	1.8 (.9)
	D	3.3 (.6)	2.9 (.8)	3.2 (.8)	3.1 (.7)	3.0 (.8)	3.1 (.9)
	H	3.6 (.8)	3.8 (.8)	3.7 (.9)	4.2 (.7)	4.1 (.8)	3.7 (.9)
	total	2.9 (1.1)	2.9 (1.1)	2.9 (1.2)	2.9 (1.1)	2.9 (1.1)	2.9 (1.2)
PW	L	1.7 (.9)	1.4 (.8)	1.7 (1.0)	1.4 (.9)	1.4 (.8)	1.6 (.9)
	D	1.7 (.7)	2.3 (.8)	2.1 (.9)	2.2 (.7)	2.4 (.9)	2.1 (.9)
	H	3.3 (.6)	3.0 (1.1)	3.2 (.9)	3.7 (.6)	3.4 (1.0)	3.2 (.9)
	total	2.3 (1.1)	2.3 (1.1)	2.3 (1.1)	2.3 (1.0)	2.3 (1.1)	2.3 (1.1)

Table 7.10: Means (and standard deviations) for the three groups - cluster analysis of CPAQ

both PW and AE, a group with low scores on both subscales and a group situated in between the first two[12], with AE scores significantly higher than PW scores[13]. Means and standard deviations for the three groups are presented in Table 7.10.

[12]Post-hoc pairwise comparisons significant for all analyses with the exception of the stage 1 cluster analysis based on items, where AE scores are almost as high as the first group ($p = .03$) and PW scores are as low as the second group ($p = 1$).

[13]T-test values significant at $\alpha = .001$ for all 6 cluster analyses.

Variable	Test statistic
Gender	$\chi^2(2) = 12.1$, $p = .002$
Education	$\chi^2(2) = 7.0$, $p = .03$
Comorbidity	$\chi^2(2) = 8.0$, $p = .02$
Vocational status	$\chi^2(2) = 28.9$, $p < .001$
Depression	$\chi^2(2) = 29.5$, $p < .001$
GP visits	$\chi^2(2) = 26.2$, $p < .001$
Pain intensity (VAS)	$F(2, 120.9) = 27.6$, $p < .001$
Pain intensity (PPI)	$F(2, 262) = 19.3$, $p < .001$
Pain intensity (T-PRI)	$F(2, 129.4) = 26.7$, $p < .001$
Pain disability	$F(2, 138.7) = 5.0$, $p < .001$
Medication use	$F(2, 262) = 5.0$, $p = .007$

Table 7.11: Test statistics for group differences – 3-cluster solutions, CPAQ

The six analyses gave considerably similar results[14]. There were no differences between the three clusters regarding marital status, pain duration, pain location, age, age at pain onset, pain spread, time since pain onset, treatment seeking (number of different types of medical services attended) and self-help (number of different self-help methods used). There were significant differences in gender, education, presence and severity of comorbid conditions, vocational status, presence of depression, number of GP visits, pain intensity (VAS, PPI, T-PRI), pain related disability (RM-SIP), medication use (number of different medication categories reported). There were 80-85% women in the high acceptance group, 70-76% in the discordant group and 52-38% in the low acceptance group. People with college/university education were classified in the high acceptance group more frequently than in the discordant or low acceptance groups (80-85%, 80-85% and 80-85%, respectively). People reporting high comorbidity, being unable to work because of pain, depression and more GP visits were also classified more in the low acceptance group compared to the discordant or high acceptance groups (60-65%, 41-58% and 34-40%; 78-88%, 43-55% and 21-39%; 58-70%, 33-39% and 19-32%; 67-78%, 40-54% and 27-43%, respectively). The high acceptance group had significantly lower levels of pain intensity, pain related disability and medication use compared to the low acceptance group, while the mean scores of the discordant group were situated in between the two[15]. Test results for one of the cluster analyses (first stage, based on subscale scores) are presented in Table 7.11 (only one solution is presented, for brevity).

The results are similar to Vowles et al. (2008b). However it is important to assess to what degree they constitute the clinical characteristics of 3 distinct groups or

[14]The order of differences was the same in all analyses, only the magnitude and in a few cases the test significance given $\alpha = .05$ were different.

[15]All post-hoc comparisons between the discordant group and the other two groups were significant with the exception of medication use.

simply a different statistical expression of the relationship between CPAQ scores and the demographic and medically-relevant variables above. If the present classification is a more suitable description of the data than an acceptance continuum, the classification should be stable and form distinct groups. The importance of these two properties was highlighted by Clatworthy et al. (2005), who explained the necessity of showing the stability of clusters by replication (by splitting the sample, or in a different sample) and noted: "all cluster analyses will provide clusters, whether true groups exist in the data or not" (p. 354).

In terms of stability, the allocation of participants to groups between the 3 stages was not random ($\chi^2(4) = 102.4 - 138.2$, $p < .001$), which reflects the stability of CPAQ scores. However, about 30% of cases changed classification between the middle group and one of the other two groups between stages, indicating that the cut-offs selected by the clustering algorithm fluctuated considerably as a response to relatively small fluctuations in the individual scores[16] (percentages of participants that were attributed to different groups between stages are presented in Table 7.12). This might be indicative of a lack of distinctiveness between the three clusters, which was explored next.

The question of whether there is a structure in the data or not can be answered to a certain extent by exploratory methods which produce visual outputs showing the degree of separation of the clusters (Everitt et al., 2001). Identifying groups characterised by internal cohesion (homogeneity) and external isolation (separation) would be a strong argument for the objectivity of the results of clustering. If the data are homogeneous, an equally plausible conclusion might be not to group data (Everitt et al., 2001, p. 4). Grouping homogeneous data is referred to as 'dissection' (similar to attributing postcodes to addresses in an area), and has its practical uses, although it is important not to treat it as reflecting the existence of distinct groups.

For multivariate data, three methods of visualising clusters are more frequently used: scatterplot matrices, principal components analyses (PCA) and multidimensional scaling (MDS). The presence of cluster patterns in the scatterplot matrices would be indicative of the existence of distinct groups at the bivariate level. Indeed, the scatterplots of the two subscale scores did not indicate the presence of

[16]Changes in the classification of individual cases were expected to a limited extent, as some patients might have changed their attitude towards pain as a result of interventions, changes in their medical condition or other events. However given the general lack of change in the sample (reported later in this section) such fluctuations cannot account for the overall instability of the clusters.

	Analysis					
	20 items			2 subscales		
Group	1to2	2to3	1to3	1to2	2to3	1to3
Stable LA	19.4	19.1	22.3	19.4	20.6	18.4
Stable D	16.7	25.4	19.9	36.5	29.2	28.2
Stable HA	22.5	23.0	23.8	14.0	17.7	19.4
LA versus D	15.3	15.3	9.7	15.8	14.8	16.5
D versus HA	24.8	16.3	18.0	14.0	17.2	16.5
LA versus HA	1.4	1.0	6.3	.5	.5	1.0

Table 7.12: Percentages of participants allocated to the same or different groups across stages - 3-cluster solution, CPAQ

groups, but rather two continua on which the clustering algorithm has applied relative cut-offs (example in Figure 7.3). As lack of clear bivariate patterns does not automatically imply lack of multivariate structure in the data (Everitt et al., 2001), PCA was also used to explore a possible structure. A similar result was apparent in the scatterplots of the first two components of a principal components analysis of CPAQ items (example presented in Figure 7.4) and in using MDS (not reported). The two graphs indicate that, while some participants remain in their groups in all three stages, a substantial proportion of cases change categorisation. They also show that the three groups were formed by 'dissecting' a relatively homogeneous sample characterised by the two correlated subscale scores (or by the two principal components.

Given the instability of classification for a considerable proportion of cases (contrasting with the stability of individual acceptance scores), the utility of this categorisation in clinical practice is limited, as discussed in Chapter 9. The use of continuous acceptance scores was therefore considered a more suitable approach and was applied in further analyses.

Another important question for the ACT model itself is whether CPA and PF are the same or different concepts. Results of correlational analyses indicated that CPA was closely related to, but distinct from PF (r = .57, .62, and .64, for stage 1, 2 and 3, respectively) thus indicating they were potentially distinct constructs. The two measures showed a degree of variation across time (test-retest r = .76, .75, and .72 for PF, and .81, .87, and .79 for CPA)[17], therefore their interrelations between the three stages were explored next.

[17]Although results of one-way ANOVA tests indicated a significant decrease in AAQ between stage 2 and 3 ($F(2, 408) = 4.44$, $p = .01$), and a significant increase in CPAQ between stage 1 and 2, maintained in stage 3 ($F(1.87, 381.2) = 14.06$, $p > .001$), the changes were minimal (mean scores range from 3.81 to 3.95 for AAQ from a 1-7 range and 2.66 to 2.87 for CPAQ from a 0–6 range).

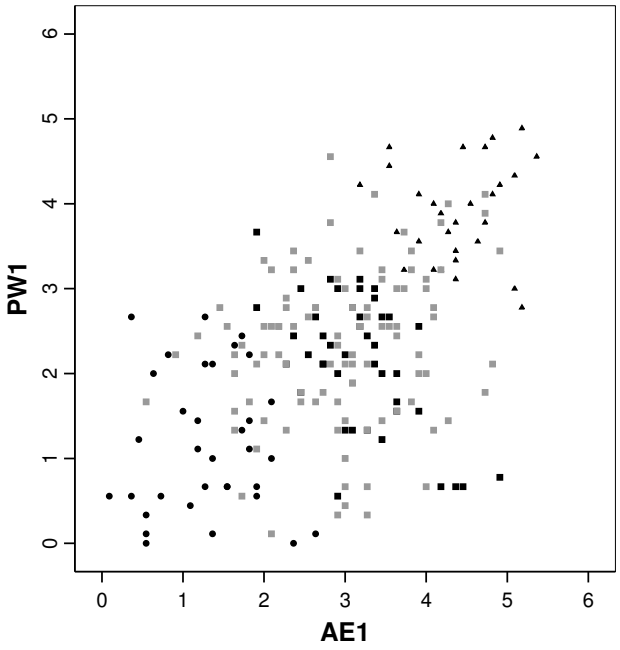


Figure 7.3: Scatterplot with PW and AE scores for CPAQ cluster solution (stage 1), cases with stable and unstable categorisation: ● - stable LA; ■ - stable D; ▲ - stable HA; ■ change between LA, D and HA across stages.

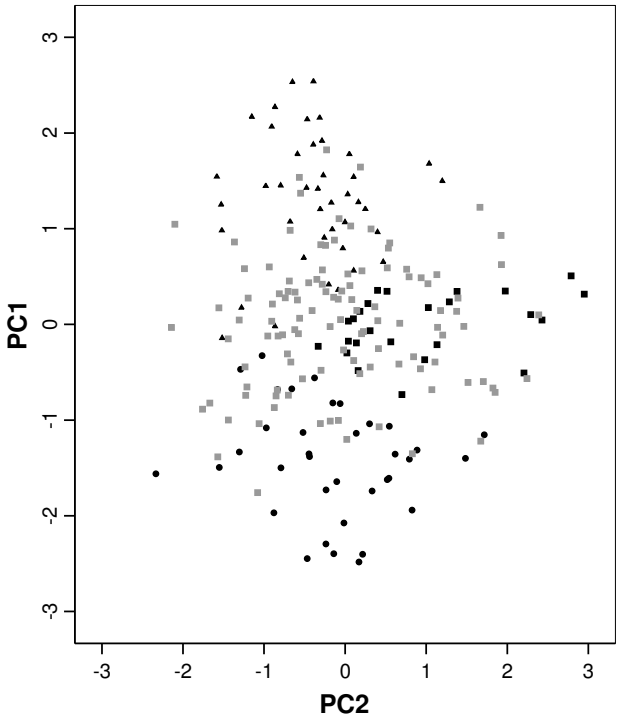


Figure 7.4: PCA scatterplot for CPAQ cluster solution (stage 1), cases with stable and unstable categorisation: ● - stable LA; ■ - stable D; ▲ - stable HA; ■ change between LA, D and HA across stages.

Statistic	Model 1	Model 2	Model 3
Yuan-Bentler	4.7(2)	20.5(4)	10.6(4)
χ^2(df)	p=.09	p<.001	p=.03
NFI	.99	.97	.99
NNFI	.97	.92	.97
CFI	.997	.98	.99
RMSEA	.07(.00-.16)	.12(.07 - .18)	.08(.02-.13)

Table 7.13: Goodness of fit statistics for longitudinal path models

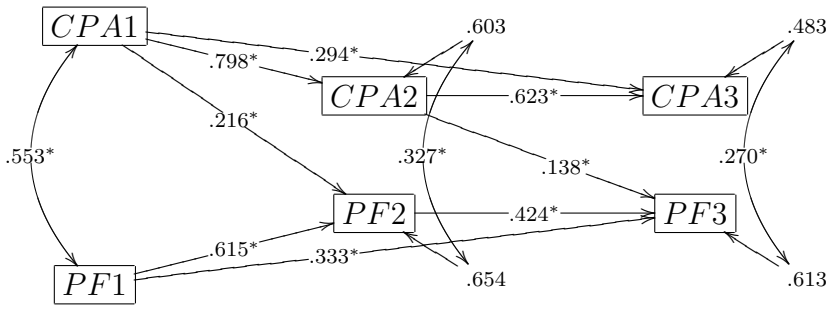


Figure 7.5: Final longitudinal path model CPA-PF

Given the possible method artifact regarding the CPAQ subscales (see Chapter 8), the distinctiveness between AE and PW was briefly examined in comparison with AAQ scores. The subscale scores were similarly distinct from PF (r = .47, .56, and .55, for AE and r = .49, .53, and .56, for PW, for stage 1, 2 and 3, respectively), and of similar stability (test-retest r = .77, .82, and .75 for AE, and .71, .81, .74 for PW). Therefore only the CPAQ and AAQ scores were considered further in this section.

In the situation where the two concepts are distinct and change across time, their interrelations in time can be explored. The total scores were used in three alternative longitudinal path models, specifying either reciprocal relationships between the two concepts between stages, or only one concept as predicting scores on both concepts at subsequent stages. The standardized parameter estimates for the best fitting model are reported in Figure 7.5. GOF indices are reported in Table 7.13[18] for the three models: Model 1 stipulating reciprocal relationships (paths from PF to CPA were not significant), Model 2 specifying only PF influencing subsequent CPA values, and Model 3 only CPA influencing subsequent PF values, as per Figure 7.5.

[18]Statistics for the MISSING=ML specification are reported.

However, all above analyses (correlations and cross-lag panel models) assumed that CPA and PF were measured without error. One method of accounting for measurement error is the correction for attenuation, which estimates the correlation between two variables in the hypothetical case of perfect reliability (Nunnally and Bernstein, 1994, p. 240–1, 256–8). Based on this formula, the two concepts still appeared as distinct ($r' = .69$, .74, and .76, for stage 1, 2 and 3, respectively), yet their longitudinal change appeared as due to measurement error (test-retest $r' = .99$, .96, and .94 for PF, and .90, .96, .88 for CPA).

The correction for attenuation assumes uncorrelated errors for the two tests, and independence of the errors of either test on the test scores themselves. For the correlations between CPA and PF, these assumptions are plausible, but the errors of test items are likely to be correlated for the same test longitudinally. In this situation, SEM allows for modelling correlated errors and was next applied. As explained in Appendix B, the use of selected items was considered more adequate in SEM.

Separate longitudinal SEmodels of each questionnaire[19] also resulted in estimated correlations between latent factors above .90 in most iterations, indicating multicollinearity at latent level and suggesting that according to this analysis no change was present. Therefore the comparison between CPA and PF was further explored cross-sectionally (in all three stages).

Two alternative models were developed for cross-sectional data, one with two latents representing PF and CPA as distinct concepts (Figure 7.6), the other with a single latent (Figure 7.7). Four items were selected from each questionnaire as indicators for the two constructs (see Appendix B for rationale; similar results were obtained with a different set of items). Method bias was modelled as an additional latent with equal unstandardized loadings on all items (fixed at 1, according to Billiet and McClendon, 2000). To account for possible distinct influences on the two constructs, several variables were included in the model, each measured by a single indicator with fixed error (as recommended by Hayduk, 1996): negative life events (NLE) and positive life events (PLE) were hypothesized to influence reports of PF, while pain-related aspects such as pain intensity (PI; the VAS of SF-MPQ), pain spread

[19]Not reported here for brevity. Models were specified with one latent factor for the questionnaire at each stage, error covariances for negatively worded items, error covariances between identical items at different stages, covariances between latents. Models were run both with all items and with selected items. Goodness of fit indices for models with all items showed inadequate fit to the data, likely due to both multicollinearity and problems at questionnaire level (reviewed in Chapter 8).

	PI	PS	CMB	PD	NLE
PS	.145*				
CMB	.210*	.372*			
PD	.104	.260*	.267*		
NLE	.067	.123	.203*	.043	
PLE	-.087	-.020	-.104	-.129	.307*

Table 7.14: Correlations between external variables in CPA-PF models

	Model 1			Model 2		
Statistic	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
S-B χ^2(df)	103.9(60) p<.001	113.1(60) p<.001	94.9(60) p=.003	104.6(61) p<.001	69.6(61) p=.21	112.3(61) p<.001
NFI	.78	.75	.78	.78	.85	.74
NNFI	.82	.78	.84	.82	.97	.77
CFI	.88	.86	.90	.88	.98	.85
RMSEA	.05 (.04–.07)	.06 (.05–.08)	.05 (.03–.07)	.05 (.04–.07)	.03 (.00–.05)	.06 (.05–.08)

Table 7.15: Goodness of fit statistics for longitudinal path models

(PS), pain duration (PD) and comorbidity (CMB) were hypothesized to influence reports of CPA (these variables were modelled to covary [20]).

GOF indices for both models are presented in Table 7.15. Parameter estimates are presented in the two figures for the first stage[21], with the exception of correlations between the external variables, which are presented in Table 7.14 (for the 2-factor model in the first stage; results were similar with the rest of the models). According to additional diagnostics (Wald and LM tests, residuals), suboptimal model fit was probably due partly to the relationships between the external variables (which are minimally related thus making covariances between some of them redundant, as were the relationships between some external variables and PF/CPA), but also to unique covariances between specific items and control variables. In the 2-latent models, additional covariances were suggested between some control variables and the latent to which they were not theoretically related in the model (e.g. pain intensity and PF), and between some items and the opposite latent. Moreover, the correlations between the two latents ranged from .72 to .85, and the χ^2 difference test reached significance only in stage 3 (i.e. $\chi^2(1) > 10.83$, at $\alpha = .001$). This information suggested a possible lack of distinctiveness between CPA and PF when structural relations are considered.

[20]The causal relationships in this part of the model were not specifically modelled, as the main focus of the model was identifying the relations between PF and CPA.

[21]Estimates were relatively similar in all stages.

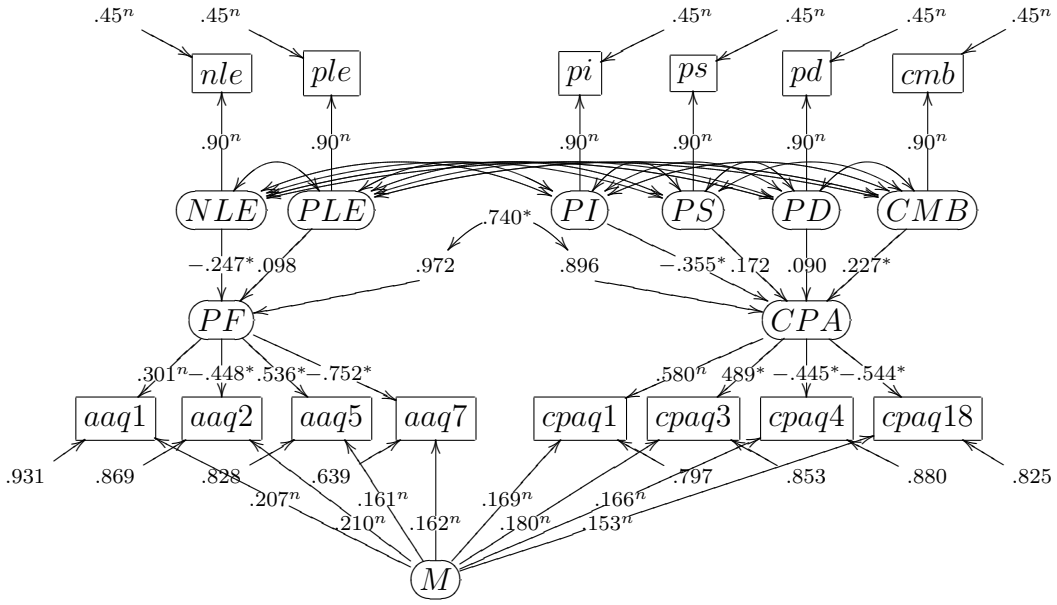


Figure 7.6: SEM cross-sectional CPA-PF (^{n} is a fixed, i.e. not tested, parameter)

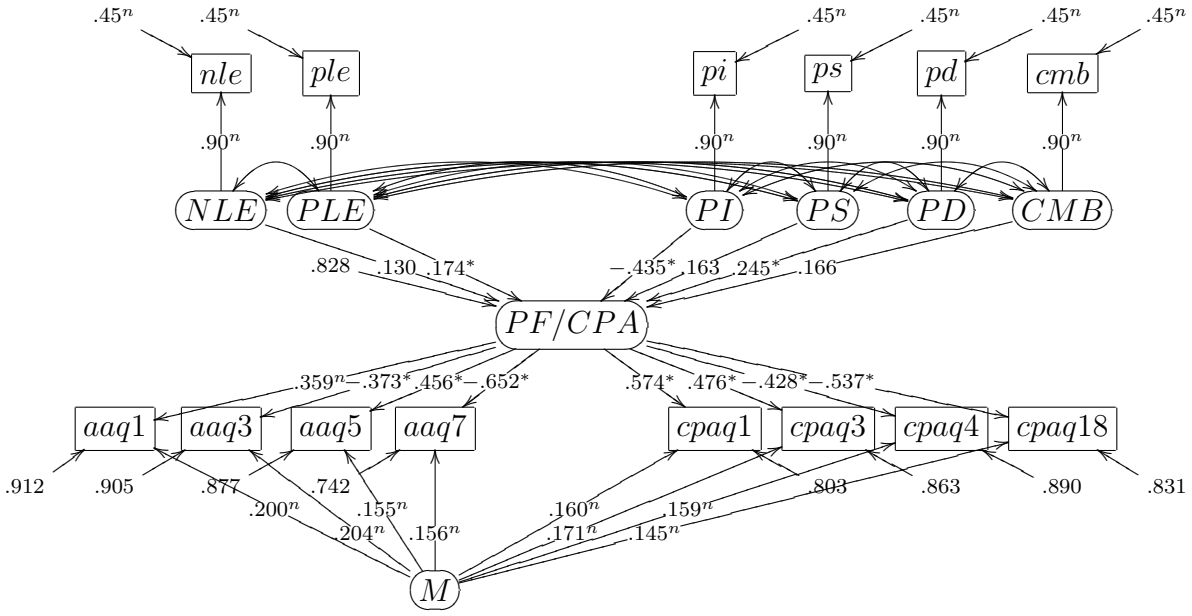


Figure 7.7: SEM cross-sectional CPA-PF - 1 latent

scale	1	2	3	4	5	6	7	8	9
1		.70**	.55**	.59**	-.48**	.59**	.57**	-.40**	-.37**
anger		.68**	.50**	.49**	-.48**	.51**	.55**	-.38**	-.52**
		.70**	.56**	.59**	-.52**	.61**	.50**	-.28**	-.40**
2	.70**		.70**	.68**	-.56**	.69**	.41**	-.38**	-.41**
sad	.66**		.62**	.56**	-.62**	.72**	.40**	-.41**	-.57**
	.66**		.71**	.64**	-.60**	.72**	.41**	-.38**	-.46**
3	.52**	.69**		.56**	-.43**	.61**	.49**	-.24**	-.32**
shame	.48**	.58**		.47**	-.42**	.59**	.41**	-.18*	-.36**
	.55**	.72**		.56**	-.49**	.62**	.42**	-.20**	-.40**
4	.59**	.67**	.53**		-.46**	.65**	.37**	-.26**	-.27**
anxiety	.47**	.56**	.46**		-.47**	.58**	.23**	-.18*	-.31**
	.59**	.63**	.55**		-.46**	.60**	.28**	-.16*	-.25**
5	-.46**	-.54**	-.39**	-.45**		-.54**	-.43**	.40**	.49**
joy	-.46**	-.60**	-.40**	-.47**		-.59**	-.41**	.42**	.57**
	-.52**	-.60**	-.44**	-.45**		-.63**	-.39**	.46**	.59**
6	.57**	.69**	.59**	.64**	-.51**		.41**	-.19**	-.36**
ID	.48**	.72**	.57**	.58**	-.59**		.40**	-.20**	-.50**
	.61**	.74**	.59**	.62**	-.63**		.42**	-.26**	-.43**
7	.62**	.42**	.47**	.38**	-.39**	.41**		-.33**	-.23**
ED	.58**	.43**	.45**	.22**	-.41**	.43**		-.23**	-.36**
	.51**	.41**	.46**	.27**	-.36**	.45**		-.24**	-.27**
8	-.39**	-.35**	-.21**	-.23**	.37**	-.14*	-.30**		.44**
IF	-.38**	-.39**	-.11	-.18*	.41**	-.19**	-.23**		.55**
	-.23**	-.34**	-.15*	-.16*	.42**	-.23**	-.16*		.44**
9	-.37**	-.39**	-.29**	-.28**	.49**	-.34**	-.19**	.44**	
EF	-.49**	-.57**	-.34**	-.33**	.58**	-.49**	-.36**	.56**	
	-.37**	-.48**	-.38**	-.26**	.57**	-.43**	-.27**	.43**	

Table 7.16: Correlations between emotions and ERSs (Spearman's ρ, left, and Pearson's r; the 3 estimates correspond to the 3 stages)

Emotions and emotion regulation strategies

Correlational analyses were performed to examine the relationships between emotions and ERSs. Dysfunctional ERSs were expected to be correlated positively to negative emotions and negatively to positive emotions, and functional ERSs to be correlated negatively to negative emotions and positively to positive emotions. Also, based on the theoretical basis of the REQ and the empirical results reported in Phillips and Power (2007), dysfunctional ERSs were expected to be unrelated with functional ERSs using the same resource type (external or internal), while dysfunctional ERSs using different resource types were expected to be related. Due to lack of normality and homoscedasticity in some variables, both Spearman's ρ and Pearson's r results are reported in Table 7.16.

Confirming expectations, dysfunctional ERSs were correlated positively to negative emotions and negatively to positive emotions, while functional ERSs were correlated negatively to negative emotions and positively to positive emotions. Notably, the

	Anger		Sad		Shame		Anxiety		Joy	
	P	S	P	S	P	S	P	S	P	S
ED	.62**	.62**	.34**	.34**	.43**	.38**	.34**	.32**	-.26**	-.26**
I2	.57**	.57**	.42**	.40**	.43**	.39**	.29**	.27**	-.37**	-.37**
	.58**	.55**	.42**	.40**	.46**	.42**	.31**	.29**	-.37**	-.34**
ID	.51**	.51**	.63**	.63**	.41**	.41**	.63**	.63**	-.47**	-.46**
I7	.48**	.46**	.61**	.60**	.45**	.44**	.57**	.57**	-.46**	-.45**
	.52**	.53**	.60**	.61**	.47**	.48**	.55**	.54**	-.48**	-.49**
IF	-.18**	-.16*	-.16*	-.15*	-.08	-.04	-.10	-.08	.16*	.15*
I9	-.15*	-.16*	-.17*	-.17*	-.11	-.07	-.08	-.07	.27**	.25**
	-.11	-.06	-.19*	-.16*	-.08	-.02	-.05	-.01	.25**	.22**
IF	-.09	-.10	-.14*	-.11	-.08	-.03	-.06	-.06	.21**	.20**
I16	-.13	-.14	-.19**	-.17*	.12	.16*	.07	.07	.15*	.14*
	-.12	-.12	-.20**	-.20**	-.08	-.10	.01	-.00	.23**	.21**
EF	-.29**	-.29**	-.22**	-.21**	-.25**	-.24**	-.11	-.12	.31**	.30**
I1	-.40**	-.38**	-.40**	-.41**	-.31**	-.31**	-.26**	-.27**	.40**	.41**
	-.36**	-.37**	-.36**	-.39**	-.37**	-.35**	-.21**	-.24**	.42**	.43**
EF	-.20**	-.23**	-.22**	-.24**	-.18**	-.18**	-.15*	-.17*	.33**	.35**
I3	-.41**	-.40**	-.41**	-.41**	-.29**	-.28**	-.26**	-.29**	.48**	.48**
	-.24**	-.25**	-.25**	-.27**	-.27**	-.27**	-.15*	-.16*	.51**	.52**
EF	-.18**	-.18**	-.17*	-.17**	-.14*	-.12	-.02	-.05	.16*	.16*
I8	-.30**	-.29**	-.30**	-.31**	-.22**	-.20**	-.17*	-.19**	.33**	.32**
	-.15*	-.13	-.22**	-.23**	-.19*	-.15*	-.02	-.02	.28**	.28**

Table 7.17: Correlations between emotions and selected REQ items (Pearson's r, left, and Spearman's ρ; the 3 estimates correspond to the 3 stages)

lowest associations were identified between internal functional strategies and shame and anxiety. Also, external and internal ERSs of the same type (dysfunctional or functional) were correlated, confirming the expected patterns. Contrary to the lack of relationship between functional and dysfunctional ERSs of the same orientation (external or internal) stipulated by Phillips and Power (2007), all correlations in the ERSs matrix were significant, although the effect size of the unexpected associations was substantially smaller than of the expected relations.

Since the REQ subscales had suboptimal properties in this sample (see Chapter 8), the analyses were repeated with selected items (Table 7.17). Mirroring the results at subscale level, the items referring to dysfunctional ERSs (items 2 and 7) were related to increased reports of negative emotions and decreased reports of joy. External functional ERSs (items 1, 3 and 8) had the opposite relationships. The low associations at subscale level between internal functional strategies and shame and anxiety consistently failed to reach significance at item level; items 9 and 16 were also weakly related to anger and sadness, but positive associations with happiness/joy were significant.

		items							
items	stage	1	2	3	4	5	6	7	8
1	1		.41**	-.13*	-.11	.54**	.58**	.10	.50**
conse-	2		.40**	-.12	-.12	.68**	.52**	-.01	.58**
quences	3		.44**	-.29**	-.18**	.66**	.63**	-.00	.65**
2	1	.23**		-.14*	-.13*	.33**	.26**	.07	.22**
timeline	2	.30**		.02	-.12	.37**	.22**	.15*	.21**
	3	.29**		-.12	-.16*	.42**	.36**	.15*	.34**
3	1	-.10*	-.13**		.44**	-.09	-.25**	.27**	-.17**
personal	2	-.11*	.03		.54**	-.03	-.14*	.28**	-.13
control	3	-.21**	-.07		.43**	-.19**	-.35**	.32**	-.21**
4	1	-.07	-.09	.36**		-.06	-.19**	.27**	-.13*
treatment	2	-.14**	-.06	.43**		-.05	-.15*	.30**	-.13
control	3	-.11*	-.12*	.36**		-.10	-.24**	.20**	-.15*
5	1	.47**	.21**	-.08	-.05		.41**	.04	.19**
identity	2	.53**	.29**	-.03	-.05		.50**	.00	.44**
	3	.54**	.30**	-.15**	-.09		.49**	.02	.44**
6	1	.48**	.20**	-.20**	-.16**	.38**		-.07	.59**
concern	2	.40**	.20**	-.10*	-.13**	.40**		-.15*	.65**
	3	.51**	.26**	-.29**	-.20**	.41**		-.21**	.67**
7	1	.12*	.12*	.18**	.19**	.11*	-.01		-.08
under-	2	.04	.20**	.24**	.22**	.04	-.09		-.05
standing	3	.03	.17**	.24**	.19**	.05	-.13*		-.09
8	1	.41**	.14**	-.13**	-.09*	.21**	.51**	-.03	
emotional	2	.48**	.18**	-.08	-.10*	.37**	.51**	-.01	
response	3	.51**	.24**	-.14**	-.10*	.35**	.53**	-.03	

Table 7.18: Correlations matrix - BIPQ items (Kendall τ, left, and Pearson r)

Remarkably, estimates were relatively stable for the three stages and the parametric versus non-parametric tests, increasing the confidence in these results in the present sample. The stability of the estimates was obviously related to the stability of the measures. As reported in Chapter 8, estimates ranged from .71 to .81 for emotions and from .65 to .84 for ERSs (with correction for attenuation values ranged from .82 to .99 for emotions and .92 to above unity for ERSs, indicating that when measurement error was accounted for, values for emotional life variables were stable). One-way ANOVA tests performed for all 9 subscales ($\alpha = .006$ for multiple comparison) also indicated nonsignificant overall mean changes, with only sadness approaching significance at $F(2, 362) = 4.92$, $p = .008$ due to a significant but minor decrease in stage 3 compared to previous two stages (mean values 3.6 to 3.4). No changes were found in the selected REQ items either.

Illness perceptions

A first research question regarding IPs is whether they reflect a single dimension, justifying therefore the use of a total score (e.g. Radat et al., 2009). The inspection

of the matrix of correlations between BIPQ items (Table 7.18[22]) revealed a non-homogeneous pattern, a first indication that BIPQ does not measure a unidimensional construct. Items related to perceived illness severity (consequences, identity, concern, emotional response) were more strongly correlated with each other, but were weakly related to personal or treatment control (which had medium correlations). Timeline had small correlations to all other items, except the control ones, while understanding had small positive correlations only with the control items and timeline. Although the pattern was in some respects different from those reported by Moss-Morris et al. (2002) based on results from IPQ-R applied on a mixed health conditions sample[23], the common aspect was a lack of close associations between the various subscales, suggesting that the items should be analysed separately. Similar results were obtained via PCA, presented below.

Since a total score was not justified, it was considered relevant to explore the suitability of the alternative data reduction strategy proposed by Hobro et al. (2004): identifying data clusters. Hobro et al. (2004) used the IPQ-R Health Threat Representations subscales (timeline, consequences, personal control, treatment control, illness coherence, timeline cyclical and emotional representations) to categorise chronic pain sufferers in a sample of newly referred patients to a local pain clinic (with various chronic pain conditions). Using a hierarchical clustering procedure they identified two groups 'adaptors' and 'non-adaptors' that accounted for 75% of respondents, and were shown to differ on several other measures of pain, mood and functioning. Overall, the 'adaptors' had beliefs of shorter timeline, more personal and treatment control, better understanding, less emotional distress, and reported perception of more pain, more need for medication, concern about the treatment effects, depression, poorer physical functioning and general and mental health, and less energy/ vitality. The groups did not differ regarding gender, age, work status, pain location and duration, expectations from the pain center, nor regarding anxiety levels, physical and emotional role limits and social functioning. The authors also found support for this classification in the similarity with the WHYMPI-derived 'adaptive copers' and 'dysfunctional' groups by (Turk and Rudy, 1990) and were optimistic regarding its clinical utility. Nevertheless they stated the necessity of

[22] As the data are ordinal and departed substantially from normality, both Kendall τ and Pearson correlations were reported comparatively.

[23] For example the acute/chronic timeline was more related to both control subscales, while the identity subscale was unrelated to emotional IPs.

replicating these results. It is therefore relevant to test whether the results of Hobro et al. (2004) replicate in the present study[24], as a first step towards identifying the relevance of these groups on health status and the relationship with the other concepts.

The authors reported using hierarchical clustering procedure for average linkage (between groups) using agglomerative scheduling, in SPSS. They did not state the similarity measure used, but it is likely they have used squared Euclidean distance, as Clatworthy et al. (2005) described it as a frequent choice when "grouping like-minded individuals" is intended[25]. They determined the number of groups by examination of the agglomeration schedule for inconsistently large increases in the similarity measure, which led to a 10-cluster solution including 118 of the original 130 participants (the elimination of the additional 12 cases is not detailed). They reported two main clusters of 60 and 38 participants, respectively, and 8 remaining clusters which included the remaining 20 and were excluded from further analyses. No data regarding the agglomeration schedule were provided for assessment by readers. No attempts to assess the stability of the clusters were reported. They validated the two main groups by identifying differences regarding several demographic and clinically relevant variables, as described above.

In the present sample, cluster analyses were performed on data from the three stages separately. The coefficients for the last 10 steps of the agglomeration schedules are presented in Table 7.19. Clatworthy et al. (2005) advised against selecting the same number of clusters for replication studies without searching for a more appropriate solution in the data, as it may result in a bias in favour of replicating previous results. Therefore specific solutions were first explored based on the agglomeration schedule for each stage[26].

For all stages, the solutions selected based on the largest increases in distance coefficients (6 and 3 for stage 1; 7, 5 and 3 for stage 2; 5, 4 and 2 for stage 3) did not support a two-cluster solution: the first cluster included between 88 and 99% of the sample. Exploring previous steps of the agglomeration schedules revealed no other notable gaps.

[24]With the exception of 'concern' and 'identity' (and 'timeline cyclical' missing), the subscales of the IPQ-R and BIPQ are analogous, therefore a similar solution would be expected.

[25]Two of the authors of Hobro et al. (2004) also authored Clatworthy et al. (2005)

[26]Determining the number of clusters by this method is largely subjective (Clatworthy et al., 2005). However, formal rules such as the pseudo-F statistic or the cubic clustering criterion are not available in SPSS.

	Stage		
No. of clusters	1	2	3
10	82.429	80.333	80.000
9	86.268	85.626	80.050
8	88.500	87.694	81.675
7	90.454	92.578	84.107
6	93.500	106.333	98.402
5	117.689	116.192	100.564
4	123.796	133.044	120.600
3	134.552	146.080	139.489
2	164.620	165.173	140.835
1	169.095	166.211	157.138

Table 7.19: Distance coefficients for the last 10 steps of the agglomeration schedules - cluster analysis of BIPQ

Examining the 10-cluster solutions for the three stages revealed the presence of two well represented groups, which were kept for further analyses, following Hobro et al. (2004). The differences between the two groups had a similar pattern to the ones identified by Hobro et al. (2004), i.e. one of the groups reported relatively less consequences, shorter timeframe, higher personal and treatment control, more understanding, less emotional impact, together with less symptoms and less concern (all t-tests significant at $\alpha = .01$, with the exception of 'understanding' in the last two stages). Therefore they were considered as representing the same two groups labelled by Hobro et al. (2004) 'adaptors' and 'non-adaptors'. They also differed in terms of pain intensity reported (T-PRI, VAS, and PPI indices of SF-MPQ), pain-related disability (RM-SIP) and frequency of GP visits[27], although they were not consistently different in terms of gender, age, vocational and marital status, education, number of comorbid conditions (including the presence of depression), pain location and spread, pain duration, and other health care use variables (medication, specialised help and self-help).

However, Hobro et al.'s (2004) analysis omitted to investigate two important features of this cluster solution: stability and distinctiveness (as explained in a previous section). The question of stability could not be answered in their study due to its cross-sectional design and relatively low number of participants. The longitudinal design of the present study made a response possible. The allocation of participants to groups was not random ($\chi^2(1) = 15$ to 24, $p < .001$), but had a low stability (Pearson's $r = .30$ to .41). This was apparent also in the differences in relative

[27] As these results were also part of the answer to a different research question, they are reported in detail in Section 7.7.1.

	Stage		
	1	2	3
1 - 'non-adaptors'	28.5	54.4	19.6
2 - 'adaptors'	55.9	18.1	43.0
other	13.7	11.1	15.6
missing	1.9	16.3	21.9

Table 7.20: Percentages of participants allocated to the different groups - 10-cluster solution, BIPQ

	Stages			
Groups	1to2	2to3	1to3	All 3
1 ('non-adaptors') - stable	20.0	18.1	10.7	10.0
2 ('adaptors') - stable	13.7	13.3	29.3	10.0
Other - stable	4.8	7.4	5.6	3.7
Change between 1 and 2	30.0	27.8	14.1	35.6
Change between other and 1 or 2	13.7	10.7	16.7	21.1
Missing	17.8	22.6	23.7	19.6

Table 7.21: Percentages of participants allocated to the same or different groups across stages - 10-cluster solution, BIPQ

percentages of the groups between stages (Table 7.20) and the sizable percentages of participants that were attributed to different groups between stages (Table 7.21).

The instability of the classification may be due partly to the instability of the items themselves[28], but also to the lack of distinctiveness between the adaptors and non-adaptors groups. Indeed, no bivariate patterns were apparent in the present sample in any of the three stages (graphs not shown). The illness perception variables showed a 2-group structure on the first two principal components: control and understanding in one group, the rest in the second group (Figure 7.8). Based on the item groupings, the two components can be interpreted as 'low cognitive control - high emotional impact', and 'high cognitive control - moderate emotional impact'[29]. Expectedly, the first component had a substantial negative skew reflecting the increased impact of chronic pain, while the second component had a rather normal distribution (in all three stages). Nevertheless, the distribution of individual cases in this two-dimensional space did not show any clear patterns. Figure 7.9 illustrates this lack of distinct clusters and also shows where the adaptors and non-adaptors are situated in this multivariate space. Even if the groups are arguably occupying relatively different areas, there is a large amount of overlap in each stage and a

[28] As reported in Chapter 8, the stability estimates of BIPQ items fluctuated between .22 and .75 (test-retest Kendall τ correlations between stages).

[29] This structure is in accord with Leventhal and colleagues' SRM and parallel-processing model, and also very informative for the application of these models in chronic pain (see Chapter 9 for discussion).

Stages	1-2		2-3		1-3	
Test	P	S	P	S	P	S
PC1	.75**	.71**	.82**	.80**	.76**	.75**
PC2	.53**	.51**	.48**	.47**	.49**	.49**

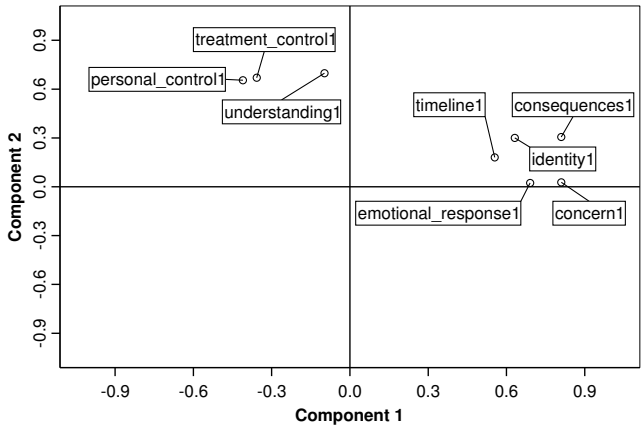
Table 7.22: Stability estimates for the two principal components of BIPQ

large proportion of cases that are both classified as 'adaptors', 'non-adaptors' or excluded from these groups in different stages.

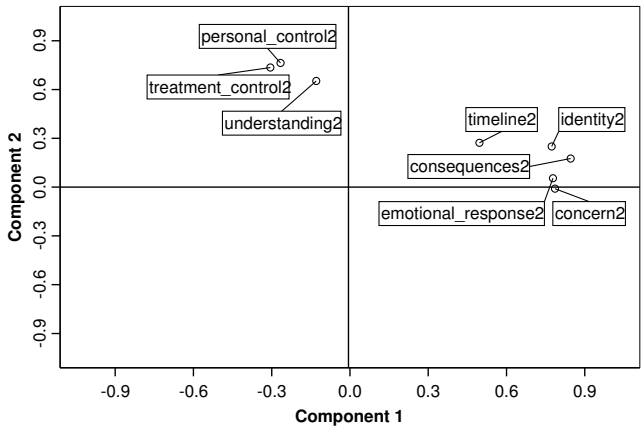
These results are likely due to the low temporal stability of the subscales (which is expected from single-item measures) and also to the instability of the classification method on this data set. Therefore it is relevant to explore the stability of the two dimensions between the three stages. As reported in Table 7.22, the first dimension was relatively stable, while the second showed substantial fluctuations, indicating that while the psychological impact of the condition was relatively constant, the participants perceptions of cognitive control were oscillating (probably with environment changes). However, results of one-way ANOVA tests were non-significant, indicating no substantial overall change; $F(1.87, 377.7) = 2.4$, $p = .09$, and $F(2, 408) = .13$, $p = .88$. Change was also not significant when considering self-reported participation to pain management programmes or other medical and demographic variables such as pain location, pain duration, comorbidity, education, marital and vocational status, gender (interaction effects in mixed design ANOVA tests non-significant at $\alpha = .01$), indicating that overall lack of change in the two PCs is not masking possible relevant changes in patient subgroups[30].

PCA describes the data in terms of several uncorrelated factors that account for the largest proportion of variance in all variables considered, and therefore it assumes that the tendency for large variation is also "interestingly structured variation", which might not always hold in practice (Everitt et al., 2001, p. 28). Therefore lack of structure in a PCA does not automatically imply lack of multivariate structure. Still, the characterisation of groups resulted from cluster analyses tends to rely on group differences between the variables included, such as the non-adaptors reporting more consequences and concern and less control and understanding than

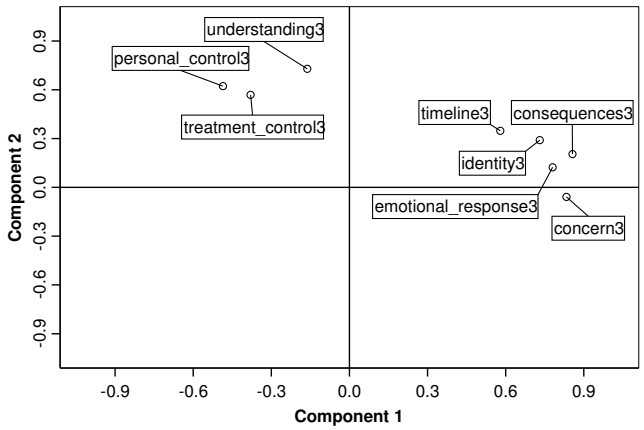
[30]It is also possible that data reduction (via total score, clustering, or principal components) masks significant changes in selected IPs between the three stages. Therefore, one-way ANOVA tests were performed for all 8 continuous subscales ($\alpha = .006$ for multiple comparison). Indeed, there were significant decreases only in reports of perceived consequences ($F(2, 408) = 9.1$, $p < .001$, significant change between first and second stage, sustained in stage 3) and concern ($F(2, 408) = 7.0$, $p = .001$, similar pattern), and an increasing trend in personal control, approaching significance ($F(2, 408) = 9.1$, $p = .007$), with no change in the rest of the IPs. These results support the approach of treating the BIPQ subscales as distinct concepts.



(a) stage 1

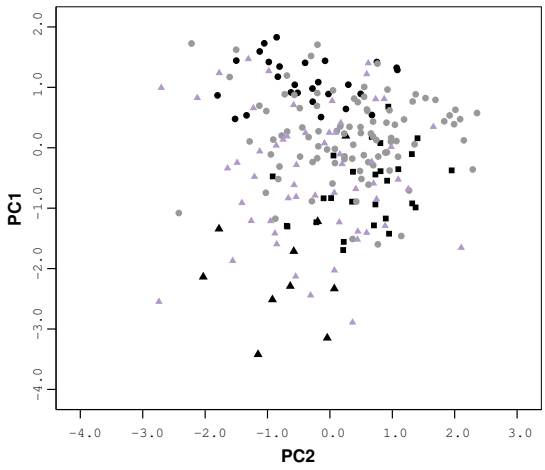


(b) stage 2

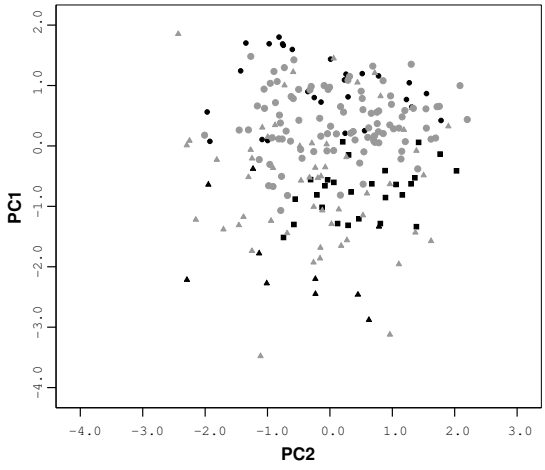


(c) stage 3

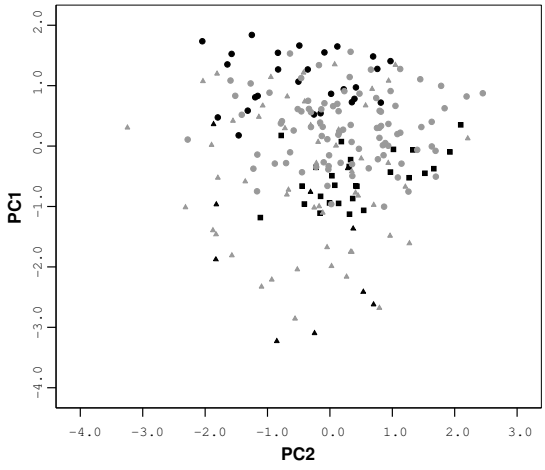
Figure 7.8: PCA - component plots for BIPQ items



(a) stage 1



(b) stage 2



(c) stage 3

Figure 7.9: PCA - cases with stable and unstable categorisation: ● - stable 'non-adaptors' (1); ■ - stable 'adaptors' (2); ▲ - stable 'other' (3); ● change between (1) and (2); ▲ change between (3) and (1) or (2).

the adaptors[31]. Besides, other methods of projection pursuit (briefly described by Everitt et al., 2001) are difficult to justify and interpret theoretically and are also not available in SPSS.

MDS computed on the same similarity measure used in the cluster analysis (squared Euclidean distance) can provide another graphical representation of the distinctiveness of the two-factor solution discussed above. No distinct clusters appeared in this two-dimensional space either (results not shown). These exploratory attempts failed to support the possibility the clustering solution is indicating a clear two-group distinction.

Thus, although a similar two-cluster solution was found in the present sample, it did not show the distinctiveness and stability required from an adequate clustering solution. The categories of 'adaptors' and 'nonadaptors' were further examined in relation to the study hypotheses in the next sections, but were considered a method of data dissection, not distinct patient groups.

To my knowledge, the analysis of the causality-related IPs in chronic pain has not been yet explored in relation to the other IPs, except a correlational analysis based on IPQ-R scores in Moss-Morris et al. (2002), which included a chronic pain subsample together with other subsamples with asthma, diabetes, MS, etc. Therefore, this analysis was performed in an exploratory manner. No differences in causal IPs were identified between clusters in any stage (all χ^2 tests non-significant even at non-corrected $\alpha = .05$). Moreover, causal IPs were mostly unrelated with any other IPs (t-tests did not show any consistent significance pattern between the three stages, at a significance level corrected for multiple comparison[32]). A single exception was identified regarding anatomical/ physiological descriptions (and the wider category of medical attributions) which were consistently associated with perceptions of understanding ($t(226.3) = 3.9$, $p < .001$, $t(212.2) = 2.8$, $p = .006$, and $t(205) = 2.1$, $p = .03$ for anatomical descriptions; $t(191.4) = 2.4$, $p = .02$, $t(165.6) = 3.0$, $p = .003$, and $t(205) = 3.0$, $p = .003$ for medical attributions).

[31] This definition is actually reproducing the first component in the PCA solution

[32] A less conservative Bonferroni correction was considered for only the 8 simultaneous t-tests, resulting in an $\alpha = .006$, even if these 8 tests were performed for all three stages for all 10 categories of causal IPs. However, the results were considered only if they presented consistency across stages.

Health status indicators

Correlational analyses were first performed for indicators of pain-related disability (RM-SIP), perceived pain intensity indices (T-PRI, S-PRI, A-PRI, VAS and PPI, based on the SF-MPQ), health care utilisation (proxy indicators: number of types of medication used, number of specialist treatments used, number of self-help methods used, and low versus high number of GP visits), and vocational status (full-time or part-time working or training versus unable to work because of pain, excluding the category of people unable to work for other reasons, such as retirement) to investigate the possible existence of a homogeneous pattern of interrelations that would justify a single pain severity score (Table 7.23[33]).

The inspection of the correlation matrix indicated that a total 'health status' score would not be adequate, due to the heterogeneity of the correlation pattern. Pain intensity and disability indicators showed medium correlations of similar sizes (with the exception of the S-PRI, A-PRI and T-PRI which were more strongly correlated, due to the fact that T-PRI scores are based on the other two scores, and to the common method of endorsing pain descriptors used in both S-PRI and A-PRI items). Medication had small to medium sized correlations to all other variables, while GP visits and work status were associated with most other variables, with the exception of specialist treatments and self-help (the higher association between work status and disability is to be expected, due to the conceptual similarity between the two indicators. These last two indicators were unrelated to most other variables except medication, which suggested they were not valid indicators of health status, but rather depending on other factors (such as perhaps the information accessed, the financial possibilities or the specific relevance of different methods for the patient's medical condition). In addition, their reliability is questionable (see Chapter 8). Therefore they were not further considered in subsequent analyses of health status.

The structure of the relationships between the remaining variables was further explored in two alternative CFA models (Figures 7.10 and 7.11), the first specifying a single factor, the second separating the pain experience indicators (SF-MPQ indices) from the variables related to pain behaviours (including disability, health

[33]Both Spearman and Pearson estimates are provided due to lack of normality of some variables and ordinal status of others. For the dichotomous variables (GP visits and work status), t-tests and χ^2 tests gave similar results (not reported).

	1	2	3	4	5	6	7	8	9	10	11
1		.97**	.86**	.61**	.64**	.48**	.38**	.12	.11	.30**	.26**
T-PRI		.96**	.85**	.64**	.57**	.55**	.40**	.16*	.23**	.25**	.28**
		.98**	.87**	.71**	.56**	.66**	.33**	.13	.12	.29**	.38**
2	.97**		.72**	.59**	.62**	.46**	.39**	.10	.13*	.28**	.24**
S-PRI	.97**		.69**	.62**	.54**	.52**	.35**	.18**	.23**	.23**	.26**
	.98**		.75**	.68**	.51**	.63**	.32**	.12	.12	.26**	.36**
3	.87**	.73**		.54**	.55**	.45**	.28**	.14*	.06	.28**	.25**
A-PRI	.84**	.69**		.57**	.55**	.52**	.38**	.15*	.16*	.25**	.29**
	.86**	.75**		.64**	.59**	.60**	.29**	.14*	.08	.31**	.38**
4	.61**	.59**	.55**		.71**	.51**	.21**	-.05	.00	.32**	.32**
VAS	.63**	.60**	.59**		.74**	.58**	.27**	.12	.12	.33**	.28**
	.72**	.69**	.66**		.72**	.61**	.25**	.18*	.07	.34**	.35**
5	.64**	.62**	.56**	.71**		.49**	.21**	.07	.08	.30**	.29**
PPI	.56**	.52**	.56**	.76**		.49**	.23**	.08	.04	.36**	.31**
	.55**	.50**	.59**	.70**		.56**	.19**	.25**	.09	.32**	.33**
6	.48**	.44**	.46**	.48**	.47**		.31**	.07	.06	.33**	.51**
RM-	.54**	.51**	.52**	.54**	.48**		.34**	.22**	.09	.29**	.58**
SIP	.66**	.63**	.61**	.60**	.55**		.38**	.16*	.12	.32**	.61**
7	.38**	.38**	.29**	.22**	.22**	.31**		.21**	.31**	.23**	.20**
Medi-	.40**	.36**	.39**	.29**	.24**	.35**		.19**	.25**	.17*	.14
cation	.33**	.31**	.30**	.24**	.18**	.37**		.18**	.28**	.21**	.24**
8	.10	.08	.12	-.04	.07	.07	.20**		.18**	.17**	-.02
Treat-	.16*	.17*	.16*	.13	.09	.20**	.22**		.12	.09	.01
ments	.12	.11	.15*	.17*	.25**	.15*	.18**		.26**	.19**	.03
9	.13*	.13*	.08	-.01	.08	.05	.32**	.18**		.06	.03
Self-	.24**	.24**	.16*	.11	.03	.07	.26**	.13*		.06	.09
help	.11	.12	.07	.06	.09	.11	.28**	.26**		.04	.12
10	.29**	.27**	.28**	.31**	.29**	.33**	.24**	.16**	.07		.16*
GP	.26**	.24**	.27**	.32**	.36**	.28**	.16*	.09	.05		.21**
visits	.28**	.26**	.30**	.35**	.32**	.33**	.22**	.18**	.03		.32**
11	.26*	.24**	.26**	.30**	.28**	.51**	.20**	-.03	.04	.16*	
Work	.29**	.27**	.29**	.25**	.30**	.58**	.14	.00	.08	.21**	
status	.38**	.35**	.38**	.33**	.31**	.61**	.24**	.02	.11	.32**	

Table 7.23: Correlations between health status indicators (Spearman's ρ, left, and Pearson's r; the 3 estimates correspond to the 3 stages)

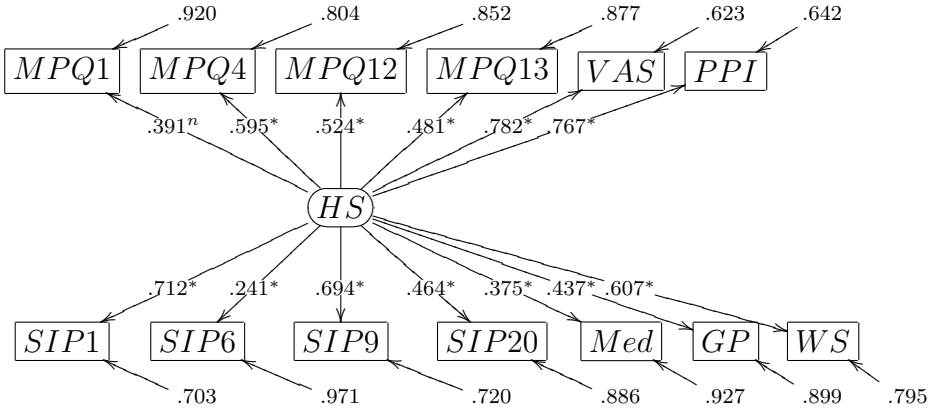


Figure 7.10: SEM cross-sectional health status indicators - 1 factor

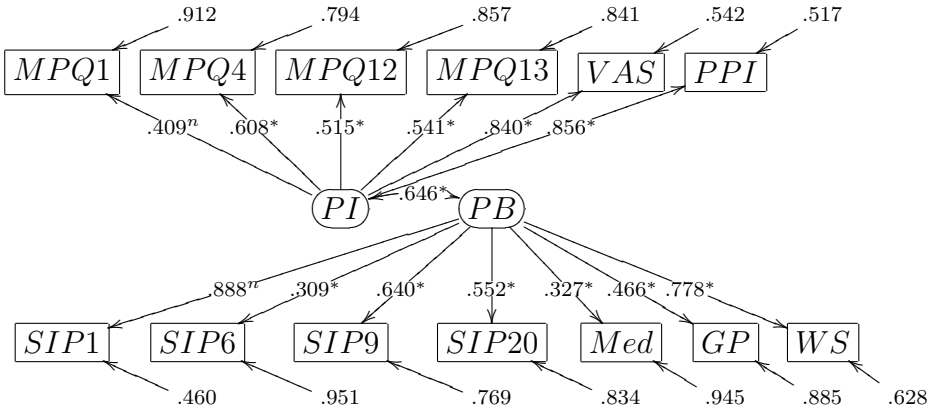


Figure 7.11: SEM cross-sectional health status indicators - 2 factors

care use, and work status)[34]. Both models had a good fit to the data (Table 7.24), however the 2-factor model consistently had a comparatively better fit, indicating that separating pain experience from pain behaviours is not redundant (all χ^2 difference tests were significant at $\alpha = .001$, i.e. $\chi^2(1) > 10.83$). A further test of the distinctiveness of these indicators is in relation to other concepts, which was explored in Section 7.7.

[34]Tests were performed for both total scores and selected items, considering binary variables as both categorical and continuous (given that considering the variables categorical increases sample size requirements and ignores variance as it is based on a correlation matrix, while assuming continuous status for all variables is a possible source of model misspecification even if robust methods are used; see Appendix B for details), and with both the exclusion and inclusion of 'work status' (given that excluding the 'unable to work because of other reason' category reduces sample size substantially). Only item level analyses with categorical items specification and 'work status' included are reported, for brevity. Parameter estimates are provided for the first stage data.

	Model 1			Model 2		
Statistic	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
S-B χ^2(df)	74.7(65) p=.19	101.5(65) p=.003	68.0(65) p=.37	32.3(64) p=.99	55.9(64) p=.75	28.3(64) p=.99
NFI	.86	.82	.89	.94	.90	.96
NNFI	.98	.91	.99	1.09	1.02	1.08
CFI	.98	.93	1.00	1.00	1.00	1.00
RMSEA	.03 (.00–.05)	.06 (.03–.08)	.02 (.00–.05)	.00 –	.00 (.00–.03)	.00 –

Table 7.24: Goodness of fit statistics for CFA models, HSIs

In terms of stability, while disability showed a high level of stability (RM-SIP $r = .83 - .86$), as did work status (83% of the participants remained in their initial status throughout the study), both SF-MPQ indices ($r = .49 - .71$, see Table 8.16) and HCU indicators ($r = .38 - .60$, see Table 8.28) fluctuated considerably. However, one-way ANOVA tests (and Friedman and Cochran's Q tests for ordinal and binary variables) indicated significant, although minor changes, only in disability ($F(2, 402) = 7.63$, $p = .001$, a steady decrease from a mean of 12.9 in the first stage to 11.9 in the third).

7.6.2 Interrelations

Emotions and acceptance

The relationships of both CPA and PF with emotion variables was explored via correlational analyses. As seen in Table 7.25, CPA and PF were, as expected, consistently related to more frequent positive emotions and functional ERSs and less frequent negative emotions and dysfunctional ERSs. The analyses were repeated at the item level concerning REQ (Table 7.26); the relations between individual REQ item scores and total CPA and PF scores were consistently significant only for the selected dysfunctional ERSs, while the external functional ERSs were comparatively less and inconsistently associated to CPA and PF. The internal functional ERSs showed weak or non-significant correlations.

It is important to note the differences between the two CPAQ subscales in terms of emotional variables. AE had comparatively stronger correlations only with joy/happiness and functional ERSs total scores. At the item level, only the internal functional ERSs were associated significantly with AE, but not with PW, the rest of the items showing similar effect sizes.

	AE		PW		CPA		PF	
	P	S	P	S	P	S	P	S
Anger	-.43**	-.41**	-.37**	-.38**	-.48**	-.47**	-.64**	-.63**
	-.50**	-.48**	-.48**	-.46**	-.55**	-.53**	-.56**	-.54**
	-.44**	-.40**	-.40**	-.42**	-.49**	-.48**	-.58**	-.57**
Sad	-.47**	-.45**	-.38**	-.37**	-.50**	-.49**	-.64**	-.65**
	-.56**	-.54**	-.55**	-.54**	-.62**	-.61**	-.68**	-.68**
	-.55**	-.52**	-.52**	-.53**	-.63**	-.61**	-.69**	-.71**
Shame	-.27**	-.22**	-.27**	-.22**	-.31**	-.26**	-.54**	-.53**
	-.29**	-.24**	-.47**	-.44**	-.41**	-.36**	-.52**	-.53**
	-.35**	-.31**	-.44**	-.43**	-.45**	-.44**	-.63**	-.64**
Anxiety	-.40**	-.38**	-.43**	-.41**	-.49**	-.46**	-.62**	-.62**
	-.39**	-.38**	-.50**	-.49**	-.49**	-.48**	-.61**	-.59**
	-.39**	-.37**	-.42**	-.41**	-.47**	-.46**	.62**	.63**
Joy	.46**	.46**	.24**	.20**	.43**	.41**	.53**	.53**
	.54**	.53**	.35**	.31**	.51**	.48**	.56**	.53**
	.63**	.60**	.40**	.39**	.61**	.59**	.65**	.63**
ID	-.39**	-.35**	-.36**	-.32**	-.44**	-.40**	-.63**	-.62**
	-.39**	-.37**	-.48**	-.45**	-.48**	-.45**	-.66**	-.65**
	-.45**	-.42**	-.46**	-.43**	-.53**	-.49**	-.69**	-.69**
ED	-.22**	-.16**	-.19**	-.16**	-.25**	-.17**	-.45**	-.42**
	-.23**	-.22**	-.24**	-.24**	-.26**	-.26**	-.33**	-.33**
	-.28**	-.22**	-.16*	-.17*	-.27**	-.22**	-.34**	-.37**
IF	.35**	.31**	.15*	.13*	.31**	.26**	.39**	.36**
	.43**	.41**	.20**	.20**	.37**	.35**	.38**	.35**
	.52**	.51**	.19**	.18**	.44**	.43**	.32**	.31**
EF	.30**	.30**	.11	.13*	.25**	.27**	.29**	.29**
	.49**	.48**	.35**	.34**	.48**	.46**	.45**	.43**
	.43**	.44**	.36**	.37**	.46**	.47**	.45**	.42**

Table 7.25: Correlations between acceptance and emotions (P - Pearson's r, and S - Spearman's ρ)

Illness perceptions and acceptance

The two categories of patients identified by cluster analysis of IPs differed significantly in terms of acceptance scores (CPA and PF): the patients characterised by increased perception of consequences, chronic timeline, concern, illness identity, and emotional impact and lower personal and treatment control and understanding were also less accepting of their condition, as presented in Table 7.27[35].

In terms of individual IPs, perceptions of consequences, timeline, identity (symptoms), concern and emotional response, were lower with increased CPA, while perceived illness understanding was unrelated to acceptance, and personal and treatment control showed medium positive associations to acceptance (Table 7.28).

[35]Results were similar when considering the two CPAQ subscales, AE and PW. Not reported here for brevity.

	AE		PW		CPA		PF	
	P	S	P	S	P	S	P	S
ED - I2	-.18**	-.17**	-.18**	-.16**	-.21**	-.18**	-.38**	-.35**
I take my feelings out	-.24**	-.23**	-.23**	-.22**	-.26**	-.26**	-.30**	-.31**
on others verbally	-.27**	-.24**	-.22**	-.22**	-.29**	-.26**	-.34**	-.35**
ID - I7	-.32**	-.31**	-.33**	-.30**	-.38**	-.37**	-.55**	-.55**
I dwell on my thoughts	-.29**	-.27**	-.39**	-.37**	-.37**	-.35**	-.57**	-.57**
and feelings	-.30**	-.30**	-.38**	-.37**	-.39**	-.39**	-.57**	-.58**
IF - I9	.23**	.21**	.06	.05	.18**	.16**	.12	.10
I review (rethink)	.23**	.21**	.08	.05	.19**	.14*	.17*	.15*
my goals or plans	.29**	.27**	.03	.03	.21**	.18**	.08	.07
IF - I16	.21**	.18**	.02	-.01	.14*	.10	.11	.10
I plan what I could	.28**	.27**	-.04	-.04	.16*	.16*	.10	.07
do better next time	.28**	.28**	.06	.10	.22**	.23**	.09	.11
EF - I1	.14*	.16**	.02	.03	.10	.13*	.14*	.16*
I talk to someone	.25**	.25**	.25**	.24**	.28**	.26**	.30**	.29**
about how I feel	.17*	.19**	.22**	.22**	.22**	.23**	.36**	.35**
EF - I3	.08	.09	.07	.08	.09	.10	.15*	.17**
I seek physical contact	.29**	.26**	.23**	.20**	.30**	.25**	.31**	.28**
from friends or family	.25**	.24**	.22**	.21**	.28**	.26**	.37**	.36**
EF - I8	.06	.09	-.02	.04	.03	.07	.11	.14*
I ask others	.21**	.21**	.18**	.18**	.22**	.21**	.29**	.31**
for advice	.18**	.21**	.21**	.21**	.22**	.24**	.18**	.15*

Table 7.26: Correlations between acceptance and selected REQ items (P - Pearson's r, and S - Spearman's ρ)

	Stage	t-test	Mean values		Pearson's r
			Adaptors	Non-adaptors	
CPA	1	$t(226) = 7.8$, $p < .001$,	2.0	2.8	.46**
	2	$t(194) = 10$, $p < .001$,	2.3	3.6	.58**
	3	$t(167) = 7.3$, $p < .001$,	2.0	3.0	.49**
PF	1	$t(226) = 3.1$, $p = .002$,	4.8	3.2	.20**
	2	$t(194) = 5.6$, $p < .001$,	4.2	3.4	.37**
	3	$t(167) = 4.7$, $p < .001$,	4.4	3.7	.35**

Table 7.27: Differences in acceptance between the IP clusters

		CPA		PF	
	stage	P	S	P	S
1	1	-.63**	-.64**	-.25**	-.26**
consequences	2	-.69**	-.67**	-.36**	-.37**
	3	-.66**	-.66**	-.35**	-.36**
2	1	-.25**	-.17**	-.19**	-.18**
timeline	2	-.26**	-.24**	-.15*	-.10
	3	-.33**	-.32**	-.18**	-.18**
3	1	.30**	.29**	.12	.11
personal	2	.25**	.23**	.16*	.18**
control	3	.30**	.29**	.21**	.19**
4	1	.17**	.16*	.17**	.16*
treatment	2	.21**	.21**	.19**	.18**
control	3	.22**	.23**	.17*	.14
5	1	-.33**	-.37**	-.15*	-.14*
identity	2	-.45**	-.46**	-.23**	-.24**
	3	-.43**	-.44**	-.13	-.14*
6	1	-.63**	-.64**	-.38**	-.37**
concern	2	-.64**	-.64**	-.37**	-.36**
	3	-.67**	-.69**	-.46**	-.44**
7	1	.07	.00	.07	.10
understanding	2	.13	.09	.16*	.15*
	3	.15*	.11	.12	.13
8	1	-.63**	-.66**	-.55**	-.56**
emotional	2	-.68**	-.67**	-.56**	-.54**
response	3	-.67**	-.68**	-.54**	-.56**

Table 7.28: Correlations between acceptance and IPs (P - Pearson's r, and S - Spearman's ρ)

Causal beliefs were unrelated to acceptance, with the exception of the broader category of external attributions, which was consistently associated with both lower CPA and PF in all three stages ($t(252) = 2.6$, $p = .01$, $t(220) = 3.1$, $p = .002$, and $t(205) = 3.6$, $p < .001$, for CPA; $t(252) = 3.0$, $p = .003$, $t(220) = 3.0$, $p = .003$, and $t(205) = 2.5$, $p = .01$, for PF in the three stages[36]).

Illness perceptions and emotion

'Non-adaptors' (as identified based on the cluster analysis of the BIPQ items) consistently reported significantly more frequent negative emotions and use of internal dysfunctional ERSs and less frequent positive emotions compared with adaptors. The differences between the three groups regarding external dysfunctional and both functional ERS reached statistical significance (at an uncorrected $\alpha = .05$) in only

[36]The statistical significance of these tests depends on the decision regarding the most suitable correction for multiple comparison; considering an α level of .001 would take into consideration the number of tests performed, but would be overly conservative given that the tests are not independent and the three stages can be considered replications of the same test.

	Stage	t-test	Mean values		Pearson's r
			Adaptors	Non-adaptors	
Anger	1	$t(187) = 3.8$, $p < .001$	4.2	4.9	-.27**
	2	$t(82) = 6.8$, $p < .001$	3.5	4.8	-.45**
	3	$t(142) = 3.8$, $p < .001$	4.0	4.9	-.30**
Sadness	1	$t(187) = 4.3$, $p < .001$	3.4	4.4	-.30**
	2	$t(165) = 7.7$, $p < .001$	2.5	4.2	-.51**
	3	$t(142) = 4.2$, $p < .001$	3.3	4.3	-.33**
shame	1	$t(94.6) = 2.4$, $p = .02$	2.8	3.4	-.19**
	2	$t(101) = 3.6$, $p = .003$	2.3	3.1	-.23**
	3	$t(142) = 3.2$, $p = .002$	2.7	3.6	-.26**
Anxiety	1	$t(187) = 4.1$, $p < .001$	4.4	5.2	-.29**
	2	$t(165) = 4.9$, $p < .001$	4.0	5.0	-.36**
	3	$t(142) = 5.0$, $p < .001$	4.4	5.5	-.39**
Joy	1	$t(187) = 3.4$, $p = .001$	5.0	4.5	.24**
	2	$t(100.8) = 7.2$, $p < .001$	5.6	4.3	.43**
	3	$t(142) = 3.5$, $p = .001$	4.9	4.2	.28**
ID	1	$t(123.6) = 3.8$, $p = .001$	2.2	2.6	-.26**
	2	$t(194) = 4.6$, $p < .001$	2.0	2.5	-.31**
	3	$t(83.4) = 4.7$, $p = .001$	2.1	2.7	-.37**
ED	1	$t(118.7) = 1.5$, $p = .14$	1.5	1.6	-.11
	2	$t(127.0) = 3.5$, $p = .001$	1.3	1.6	-.20**
	3	$t(87.9) = 2.8$, $p = .007$	1.4	1.7	-.22**
IF	1	$t(226) = 2.1$, $p = .04$	3.0	2.8	.14*
	2	$t(194) = 5.3$, $p < .001$	3.3	2.8	.36**
	3	$t(167) = 1.9$, $p = .07$	3.0	2.8	.14
EF	1	$t(226) = 1.7$, $p = .09$	2.7	2.5	.11
	2	$t(194) = 4.9$, $p < .001$	3.0	2.5	.33**
	3	$t(167) = 4.0$, $p < .001$	2.8	2.3	.30**

Table 7.29: Differences in emotion between the IP clusters

two stages; nevertheless the differences between the mean values were in the expected directions, albeit small (see Table 7.29).

As the two clusters are unlikely to reflect the complexity of the relationships between illness perceptions and emotional life due to heterogeneous structure of IPs as measured by BIPQ, the connections between the two domains were further explored via correlational analyses. As it can be seen in Table 7.30, only perceptions of emotional response are consistently associated with all five emotions. Perceptions of consequences and concern were less strongly associated with shame and joy/happiness. Perceptions of illness identity and personal and treatment control were less associated with all emotions, showing particular lack of association with reports of shame (and joy, only for identity). Perceptions of timeline and understanding were largely unrelated to emotions. Among the five emotions, shame reports were overall the least connected to IPs.

The reported use of ERSs was not consistently related to IPs, except perceptions of emotional response (Table 7.31). Perceptions of consequences and concern were particularly unrelated to external dysfunctional ERSs, while showing small to medium correlations to the other ERSs. Perceptions of understanding and personal and treatment control were largely unrelated to external dysfunctional and internal functional ERSs, while showing higher (though inconsistent) associations with the internal dysfunctional and external functional ERSs. Perceptions of timeline and illness identity were largely unrelated to ERSs. As it can be seen, the associations with ERSs mirror closely the associations to discrete emotions, with the exception of understanding (which appears more related to ERSs) and identity (which seems connected only to emotion reports).

Causal beliefs were not consistently related to emotional variables, with the exception of the broader category of external attributions, which was consistently associated with more frequent anger and sadness and use of internal dysfunctional ERSs, and less frequent happiness and external functional ERSs, in all three stages (see Table 7.32[37]).

7.7 Exploratory data analysis

7.7.1 Health status and psychosocial variables

The health status indicators (HSIs) considered for this analysis were pain intensity (S-PRI, A-PRI, T-PRI, VAS and PPI), pain disability (RM-SIP), health care use (medication use and GP visits) and work status. Correlation analyses and multiple regression models (MRMs) were performed with all HSIs for each domain separately. First, analyses were performed to explore the relation between contextual factors and health status, followed by the separate examination of the role of acceptance, emotions and illness perceptions. Finally, a comparative analysis was performed by fitting MRMs including all four types of predictors for T-PRI and RM-SIP scores.

Although the cross-sectional design used in these analyses does not allow a clear statement of unidirectionality, the health status indicators were selected as dependent variables since this research aims at predicting and improving health status, and the comparison between the psychological adjustment variables is based in this

[37] As with the previous analysis related to acceptance, the adequacy of a conservative correction for the significance level is questionable. Therefore these associations were reported based on the fact that they were the only consistent pattern across the three stages at a typical $\alpha = .05$, rather than due to its statistical significance based on an adjusted α level.

		Anger		Sad		Shame		Anxiety		Joy	
	st.	P	S	P	S	P	S	P	S	P	S
1	1	.33**	.35**	.31**	.33**	.12	.09	.24**	.27**	-.16*	-.14*
conse-	2	.38**	.38**	.38**	.38**	.17*	.14	.34**	.37**	-.29**	-.30**
quences	3	.31**	.32**	.37**	.38**	.23**	.22**	.29**	.32**	-.36**	-.36**
2	1	.21**	.19**	.20**	.18**	.10	.11	.12	.14*	-.07	-.04
timeline	2	.14	.11	.15*	.17*	-.06	-.06	.07	.10	-.04	-.07
	3	.19**	.15*	.14	.15*	.03	-.00	.10	.09	-.13	-.10
3	1	-.23**	-.24**	-.23**	-.22**	-.17*	-.14*	-.21**	-.21**	.23**	.23**
personal	2	-.20**	-.18*	-.22**	-.23**	-.12	-.15*	-.16*	-.14	.20**	.23**
control	3	-.13	-.13	-.22**	-.23**	-.19**	-.20**	-.23**	-.22**	.18*	.16*
4	1	-.18**	-.20**	-.20**	-.21**	-.11	-.10	-.17*	-.18**	.18**	.16*
treatment	2	-.21**	-.21**	-.23**	-.24**	-.13	-.12	-.21**	-.22**	.29**	.28**
control	3	-.12	-.11	-.09	-.10	-.10	-.07	-.15*	-.14	.22**	.21**
5	1	.21**	.24**	.19**	.21**	.11	.05	.19**	.24**	.02	.02
identity	2	.27**	.27**	.24**	.24**	.16*	.13	.26**	.28**	-.05	-.03
	3	.15*	.18*	.18*	.18*	.13	.12	.19**	.21**	-.09	-.11
6	1	.37**	.38**	.37**	.38**	.23**	.19**	.33**	.34**	-.16*	-.13
concern	2	.44**	.42**	.43**	.42**	.28**	.26**	.37**	.39**	-.27**	-.25**
	3	.40**	.40**	.46**	.46**	.32**	.30**	.42**	.42**	-.37**	-.34**
7	1	-.07	-.07	-.06	-.06	-.09	-.12	-.06	-.05	.09	.11
under-	2	-.15*	-.12	-.15*	-.13	-.18*	-.18*	-.11	-.08	.08	.10
standing	3	-.14	-.12	-.16*	-.19*	-.20**	-.22**	-.18*	-.13	.19*	.20**
8	1	.62**	.63**	.59**	.61**	.43**	.42**	.55**	.55**	-.41**	-.42**
emotional	2	.60**	.59**	.58**	.60**	.36**	.35**	.51**	.54**	-.52**	-.51**
response	3	.55**	.57**	.61**	.63**	.44**	.43**	.52**	.55**	-.50**	-.50**

Table 7.30: Correlations between emotions and IPs (P - Pearson's r, and S - Spearman's ρ)

		ID		ED		IF		EF	
	st.	P	S	P	S	P	S	P	S
1	1	.19**	.18**	.09	.06	-.14*	-.12*	-.12	-.12
consequences	2	.25**	.24**	.16*	.15*	-.22**	-.22**	-.28**	-.29**
	3	.24**	.23**	.14*	.11	-.23**	-.25**	-.31**	-.32**
2	1	.15*	.12	.04	.06	-.05	-.06	-.16*	-.13*
timeline	2	.07	.05	-.05	-.06	-.09	-.08	-.14*	-.10
	3	.11	.06	.09	.07	-.19**	-.21**	-.20**	-.21**
3	1	-.19**	-.18**	-.11	-.11	.09	.07	.11	.12
personal	2	-.13	-.16*	-.13	-.15*	.08	.07	.20**	.20**
control	3	-.26**	-.25**	-.03	-.04	.10	.09	.25**	.22**
4	1	-.20**	-.19**	-.07	-.07	.09	.11	.16**	.18**
treatment	2	-.11	-.13	-.09	-.09	.19**	.14*	.23**	.22**
control	3	-.21**	-.22**	-.16*	-.20**	.16*	.19**	.23**	.25**
5	1	.13*	.14*	.12	.07	-.02	-.03	-.02	.00
identity	2	.18**	.18**	.11	.06	-.06	-.06	-.07	-.06
	3	.09	.07	.04	.01	-.15*	-.19**	-.16*	-.17*
6	1	.30**	.29**	.11	.08	-.15*	-.16*	-.12	-.14*
concern	2	.35**	.34**	.13*	.11	-.20**	-.19**	-.21**	-.21**
	3	.33**	.31**	.18*	.16*	-.19**	-.22**	-.25**	-.26**
7	1	-.11	-.13*	-.10	-.09	.09	.11	.18**	.18**
under-	2	-.19**	-.22**	-.13	-.10	.12	.08	.23**	.22**
standing	3	-.18**	-.24**	-.18**	-.12	.02	.04	.16*	.14*
8	1	.48**	.47**	.32**	.33**	-.38**	-.38**	-.25**	-.29**
emotional	2	.49**	.50**	.34**	.36**	-.35**	-.36**	-.40**	-.41**
response	3	.48**	.50**	.34**	.35**	-.26**	-.28**	-.37**	-.39**

Table 7.31: Correlations between ERSs and IPs (P - Pearson's r, and S - Spearman's ρ)

	st.	t-test	Mean values		Pearson's r
			No ext. causes	External causes	
Anger	1	$t(210) = 3.5$, $p < .001$	4.1	4.7	.24**
	2	$t(188) = 3.9$, $p < .001$	4.0	4.6	.27**
	3	$t(176) = 2.5$, $p = .01$	3.9	4.4	.19*
Sadness	1	$t(210) = 3.0$, $p = .003$	3.3	3.9	.20**
	2	$t(188) = 3.4$, $p = .001$	3.2	3.9	.24**
	3	$t(176) = 2.4$, $p = .02$	3.1	3.6	.18*
ID	1	$t(252) = 2.5$, $p = .01$	2.2	2.4	.16*
	2	$t(220) = 2.5$, $p = .01$	2.2	2.4	.17*
	3	$t(205) = 2.3$, $p = .02$	2.1	2.4	.16*
Happy	1	$t(210) = 2.3$, $p = .02$	5.0	4.6	-.16*
	2	$t(188) = 2.6$, $p = .01$	5.0	4.5	-.18*
	3	$t(176) = 2.6$, $p = .01$	5.1	4.6	-.19*
EF	1	$t(252) = 2.1$, $p = .04$	2.7	2.6	-.13*
	2	$t(220) = 3.4$, $p = .001$	2.8	2.5	-.22**
	3	$t(205) = 2.7$, $p = .007$	2.8	2.6	-.19**

Table 7.32: Relations between emotion variables and external causal attributions

analysis on their individual and common contribution to (statistical) prediction of health status. It is important however to acknowledge that these relationships are likely bidirectional, and refer rather to pain reporting than to the actual experience of pain and pain behaviours.

Contextual factors and health status

Several demographic and medical history variables were considered in this analysis: gender, age, education, annual income, marital status, pain location, pain spread, pain duration, age at pain onset, previous and current pain treatment (antidepressants, sleeping tablets, surgery, physiotherapy, pain management programmes, complementary therapies), comorbidity (and separately presence of depression and osteoarthritis), negative and positive life events (total scores and selected events, such as recent injuries or financial problems)[38]. Correlational analyses indicated[39] that each health status indicator was related to a slightly different set of contextual factors.

Pain-related disability was associated with gender (males reporting more disability, $r = .14 - .17$), annual income level ($r = .18 - .29$) and recent financial problems ($r = .20 - .27$), recent total number of negative events ($r = .12 - .24$), pain spread (for both NHS and SO subsamples, both as number of pain locations, $r = .25 - .45$ and number of broader pain areas, $r = .27 - .43$[40]), comorbidity ($r = .25 - .32$; and separately depression, $r = .24 - .27$, and arthritis, $r = .15 - .21$), previous and current intake of antidepressants ($r = .15 - .22$) and sleeping tablets ($r = .17 - .33$), and previous surgery ($r = .20 - .24$). Work status was associated with education ($r = .17 - .20$), age ($r = .15 - .20$), annual income ($r = .25 - .32$), occurrence of positive events ($r = .18 - .23$, in particular better financial status, $r = .18 - .23$), pain duration ($r = .24 - .27$), previous and current intake of sleeping tablets ($r = .17 - .23$), current attendance to pain management programmes ($r = .15 - .22$), and comorbidity ($r = .24 - .29$, and depression in particular, $r = .22 - .20$).

[38]Contextual factors were characterised by non-significant or low significant inter-correlations (not reported here), therefore the overlap between them was minimal in this sample.

[39]Variables were selected only if significant associations were found at an $\alpha = .05$ in all three stages. Although such an α level may be considered extremely liberal given the number of tests performed, it was seen as adequate given the exploratory nature of the analysis and the equally important concern of type II error; the criterion of consistency between the three stages was applied as a measure against type I error. Exceptions to this criterion were considered only when consistent associations were present at trend level (although not all reaching significance), and are reported where applied.

[40]Subsample size varied between 188 for the NHS subsample in stage 1, and 47 for the SO subsample in stage 3.

Medication use was related to the number of recent negative events ($r = .16 - .26$, in particular financial problems, $r = .12 - .22$), previous and current use of sleeping tablets ($r = .24 - .35$), previous attendance to physiotherapy ($r = .17 - .23$), current use of antidepressants ($r = .19 - .27$) and complementary therapies ($r = .16 - .22$), depression ($r = .14 - .16$) and arthritis ($r = .14 - .19$). The frequency of GP visits (low versus high) was related to a recent serious injury or illness ($r = .14 - .33$), financial problems ($r = .14 - .22$), current intake of antidepressants ($r = .14 - .22$) and sleeping pills ($r = .14 - .18$) and depression ($r = .17 - .24$).

All pain intensity indicators (S-PRI, A-PRI, T-PRI, VAS and PPI) were similarly related to previous and present intake of antidepressants ($r = .09 - .31$) and sleeping tablets ($r = .11 - .37$) and to high comorbidity, especially the presence of depression[41]. In addition, education status consistently reached significance in all three stages only for VAS ($r = -.14 - -.18$), previous surgical treatment was related only to S-PRI, T-PRI and VAS, age at pain onset was associated only with S-PRI and T-PRI, while a recent injury or illness was consistently reflected only in S-PRI.

Contextual factors were also associated selectively with measures of acceptance, emotions and illness perceptions. Thus, lower chronic pain acceptance and psychological flexibility were associated with male gender ($r = .10 - .23$), lower education ($r = .07 - .24$) and income ($r = .11 - .27$, no recent improvement of financial status, $r = .09 - .23$) higher comorbidity ($r = .12 - .23$, especially presence of depression, $r = .20 - .47$), previous and current intake of antidepressants ($r = .12 - .30$) and sleeping pills ($r = .11 - .30$), previous surgery ($r = .09 - .24$), and not having used complementary therapies ($r = .09 - .22$). In general, both PF and CPA (and AE and PW subscales) showed similar consistent associations, with very few exceptions (some correlations followed the general trend, but failed to reach significance). However, three notable exceptions were present: PW was not associated to previous intake of antidepressants and sleeping tablets, nor with recent improvements in financial status.

More frequent negative emotions and less frequent positive emotions were associated with lower annual income ($r = .16 - .22$), higher number of recent negative events ($r = .14 - .29$), higher comorbidity ($r = .10 - .26$, especially the presence of

[41] With very few exceptions, i.e. some correlations not reaching significance in one of the stages for some indices, although values were following the general trend.

depression, $r = .35 - .53$), and increased previous and current intake of antidepressants ($r = .11 - .38$) and sleeping tablets ($r = .08 - .37$)[42]. In addition, increased sadness was also associated with lower education ($r = .20 - .24$), while females reported higher scores for happiness ($r = .17 - .24$).

Increased use of dysfunctional ERSs and decreased use of functional ERSs was associated with presence of depression ($r = .12 - .44$) and increased previous and current intake of antidepressants ($r = .10 - .31$) (again, with few exceptions). Dysfunctional ERSs were used more by participants who had experienced more negative events recently ($r = .18 - .26$), especially financial difficulties ($r = .16 - .29$). In addition, internal dysfunctional ERSs were associated with younger age ($r = .18 - .30$), and with previous intake of sleeping tablets ($r = .17 - .20$). Functional ERSs were used more by females ($r = .17 - .35$), people with higher education ($r = .13 - .24$) and higher income ($r = .13 - .30$)[43].

Illness perceptions of timeline, personal and treatment control and understanding were not consistently related to any contextual variables. Perceptions of more consequences and emotional impact were also associated with lower education ($r = .12 - .19$), lower income ($r = .18 - .24$), comorbidity ($r = .17 - .26$, especially presence of depression, $r = .20 - .40$), previous intake of sleeping tablets ($r = .15 - .27$), and current intake of antidepressants ($r = .15 - .28$). Perceptions of more consequences were also related to previous surgery ($r = .22 - .26$) and current intake of sleeping tablets ($r = .13 - .28$). Perceptions of illness identity (more symptoms) were consistently related only to comorbidity ($r = .15 - .23$) and previous intake of sleeping tablets ($r = .17 - .20$), while perceptions of concern were associated with previous surgery ($r = .14 - .23$) and presence of depression ($r = .20 - .32$). Causal attributions were also related to selected contextual variables. Respondents that considered their own behaviours as causes reported longer pain duration ($r = .14$), and previous use of complementary therapies ($r = .13$). Considering poorcare as a cause of chronic pain was associated with higher education ($r = .12$), previous surgery ($r = .27$), lower comorbidity ($r = -.14$, especially not reporting arthritis as a comorbid condition, $r = -.14$). Accident as a cause was reported more by men ($r = .14$), with higher education ($r = .13$), but lower income ($r = -.17$), who have previously used physiotherapy ($r = .14$) and pain management ($r = .14$). Work conditions was reported as a cause of chronic pain by people with

[42]Happiness had associations of the opposite sign. Again, only a few estimates did not reach significance in all stages, while following the same trend.

[43]These associations are partly replicated at the level of selected items - not reported here for brevity.

lower education ($r = -.15$), and low income ($r = -.19$), with no previous surgical treatment ($r = -.16$). Physiological attributions were reported by people with higher income ($r = .13$), while comorbid conditions were given as an explanation more by older ($r = .18$) women ($r = .19$) reporting higher comorbidity scores ($r = .23$, especially arthritis, $r = .32$). In general, reporting psychological causes was associated with previous use of complementary therapies ($r = .19$); risk factors with previous surgery ($r = .17$); external factors with male gender ($r = .13$), low income ($r = -.25$), previous physiotherapy ($r = .20$), psychological therapies ($r = .14$), and pain management ($r = .15$); medical attributions with longer pain duration ($r = .14$), lack of depression ($r = -.13$), older age ($r = .20$), and later pain onset ($r = .14$).

It is important to note that most associations with contextual factors are small to medium, and therefore each explains only a limited amount of variance in the health status indicators (and also in the psychological factors). Multiple regressions and logistic regressions were next performed to identify the individual role of each contextual variable and the total predictive power of these factors in predicting health status, as a preliminary step for comparative analyses of the role of psychological factors. To this end, contextual factors that were overlapping conceptually with psychological factors, such as previous and current intake of antidepressants and sleeping tablets or participation in pain management programmes and presence of comorbid depression, were excluded from the analysis[44]. Also, if two contextual variables computed from the same data, only one was selected[45]. Hierarchical (sequential) regression was used in order to identify the incremental contribution of each factor (Tabachnick and Fidell, 2001). Only factors that had significant relationships with the HSI in all three stages were selected for initial models and the consistency criterion was applied for selecting factors that remained significant in the final modeling sequence.

Pain disability (RM-SIP) was regressed on gender, annual income, recent negative events, spread of pain, comorbidity and previous surgery. The results are presented

[44]It was considered that the diagnosis of depression and the prescription of such treatment was partly based on assessment of psychological adjustment, therefore controlling for such variables would artificially diminish the contribution of psychological factors to health status.

[45]For example, the total number of pain locations was selected as indicator of pain spread, but not the total number of body areas affected by pain, as it was computed by grouping pain locations into broader categories.

in Table 7.33[46]. It is important to note that pain disability was related to several demographic and medical variables, which explained in total a considerable proportion of the variance (19–23%). Some of these relationships might reflect bidirectional relationships from a causal point of view, for example in the case of surgical interventions, for which the decision might have been taken based on the level of disability and in some cases might have increased disability subsequently. A minor suppression effect of comorbidity and pain spread on the relationship between gender and disability may be noticed.

Medication use was regressed on presence of arthritis, previous use of complementary therapies, previous physiotherapy and recent negative events, only the last two remaining significant in the final prediction equation in all three stages (Table 7.33). The number of GP visits was not consistently predicted by neither reports of recent injuries or illnesses nor reports of recent financial problems[47]. Work status was consistently predicted only by level of comorbidity (education, age, pain duration and recent positive events did not remain significant when comorbidity was included in the model), however the contribution of this predictor was minor (Table 7.34).

Among the pain intensity indicators, S-PRI was predicted by age of pain onset and previous surgery, while A-PRI was predicted only by comorbidity. T-PRI scores were only related to comorbidity and age at pain onset and VAS by education level and previous surgery. In contrast to pain disability, these factors explained only a small proportion of the variance in pain intensity indicators (Table 7.35). PPI was not consistently predicted by any contextual factor.

Acceptance and health status

Correlation and multiple regression analyses provided support for the hypothesised relationship between increased CPA and PF and better health status. CPA (and AE and PW) and PF correlated significantly in the three stages with pain-related disability (RM-SIP), vocational status (full/part-time working versus not working because of pain), pain intensity (S-PRI, A-PRI, T-PRI, VAS and PPI) and health

[46]With the exception of annual income and negative events which did not reach significance in the first stage when medical factors were introduced in the model, all variables remained significant in all stages of the model.

[47]Despite significant but trivial correlations, none improved the percentage of correct predictions of high versus low number of GP visits in all stages.

Model RM-SIP		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	(Constant)	16.24	16.00	15.94	1.38	1.56	1.66									
	Gender	-1.75	-2.12	-2.38	.79	.89	.94	-.14*	-.16*	-.18*	.02 (.02)	.03 (.02)	.03 (.03)	.02*	.03*	.03*
2	(Constant)	14.68	14.03	13.81	1.37	1.51	1.63									
	Gender	-2.16	-2.56	-2.67	.76	.84	.90	-.17**	-.19**	-.20**						
	Spread of pain	.50	.60	.57	.10	.11	.12	.29**	.34**	.32**	.10 (.10)	.14 (.13)	.13 (.12)	.11**	.09**	.10**
3	(Constant)	11.68	11.10	9.67	1.59	1.79	1.87									
	Gender	-2.32	-2.63	-2.82	.74	.83	.86	-.18**	-.20**	-.21**						
	Spread of pain	.39	.48	.41	.11	.12	.12	.23**	.27**	.23**						
	Comorbidity	2.48	2.39	3.40	.71	.81	.84	.21**	.20**	.28**	.14 (.13)	.17 (.16)	.20 (.19)	.03**	.02*	.07**
4	(Constant)	9.47	9.13	7.61	1.61	1.83	1.92									
	Gender	-1.81	-2.14	-2.35	.726	.82	.85	-.14*	-.16**	-.17**						
	Spread of pain	.42	.49	.43	.10	.12	.12	.24**	.27**	.24**						
	Comorbidity	2.48	2.40	3.50	.69	.79	.82	.21**	.20**	.28**						
	Previous surgery	3.06	2.80	2.79	.67	.77	.81	.26**	.22**	.22**	.21 (.19)	.22 (.21)	.25 (.23)	.05**	.05**	.05**
	Medication use															
1	(Constant)	1.88	2.13	2.08	.17	.19	.19									
	previous physio	.76	.58	.56	.20	.22	.22	.23**	.18**	.17*	.05 (.05)	.03 (.03)	.03 (.03)	.05**	.03**	.03*
2	(Constant)	1.67	1.88	1.82	.19	.20	.20									
	previous physio	.71	.46	.53	.20	.21	.22	.22**	.14*	.16*						
	negative events	.15	.26	.20	.05	.06	.06	.17**	.26**	.23**	.08 (.08)	.10 (.09)	.08 (.07)	.03**	.07**	.05**

Table 7.33: Contextual variables as predictors of disability and medication use

	st.	B	SE B	95% CI for exp b lower	exp b	upper	Cox & Snell R^2	Nagelkerke R^2	% correct classif.
(Constant)	1	-1.27**	.44		.28				
	2	-1.48**	.48		.23				
	3	-1.56**	.49		.21				
Comorbidity	1	.97**	.28	1.51	2.64	4.60	.06	.07	62
	2	1.16**	.31	1.73	3.18	5.86	.08	.10	64
	3	1.20**	.32	1.77	3.32	6.24	.08	.10	64

Table 7.34: Comorbidity as predictor of work status

care utilisation (number of GP visits and number of different types of medication, see Table 7.36[48]).

CPA and PF were included in MRMs controlling for the relevant contextual factors, in order to identify their unique contribution in predicting HSIs (since AE and PW correlations to most HSIs were similar, only CPA was considered in these analyses, for reasons of brevity). The contextual factors were introduced first in a single stage in sequential MRMs, followed by CPA and then PF, in separate stages. As CPA showed higher correlations to HSIs (naturally due to the health-focused wording of the items) and the distinction between PF and CPA was questioned based on previous analyses, this modeling strategy attempted to investigate whether PF accounts for a significant share of the variance in HSIs in addition to the variance explained by CPA.

The results of the MRMs are presented in Tables 7.37, 7.38 and 7.39[49]. In all models, adding CPA significantly improved prediction of HSIs, the effect size being most notable in the case of disability and work status. Some contextual factors (such as comorbidity and previous surgery) did not remain significant when CPA was added, indicating a probable mediation effect of CPA. Also, PF did not consistently improve prediction in any of the models (therefore, the results are not presented), suggesting either a mediation mechanism or the lack of a conceptual distinction between them (as suggested by previous analyses).

[48]Pearson correlations are presented. Non-parametric correlations resulted in similar estimates and were not reported.

[49]The first stages are obviously identical with the final stages of the analyses reported in the previous section, therefore they are not repeated here.

Model S-PRI		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	(Constant)	19.12	19.45	19.69	1.52	1.72	1.86									
	age pain onset	-.13	-.13	-.13	.04	.04	.05	-.21**	-.20**	-.20**	.05 (.04)	.04 (.04)	.04 (.04)	.05**	.04**	.04**
2	(Constant)	18.24	18.37	18.63	1.55	1.73	1.89									
	age pain onset	-.13	-.13	-.13	.04	.04	.05	-.21**	-.21**	-.20**						
	previous surgery	2.34	3.12	2.68	.92	1.00	1.08	.15*	.20**	.17*	.07 (.06)	.08 (.07)	.08 (.07)	.02*	.04**	.04**
	A-PRI															
1	(Constant)	2.70	2.21	2.17	.67	.69	.71									
	comorbidity	1.39	1.73	1.60	.42	.43	.45	.20**	.26**	.24**	.04 (.04)	.07 (.07)	.06 (.06)**	.04**	.07**	.06**
	T-PRI															
1	(Constant)	14.43	12.67	10.58	2.01	2.13	2.21									
	comorbidity	2.89	4.52	5.60	1.25	1.34	1.39	.14*	.22**	.27**	.02 (.02)	.05 (.05)	.07 (.07)	.02*	.04**	.07**
2	(Constant)	20.62	18.53	17.79	2.81	3.07	3.22									
	comorbidity	2.85	4.48	5.50	1.23	1.33	1.37	.14*	.22**	.27**						
	age - pain onset	-.16	-.15	-.18	.05	.06	.06	-.19**	-.17**	-.20**	.05 (.05)	.08 (.07)	.11 (.11)	.04**	.03**	.04**
3	(Constant)	19.13	16.99	15.93	2.83	3.06	3.24									
	comorbidity	2.96	4.58	5.72	1.22	1.30	1.35	.14**	.22**	.28**						
	age - pain onset	-.16	-.15	-.18	.05	.06	.06	-.19**	-.17**	-.20**						
	previous surgery	3.51	3.99	3.84	1.25	1.34	1.40	.17**	.19**	.18**	.08 (.07)	.11 (.10)	.15 (.13)	.03**	.04**	.03**
	VAS															
1	(Constant)	109.92	110.57	107.68	6.59	6.69	7.04									
	education	-9.02	-10.95	-9.71	4.08	4.13	4.37	-.14*	-.18**	-.15*	.02 (.02)	.03 (.03)	.02 (.02)	.02*	.03**	.02*
2	(Constant)	106.29	106.26	101.51	6.76	6.80	7.11									
	education	-8.94	-10.87	-9.22	4.05	4.08	4.27	-.13*	-.18**	-.15*						
	previous surgery	9.04	11.07	14.89	4.14	4.19	4.43	.17*	.17**	.23**	.04 (.03)	.06 (.05)	.08 (.07)	.02*	.03**	.05**

Table 7.35: Contextual variables as predictors of perceived pain intensity

	st	PF	CPA	AE	PW
Disability	1	-.30**	-.54**	-.48**	-.42**
	2	-.38**	-.58**	-.57**	-.46**
	3	-.35**	-.57**	-.48**	-.50**
Work status	1	-.15*	-.40**	-.45**	-.21**
	2	-.20**	-.46**	-.49**	-.32**
	3	-.20**	-.48**	-.49**	-.33**
S-PRI	1	-.21**	-.32**	-.27**	-.26**
	2	-.17**	-.31**	-.27**	-.28**
	3	-.31**	-.42**	-.32**	-.40**
A-PRI	1	-.32**	-.42**	-.34**	-.38**
	2	-.38**	-.48**	-.41**	-.46**
	3	-.43**	-.60**	-.52**	-.51**
T-PRI	1	-.26**	-.37**	-.31**	-.32**
	2	-.26**	-.38**	-.34**	-.36**
	3	-.36**	-.50**	-.40**	-.46**
VAS	1	-.28**	-.40**	-.34**	-.34**
	2	-.30**	-.43**	-.40**	-.36**
	3	-.25**	-.46**	-.36**	-.45**
PPI	1	-.24**	-.35**	-.31**	-.28**
	2	-.26**	-.45**	-.41**	-.39**
	3	-.28**	-.49**	-.40**	-.45**
Medication use	1	-.12	-.17**	-.18**	-.11
	2	-.17*	-.18**	-.15*	-.18**
	3	-.20**	-.21**	-.17*	-.19**
GP visits	1	-.22**	-.27**	-.25**	-.20**
	2	-.32**	-.33**	-.32**	-.27**
	3	-.18*	-.28**	-.29**	-.18**

Table 7.36: Correlations between chronic pain acceptance and HSIs

Emotions and health status

Better health status was associated in general with less frequent negative emotions and more frequent happiness/joy, as presented in Table 7.40[50]. However it can be noted that work status was not consistently associated with shame and anxiety, while the relation between medication use and anger, shame and joy failed to reach significance in all stages (although parameters followed the main trend). Also, happiness/joy was not consistently related with sensory pain, VAS, PPI and GP visits.

In contrast, ERSs were consistently related only to disability and affective pain intensity. Only internal dysfunctional ERSs showed consistent associations with all HSIs[51]. External dysfunctional ERSs were also related to higher S-PRI and T-PRI,

[50]Pearson correlations are presented. Non-parametric correlations resulted in similar estimates and were not reported.

[51]Except its correlation to work status failing to reach significance in stage 1.

Model RM-SIP		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	17.49	18.69	16.79	1.63	1.83	1.92									
	Gender	-.60	-.87	-1.09	.64	.70	.74	-.05	-.07	-.08						
	Spread of pain	.45	.52	.44	.09	.10	.10	.26**	.29**	.25**						
	Comorbidity	1.10	.72	2.00	.61	.68	.71	.09	.06	.16**						
	Previous surgery	2.26	1.41	1.86	.59	.66	.69	.19**	.11*	.14**						
	CPA	-2.97	-3.19	-3.06	.31	.33	.34	-.48**	-.53**	-.50**	.41 (.40)	.46 (.44)	.46 (.45)	.21**	.24**	.22**
	Medication															
1	...															
2	(Constant)	2.33	2.49	2.60	.29	.30	.33									
	previous physio	.74	.49	.54	.19	.21	.21	.22**	.15*	.17*						
	negative events	.15	.24	.18	.05	.06	.06	.17**	.25**	.20**						
	CPA	-.25	-.22	-.27	.09	.09	.09	-.17**	-.17**	-.20**	.11 (.10)	.13 (.11)	.12 (.11)	.03**	.03**	.04**

Table 7.37: Acceptance as predictor of disability and medication use

Model	work status	st.	B	SE B	95% CI for exp b lower	exp b	upper	Cox & Snell R^2	Nagelkerke R^2	% correct classif.	χ^2 diff.
1	...										
2	(Constant)	1	1.62*	.73		5.03					
		2	1.98*	.81		7.26					
		3	.95**	.36		11.16					
	Comorbidity	1	.74*	.31	1.15	2.09	3.81				
		2	.86*	.35	1.21	2.37	4.68				
		3	.95**	.36	1.27	2.59	5.28				
	CPA	1	-.98**	.20	.25	.38	.55	.18	.24	67	30(1), $p < .001$
		2	-1.10**	.21	.22	.33	.50	.24	.33	70	36.39(1), $p < .001$
		3	-1.26**	.23	.18	.29	.45	.28	.37	73	41.54(1), $p < .001$
	GP visits										
1	(Constant)	1	1.88**	.42		6.56					
		2	2.04**	.46		7.69					
		3	1.56**	.47		4.74					
	CPA	1	-.62**	.15	.40	.54	.72	.07	.10	64	–
		2	-.74**	.16	.35	.48	.65	.11	.15	62	–
		3	-.61**	.16	.40	.54	.74	.08	.10	63	–

Table 7.38: Acceptance as predictor of work status and GP visits

Model T-PRI		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	31.57	29.00	34.13	3.41	3.75	3.71									
	comorbidity	1.53	3.06	3.59	1.17	1.27	1.22	.07	.15*	.17**						
	age - pain onset	-.16	-.15	-.20	.05	.05	.05	-.19**	-.17**	-.22**						
	previous surgery	2.24	2.31	2.13	1.20	1.32	1.25	.11	.11	.10						
	CPA	-3.70	-3.31	-4.73	.63	.66	.62	-.34**	-.32**	-.46**	.19 (.18)	.21 (.19)	.34 (.33)	.11**	.09**	.19**
	S-PRI															
1	...															
2	(Constant)	24.78	24.38	28.58	1.95	2.18	2.27									
	age pain onset	-.13	-.13	-.14	.04	.04	.04	-.21**	-.21**	-.22**						
	previous surgery	1.56	2.10	1.61	.89	.99	.99	.10	.14*	.10						
	CPA	-2.37	-2.06	-3.17	.46	.48	.48	-.30**	-.27**	-.42**	.15 (.15)	.15 (.14)	.24 (.22)	.09**	.07**	.17**
	A-PRI															
1	...															
2	(Constant)	7.39	7.27	8.74	.91	.92	.88									
	comorbidity	.84	1.08	.80	.39	.40	.38	.12*	.16**	.12*						
	CPA	-1.46	-1.47	-1.87	.21	.20	.19	-.40**	-.44**	-.57**	.19 (.19)	.25 (.25)	.37 (.36)	.15**	.19**	.31**
	VAS															
1	...															
2	(Constant)	137.41	137.10	132.31	7.89	7.95	7.87									
	education	-5.34	-7.26	-2.78	3.80	3.80	3.98	-.08	-.12	-.04						
	previous surgery	4.69	5.00	10.57	3.90	3.98	4.06	.07	.08	.16**						
	CPA	-13.28	-12.33	-13.62	2.04	1.96	2.01	-.38**	-.40**	-.43**	.17 (.16)	.21 (.20)	.25 (.24)	.14**	.15**	.17**
	PPI															
1	(Constant)	4.30	4.65	4.57	.20	.20	.20									
	CPA	-.43	-.52	-.54	.07	.07	.07	-.35**	-.45**	-.49**	.12 (.12)	.20 (.20)	.24 (.23)	.12**	.20**	.24**

Table 7.39: Acceptance as predictor of perception of pain intensity

	st	Anger	Sad	Shame	Anxiety	Joy
Disability	1	.37**	.34**	.27**	.32**	-.23**
	2	.44**	.46**	.36**	.32**	-.36**
	3	.41**	.44**	.38**	.36**	-.33**
Work status	1	.19*	.21**	.08	.11	-.20**
	2	.17*	.18*	.11	.16*	-.25**
	3	.22**	.25**	.20*	.22**	-.26**
S-PRI	1	.29**	.31**	.24**	.30**	-.13
	2	.30**	.33**	.30**	.31**	-.17*
	3	.39**	.43**	.42**	.36**	-.22**
A-PRI	1	.41**	.42**	.30**	.40**	-.21**
	2	.35**	.47**	.41**	.42**	-.24**
	3	.40**	.50**	.45**	.41**	-.37**
T-PRI	1	.35**	.37**	.28**	.36**	-.17*
	2	.33**	.40**	.36**	.37**	-.20**
	3	.42**	.47**	.46**	.40**	-.28**
VAS	1	.32**	.25**	.17*	.20**	-.13*
	2	.35**	.40**	.22**	.26**	-.10
	3	.37**	.41**	.31**	.22**	-.24**
PPI	1	.34**	.32**	.29**	.20**	-.11
	2	.33**	.36**	.21**	.26**	-.19**
	3	.34**	.34**	.25**	.27**	-.22**
Medication use	1	.14*	.19**	.13*	.24**	-.13
	2	.13	.19**	.11	.25**	-.17*
	3	.24**	.18*	.10	.17*	-.07
GP visits	1	.25**	.20**	.15*	.25**	-.16*
	2	.32**	.39**	.21**	.29**	-.25**
	3	.27**	.28**	.16*	.16*	-.11

Table 7.40: Correlations between emotions and HSIs

and internal functional ERSs to lower VAS[52]. Since these results might be due to the suboptimal psychometric properties of REQ in this sample, selected items were also examined. Only ID item 7 ('I dwell on my thoughts and feelings') and ED item 2 ('I take my feelings out on others verbally) were associated (in most stages) with the HSIs (except work status and medication use); all other items describing functional ERSs were not related to any HSI.

The emotion-related variables were next included in MRMs controlling for contextual variables, in order to identify their unique contribution to predicting HSIs. The basic emotions were introduced in a second stage together, followed by the relevant ERS variables; this strategy was used to identify any unique contribution of ERSs in addition to the one shared with basic emotion variables.

[52]Similar results were obtained with nonparametric correlations (Spearman's ρ), except the relations between ED and S-PRI and IF and VAS not reaching significance in one stage, and ID and work not being significant. Also, external functional ERSs were consistently related to GP visits in this analysis ($r = .14 - 19$). However, the small sizes of the correlations led to a similar conclusion of limited relationships between ERSs and some HSIs.

	st	ID	ED	IF	EF
Disability	1	.23**	.25**	-.17**	-.20**
	2	.31**	.31**	-.19**	-.34**
	3	.27**	.23**	-.20**	-.27**
Work status	1	.10	.08	-.02	-.09
	2	.18*	.07	-.04	-.18*
	3	.21**	.12	-.08	-.15*
S-PRI	1	.33**	.15*	.04	-.06
	2	.28**	.21**	-.06	-.14*
	3	.28**	.25**	-.16*	-.21**
A-PRI	1	.37**	.21**	-.18**	-.14*
	2	.37**	.15*	-.22**	-.23**
	3	.39**	.27**	-.28**	-.29**
T-PRI	1	.36**	.18**	-.04	-.09
	2	.33**	.19**	-.12	-.19**
	3	.33**	.27**	-.21**	-.25**
VAS	1	.16**	.13*	-.16**	-.15*
	2	.23**	.11	-.13*	-.10
	3	.20**	.16*	-.21**	-.20**
PPI	1	.23**	.13*	-.10	-.10
	2	.24**	.13	-.18**	-.16*
	3	.21**	.08	-.21**	-.16*
Medication	1	.14*	.06	.07	.01
	2	.28**	.18**	.10	-.02
	3	.18*	.18*	.05	.02
GP visits	1	.19**	.14*	-.14*	-.11
	2	.25**	.24**	-.27**	-.19**
	3	.17*	.03	-.09	-.11

Table 7.41: Correlations between ERSs and HSIs

The results of the MRMs are presented in Tables 7.42 to 7.46. In most models, adding the basic emotions frequencies scores significantly improved prediction of HSIs, similarly with CPA. A single exception was medication use, where emotion variables (sadness, anxiety and internal dysfunctional ERSs) did not consistently explain a significant amount of the variance in addition to amount explained by the number of negative recent events and previous physiotherapy.

The effect size was most notable in the case of A-PRI scores. Here too, some contextual factors (such as comorbidity and previous surgery) did not remain significant when emotion variables were added, possibly indicating a mediation effect. It is important to note that, although each emotion variable was significantly related to the HSIs when the effect of contextual variables was controlled for, their unique contributions when introduced simultaneously were mostly not significant, suggesting a common contribution.

None of the ERSs consistently improved prediction in any of the models (results not presented), suggesting a possible mediation mechanism or emotion frequencies

between ERSs and health status, or perhaps that the strategies relevant for health status in chronic pain are not included the REQ (these possibilities are further discussed in Chapter 9).

Illness perceptions and health status

Non-adaptors (as identified by cluster analysis of illness perceptions) had consistently lower health status indicators compared with adaptors in terms of pain intensity reported (S-PRI, A-PRI, T-PRI, VAS, and PPI), pain-related disability (RM-SIP) and frequency of GP visits. Differences in work status and medication use were only present in some of the stages (Table 7.47).

Given that the identified clusters were potentially masking different relationships between individual IPs and HSIs, these relationships were further explored via correlational analyses (Table 7.48[53]). Only perceptions of consequences and illness identity (symptoms) were consistently related to all indicators. Perceptions of concern and emotional impact were related to all HSIs except medication use. Perceptions of timeline were related only to pain disability and pain intensity, while perceptions of treatment control were related only to affective pain intensity and VAS. Personal control and understanding were not related to any HSI. None of the causal attributions categories were related consistently with disability, work status, HCUs or pain intensity indicators.

The relevant IPs were next included in MRMs controlling for contextual variables, in order to identify their unique contribution to predicting HSIs. The results are presented in Tables 7.49 to 7.53. Adding the relevant illness perception variables significantly improved prediction of all HSIs. The effect size was most notable in the case of pain disability, VAS and PPI scores. Some contextual factors (such as comorbidity and previous surgery) did not remain significant when IPs were added, suggesting mediation effects. Here too, a common contribution of IP variables is suggested by the non-significance of the unique contributions of most variables when introduced simultaneously, although each IP variable was significantly related to the HSIs when the effect of contextual variables was controlled for.

Comparative analysis

Comparative MRMs and SEMs were next built for T-PRI and RM-SIP scores in order to compare the contributions of all types of psychological variables to these

[53]Similar relations were identified based on Spearman's ρ correlations.

Model RM-SIP		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	4.219	4.403	2.641	3.114	3.214	3.362									
	Gender	-1.074	-.939	-1.598	.795	.820	.870	-.09	-.07	-.12						
	Spread of pain	.372	.519	.412	.108	.114	.122	.21**	.29**	.23**						
	Comorbidity	1.415	1.247	1.992	.735	.775	.825	.12	.10	.16*						
	Previous surgery	2.606	2.076	2.008	.729	.757	.810	.22**	.17**	.16						
	Anger	.962	.821	.670	.407	.408	.456	.21**	.17*	.14						
	Sad	.199	.711	.960	.419	.393	.457	.05	.18	.23*						
	Shame	.234	.231	.144	.345	.309	.385	.06	.06	.04						
	Anxiety	.217	.087	.156	.402	.374	.425	.05	.02	.03						
	Joy	-.169	-.580	-.196	.352	.356	.384	-.04	-.13	-.04	.28 (.24)	.39 (.36)	.36 (.33)	.11**	.18**	.16**

Table 7.42: Emotions and ERSs as predictors of pain-related disability

Model	work status	st.	B	SE B	95% CI for exp b lower	exp b	upper	Cox & Snell R^2	Nagelkerke R^2	% correct classif.	χ^2 diff.
1	...										
2	(Constant)	1	-.57	1.36		.57					
		2	.73	1.37		2.08					
		3	-.38	1.43		.69					
	Comorbidity	1	.64*	.32	1.00	1.89	3.56				
		2	.78*	.35	1.11	2.19	4.30				
		3	.71*	.35	1.02	2.03	4.07				
	Anger	1	.10	.18	.78	1.10	1.55				
		2	.07	.20	.72	1.07	1.59				
		3	.08	.20	.72	1.08	1.60				
	Sad	1	.11	.16	.82	1.12	1.54				
		2	-.04	.17	.69	.96	1.36				
		3	.14	.18	.81	1.15	1.63				
	Joy	1	-.20	.17	.58	.82	1.14	.08	.10	64	7.27 (1), $p < .01$
		2	-.38*	.17	.49	.68	.95	.09	.13	61	8.81 (1), $p < .01$
		3	-.27	.18	.54	.76	1.08	.11	.14	62	9.53 (1), $p < .01$

Table 7.43: Emotions and ERSs as predictors of work status

Model	GP visits	st.	B	SE B	95% CI for exp b			Cox & Snell R^2	Nagelkerke R^2	% correct classif.
					lower	exp b	upper			
1	(Constant)	1	-2.21*	.63		.11				
		2	-3.06**	.76		.05				
		3	-1.84	.66		.16				
	anger	1	.32	.16	1.00	1.37	1.89			
		2	.19	.18	.86	1.21	1.71			
		3	.29	.19	.93	1.34	1.92			
	sad	1	-.03	.17	.70	.97	1.35			
		2	.47**	.16	1.16	1.60	2.21			
		3	.39*	.18	1.03	1.47	2.11			
	shame	1	-.04	.15	.73	.97	1.28			
		2	-.12	.14	.68	.89	1.17			
		3	-.12	.16	.65	.88	1.20			
	anxiety	1	.28	.16	.97	1.32	1.79	.08	.11	64
		2	.19	.16	.89	1.21	1.64	.16	.21	70
		3	-.10	.17	.66	.91	1.25	.09	.12	63

Table 7.44: Emotions and ERSs as predictors of GP visits

Model T-PRI		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	5.44	-1.34	2.91	6.19	6.11	6.23									
	comorbidity	.07	2.66	3.33	1.28	1.34	1.34	.00	.13*	.16*						
	age - pain onset	-.13	-.14	-.16	.05	.06	.06	-.15*	-.15*	-.17**						
	previous surgery	2.26	2.98	2.62	1.29	1.35	1.36	.11	.14*	.12						
	Anger	1.14	.44	.82	.72	.74	.77	.14	.05	.10						
	Sad	1.06	1.54	1.47	.76	.72	.77	.16	.23*	.21						
	Shame	-.16	.80	1.22	.62	.56	.64	-.02	.12	.18						
	Anxiety	1.47	1.50	.66	.69	.66	.69	.19*	.18*	.08						
	Joy	.44	.91	.25	.64	.64	.65	.05	.11	.03	.20 (.17)	.27 (.24)	.22 (.30)	.14**	.16**	.21**
	S-PRI															
1	...															
2	(Constant)	8.944	8.271	6.968	2.635	2.782	2.709									
	age pain onset	-.108	-.121	-.109	.039	.043	.043	-.18**	-.19**	-.16*						
	previous surgery	1.596	2.326	1.852	.957	1.019	1.027	.11	.15*	.12						
	Anger	.611	.519	.732	.529	.558	.580	.10	.09	.12						
	Sad	.495	.579	.734	.545	.507	.569	.10	.12	.14						
	Shame	-.040	.470	.978	.461	.424	.481	-.01	.10	.19*						
	Anxiety	.908	.897	.565	.509	.487	.518	.16	.15	.10	.16 (.13)	.20 (.18)	.27 (.24)	.10**	.13**	.22**
	A-PRI															
1	...															
2	(Constant)	-2.85	-4.00	.02	1.77	1.79	1.80									
	comorbidity	.41	.93	.88	.43	.43	.44	.06	.14*	.13*						
	Anger	.53	-.00	.10	.24	.24	.25	.19*	-.00	.04						
	Sad	.52	.70	.54	.25	.23	.25	.23*	.31**	.24*						
	Shame	-.11	.32	.33	.21	.18	.21	-.05	.14	.15						
	Anxiety	.47	.55	.21	.23	.21	.22	.18*	.20**	.08						
	Joy	.20	.29	-.20	.21	.21	.21	.07	.11	-.08	.23 (.20)	.30 (.28)	.30 (.27)	.20**	.23**	.23**

Table 7.45: Emotions and ERSs as predictors of pain intensity

Model VAS		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	65.41	63.17	65.08	11.90	12.12	11.92									
	education	-4.75	-5.86	-5.38	4.44	4.24	4.48	-.07	-.09	-.08						
	previous surgery	6.34	6.85	11.22	4.46	4.30	4.54	.09	.11	.17*						
	Anger	7.29	3.23	4.62	2.47	2.36	2.56	.27**	.13	.18						
	Sad	.81	6.24	6.71	2.60	2.16	2.58	.04	.30**	.31**						
	Shame	-.42	-1.06	.69	2.18	1.80	2.14	-.02	-.05	.03						
	Anxiety	.38	.79	-2.60	2.37	2.06	2.29	.01	.03	-.10	.11 (.09)	.19 (.17)	.23 (.20)	.09**	.14**	.16**
	PPI															
1	(Constant)	1.98	1.80	1.69	.30	.33	.32									
	Anger	.21	.15	.17	.08	.09	.09	.23**	.16	.19						
	Sad	.12	.18	.15	.09	.08	.09	.15	.24*	.20						
	Shame	.09	-.04	-.02	.07	.07	.08	.11	-.06	-.03						
	Anxiety	-.09	.07	.04	.08	.08	.08	-.11	.08	.04	.14 (.12)	.15 (.13)	.14 (.12)	.14**	.15**	.14**

Table 7.46: Emotions and ERSs as predictors of pain intensity (cont.)

	st.	t-test / χ^2	Mean values		Pearson's r
			Adaptors	Non-adaptors	
RM-SIP*	1	$t(226) = 4.3$, $p < .001$	13	16	.28**
	2	$t(101) = 8.2$, $p < .001$	13	16	.47**
	3	$t(167) = 2.9$, $p = .004$	12	15	.22**
Work status	1	$\chi^2(1) = 7.2$, $p = .007$	–	–	.20**
	2	$\chi^2(1) = 8.1$, $p = .004$	–	–	.22**
	3	ns	–	–	–
S-PRI*	1	$t(226) = 3.9$, $p < .001$	13	17	.25**
	2	$t(193) = 4.7$, $p < .001$	11	16	.32**
	3	$t(167) = 2.6$, $p = .009$	14	18	.20**
A-PRI*	1	$t(226) = 4.3$, $p < .001$	4	6	.27**
	2	$t(137) = 7.4$, $p < .001$	3	6	.39**
	3	$t(167) = 4.2$, $p < .001$	4	6	.31**
T-PRI*	1	$t(226) = 4.3$, $p < .001$	17	23	.28**
	2	$t(106) = 6.2$, $p < .001$	14	22	.36**
	3	$t(167) = 3.3$, $p = .001$	19	24	.25**
VAS*	1	$t(191) = 5.6$, $p < .001$	92	113	.32**
	2	$t(194) = 6.0$, $p < .001$	78	104	.40**
	3	$t(167) = 3.5$, $p = .001$	94	110	.26**
PPI*	1	$t(226) = 5.1$, $p < .001$	3	4	.32**
	2	$t(194) = 5.8$, $p < .001$	3	4	.38**
	3	$t(167) = 3.1$, $p = .002$	3	4	.23**
Medication use	1	ns	–	–	–
	2	$t(194) = 2.5$, $p = .013$	2.3	2.8	.18*
	3	ns	–	–	–
GP visits	1	$\chi^2(1) = 8.2$, $p = .004$	–	–	.19**
	2	$\chi^2(1) = 8.9$, $p = .003$	–	–	.21**
	3	$\chi^2(1) = 7.0$, $p = .008$	–	–	.20**

Table 7.47: Differences in HSIs between the two IP clusters (*results confirmed by Mann-Whitney tests)

HSIs, controlling for the relevant demographic and medical variables. The selection of only two HSIs for this analysis was due to several reasons. First, the number of analyses necessary for all 9 HSIs for the 3 stages was considered too high to report adequately within the space limits of this section. Second, the analysis of the relationships between HSIs in Section 7.6.1 indicated that considering two categories, pain experience and pain behaviour, may be an adequate data reduction method, and therefore selecting one measure in each category was considered appropriate (although each HSI may be subject to different influences, as the correlational analyses indicated). Third, the two indices selected were psychometrically more reliable (as total scores) than the rest (as single item indicators) and also normally distributed and therefore were more suitable for regression analyses.

For the purpose of the comparative MRM analyses, the basic emotions and illness perceptions variables were considered as distinct functional sets, according to Cohen et al. (2003, Ch. 5). The demographic and medical variables were introduced first,

	st	1	2	3	4	5	6	7	8
Disability	1	.64**	.31**	-.12*	-.11	.48**	.38**	.05	.36**
	2	.67**	.30**	-.17*	-.18**	.47**	.36**	.01	.48**
	3	.66**	.33**	-.13	-.17*	.53**	.48**	.03	.49**
Work status	1	.48**	.24**	-.12	-.10	.28**	.22**	.08	.21**
	2	.50**	.14	-.10	-.03	.36**	.21**	.03	.29**
	3	.53**	.14	-.03	-.05	.37**	.33**	-.04	.33**
Medication	1	.24**	.14*	.08	.12	.23**	.12	.17**	.06
	2	.29**	.13*	-.00	.05	.24**	.13	.09	.19**
	3	.21**	.14	.08	.12	.15*	.18**	.03	.14*
GP visits	1	.30**	.13*	-.12	-.06	.18**	.28**	-.00	.26**
	2	.32**	.07	.03	-.06	.21**	.20**	-.03	.36**
	3	.27**	.03	-.02	-.04	.18**	.25**	-.03	.23**
S-PRI	1	.41**	.27**	-.11	-.11	.40**	.30**	.08	.25**
	2	.42**	.17**	-.18**	-.22**	.47**	.37**	-.09	.28**
	3	.52**	.28**	-.18**	-.11	.53**	.44**	-.07	.43**
A-PRI	1	.42**	.19**	-.12	-.17**	.32**	.39**	.07	.43**
	2	.49**	.19**	-.09	-.18**	.46**	.47**	-.09	.45**
	3	.58**	.30**	-.20**	-.17*	.47**	.51**	-.10	.54**
T-PRI	1	.44**	.26**	-.12	-.14*	.40**	.35**	.08	.32**
	2	.46**	.19**	-.16*	-.22**	.49**	.43**	-.09	.35**
	3	.57**	.30**	-.20**	-.13	.54**	.49**	-.08	.49**
VAS	1	.53**	.36**	-.19**	-.16**	.47**	.39**	.12	.32**
	2	.61**	.28**	-.11	-.16*	.58**	.46**	.00	.35**
	3	.61**	.35**	-.26**	-.20**	.60**	.54**	-.05	.51**
PPI	1	.50**	.30**	-.12	-.11	.51**	.34**	.10	.30**
	2	.56**	.26**	-.13*	-.22**	.52**	.40**	.01	.31**
	3	.58**	.27**	-.19**	-.17*	.57**	.47**	-.06	.46**

Table 7.48: Correlations between IPs and HSIs (1=consequences, 2=timeline, 3=personal control, 4=treatment control, 5=identity/symptoms, 6=concern, 7=understanding, 8=emotional impact)

followed by the basic emotions set, by the illness perceptions and finally by the CPA score. Although alternative sequences can be proposed for the three concepts, this particular sequence was intended to reflect the widely spread theoretical view of emotion as partly automatic and thus preceding cognitive evaluation[54], both being internal events that precede and determine to a certain extent overt behaviour (on which CPA focuses, as discussed in Chapter 3). Certainly, the relationships are bidirectional and a definitive temporal sequence cannot be established, especially given the inevitably retrospective character of the self-report data. Also, the IPs related to health status were to a large degree affect-related, and therefore the cognitive aspects relevant for adjustment to chronic pain were underrepresented in this analysis.

[54]Of course, another possibility would be to consider IPs as emotion appraisals preceding basic emotions, however as discussed in Chapter 4 emotions can be elicited via multiple levels, cognition being only one of them.

Model RM-SIP		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	-3.000	-.541	-2.833	2.057	2.178	2.271									
	Gender	-.846	-1.437	-1.313	.607	.650	.712	-.07	-.11	-.10						
	Spread of pain	.260	.327	.282	.084	.093	.097	.15**	.18**	.16**						
	Comorbidity	1.111	.775	2.051	.566	.641	.681	.10*	.06	.17**						
	Previous surgery	1.164	.855	1.325	.566	.635	.671	.10*	.07	.10*						
	consequences	1.357	1.437	1.005	.202	.210	.224	.47**	.53**	.38**						
	time line	.018	.070	-.047	.182	.198	.199	.01	.02	-.01						
	identity	.362	.035	.474	.176	.205	.217	.12*	.01	.15*						
	concern	-.057	-.171	.002	.159	.161	.177	-.02	-.07	.00						
	emotional resp.	.225	.360	.292	.151	.170	.186	.09	.15*	.12	.49 (.47)	.53 (.51)	.53 (.50)	.29**	.31**	.28**
Medication																
1	...															
2	(Constant)	.09	.73	1.03	.39	.35	.41									
	previous physio	.66	.46	.57	.19	.20	.22	.20**	.14*	.18**						
	negative events	.14	.22	.17	.05	.06	.06	.16**	.23**	.19**						
	consequences	.07	.13	.12	.05	.05	.06	.10	.21*	.21*						
	identity	.14	.04	-.01	.05	.06	.07	.19**	.06	-.02	.15 (.14)	.16 (.15)	.12 (.10)	.07**	.07**	.04*

Table 7.49: IPs as predictors of disability and medication use

Model	work status	st.	B	SE B	95% CI for exp b lower	exp b	upper	Cox & Snell R^2	Nagelkerke R^2	% correct classif.	χ^2 diff.
1	...										
2	(Constant)	1	-5.83*	1.07		.00					
		2	-5.27**	1.05		.01					
		3	-5.49**	1.10		.00					
	Comorbidity	1	.66*	.33	1.02	1.94	3.69				
		2	.73*	.36	1.03	2.07	4.17				
		3	.97**	.38	1.26	2.63	5.52				
	consequences	1	.70**	.14	1.53	2.01	2.64				
		2	.58**	.14	1.37	1.79	2.33				
		3	.62**	.14	1.40	1.85	2.45				
	identity	1	.07	.09	.89	1.07	1.28				
		2	.07	.13	.83	1.07	1.37				
		3	.05	.13	.81	1.05	1.36				
	concern	1	-.12	.10	.73	.89	1.09				
		2	-.07	.10	.77	.93	1.12				
		3	-.05	.11	.77	.95	1.17				
	emotional resp.	1	-.00	.10	.83	1.00	1.21	.26	.35	74	52.39(1), $p < .001$
		2	.03	.10	.85	1.03	1.25	.27	.36	73	43.35(1), $p < .001$
		3	-.03	.11	.78	.97	1.20	.31	.41	76	48.78(1), $p < .001$

Table 7.50: IPs as predictors of work status

GP					95% CI for exp b			Cox &	Nagelkerke	% correct
Model	visits	st.	B	SE B	lower	exp b	upper	Snell R^2	R^2	classif.
1	(Constant)	1	-2.86**	.67		.06				
		2	-2.74**	.65		.06				
		3	-2.27**	.65		.10				
	consequences	1	.19*	.09	1.01	1.21	1.45			
		2	.22*	.10	1.02	1.24	1.52			
		3	.17	.10	.97	1.19	1.45			
	identity	1	.03	.08	.88	1.03	1.21			
		2	-.03	.10	.80	.97	1.19			
		3	-.01	.10	.81	.99	1.20			
	concern	1	.09	.07	.95	1.10	1.27			
		2	-.09	.08	.78	.92	1.07			
		3	.11	.08	.95	1.11	1.31			
	emotional resp.	1	.11	.07	.97	1.11	1.28	.11	.15	67
		2	.29	.09	1.13	1.33	1.58	.15	.20	67
		3	.03	.09	.87	1.03	1.22	.09	.12	65

Table 7.51: IPs as predictors of GP visits

Model T-PRI		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	-2.52	1.54	-2.62	4.14	4.00	3.81									
	comorbidity	.76	1.83	2.76	1.13	1.21	1.15	.04	.09	.13*						
	age - pain onset	-.13	-.11	-.14	.05	.05	.05	-.15**	-.13*	-.16**						
	previous surgery	1.04	1.10	1.49	1.17	1.25	1.19	.05	.05	.07						
	consequences	.88	.58	.84	.41	.41	.39	.17*	.13	.19*						
	time line	.35	.01	.01	.38	.39	.35	.05	.00	.00						
	identity	1.14	1.26	1.30	.36	.41	.38	.21**	.25**	.25**						
	concern	.15	.82	.47	.33	.32	.32	.03	.21**	.12						
	emotional resp.	.71	.00	.54	.31	.33	.33	.16*	.00	.13	.28 (.26)	.32 (.30)	.45 (.42)	.20**	.21**	.30**
	S-PRI															
1	...															
2	(Constant)	.27	4.89	1.74	3.06	2.87	2.78									
	age pain onset	-.11	-.11	-.10	.03	.04	.04	-.17**	-.17**	-.15**						
	previous surgery	.81	1.24	1.15	.87	.93	.92	.05	.08	.07						
	consequences	.52	.39	.53	.31	.31	.31	.14	.12	.16						
	time line	.28	-.01	-.03	.30	.29	.27	.06	-.00	-.01						
	identity	1.09	1.06	1.12	.28	.30	.29	.27**	.29**	.29**						
	concern	.03	.56	.36	.24	.24	.25	.01	.19*	.12						
	emotional resp.	.35	-.18	.33	.23	.25	.26	.11	-.06	.11	.26 (.24)	.29 (.26)	.25 (.23)	.19**	.21**	.05**
	A-PRI															
1	...															
2	(Constant)	-2.69	-1.75	-3.23	1.33	1.23	1.17									
	comorbidity	.73	.92	.58	.38	.38	.38	.10	.14*	.09						
	consequences	.26	.24	.34	.14	.13	.13	.15	.16	.24**						
	time line	-.08	-.06	.00	.13	.12	.11	-.04	-.03	.00						
	treatment control	-.12	-.16	-.05	.07	.07	.07	-.09	-.12*	-.04						
	identity	.30	.29	.29	.13	.13	.13	.17*	.18*	.17*						
	concern	.07	.23	.14	.11	.10	.10	.05	.18*	.11						
	emotional resp.	.40	.16	.27	.11	.11	.11	.27**	.12	.20*	.28 (.27)	.35 (.33)	.42 (.40)	.24**	.28**	.36**

Table 7.52: IPs as predictors of pain intensity

Model VAS		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	...															
2	(Constant)	3.73	33.80	16.64	13.94	11.82	12.27									
	education	-1.53	-4.59	-1.63	3.46	3.22	3.30	-.02	-.07	-.03						
	previous surgery	-2.07	-.17	6.66	3.59	3.40	3.48	-.03	-.00	.10						
	consequences	5.04	5.24	2.46	1.26	1.12	1.17	.30**	.38**	.18*						
	time line	2.12	.07	-.04	1.21	1.05	1.03	.10	.00	-.00						
	treatment control	-.91	-1.08	-.65	.65	.63	.67	-.07	-.09	-.05						
	identity	4.08	4.06	5.23	1.15	1.11	1.19	.23**	.27**	.32**						
	concern	.31	2.22	1.86	1.01	.87	.93	.02	.18*	.15*						
	emotional resp.	1.33	-1.57	1.72	.95	.90	.95	.09	-.13	.13	.36 (.34)	.45 (.43)	.50 (.48)	.33**	.40**	.43**
	PPI															
1	(Constant)	-.16	.70	.52	.37	.36	.32									
	consequences	.14	.21	.12	.04	.04	.04	.24**	.40**	.25**						
	time line	.05	.01	-.03	.04	.04	.04	.07	.01	-.05						
	identity	.20	.12	.19	.04	.04	.04	.35**	.21**	.33**						
	concern	-.01	.08	.04	.03	.03	.03	-.03	.18*	.09						
	emotional resp.	.06	-.06	.05	.03	.04	.04	.12	-.14	.11	.34 (.33)	.37 (.35)	.41 (.40)	.34**	.37**	.41**

Table 7.53: IPs as predictors of pain intensity (cont.)

	N	Δ R^2	Residual variance	Numerator df	Number predictors	Power
context	203	.03/.02/.01	.70	1	6	.84/.67/.40
context+CPA	203	.22	.55	1	5	1.00
context+BEs (set)	179	.16	.66	5	9	1.00
context+BEs (ind)	179	.03/.02/.01	.66	1	9	.81/.64/.37
context+IPs (set)	203	.28	.50	5	9	1.00
context+IPs (ind)	203	.03/.02/.01	.50	1	9	.93/.81/.52
context+BEs+IPs (set)	178	.20	.48	5	14	1.00
context+BEs+IPs (ind)	178	.03/.02/.01	.48	1	14	.91/.77/.48
All (+CPA)	178	.01	.47	1	15	.47

Table 7.54: Power calculations for regression models (RM-SIP, stage 3 data)

Basic emotions and illness perceptions were both significant additions to the prediction equation for both T-PRI and RM-SIP. In contrast, the component that CPA did not share with emotion and cognition as reflected in the BES and BIPQ scores was only relevant for disability, and not for pain intensity (the last steps of the MRMs are presented in Tables 7.55 and 7.56). The partial coefficients of each basic emotion and illness perception were mostly non-significant (although each variable was significant at entry), suggesting that they explained a shared proportion of the variance in health status. Since the selection of only some basic emotions or illness perceptions would not be justified theoretically and would likely capitalise on chance fluctuations in the data set, no further model trimming was attempted[55].

The above exploratory MRM analyses used various numbers of predictors, different sample sizes (due to attrition and missing data related to BES) and obtained coefficients of different effect sizes. It is important therefore to identify post-hoc their statistical power. Since the number of cases was lower in the last stage due to attrition and pain-related disability had the highest number of contextual predictors, post-hoc computations for achieved power of identifying R^2 increase due to single predictors of sets of predictors were computed only for disability MRMs in stage 3, to illustrate the minimum power estimates at $\alpha = .05$. The power calculations are presented in Table 7.54 and indicate that, while identifying the contribution of sets of variables is characterised by more than sufficient power, small contributions of individual predictors are more difficult to assess based on this data set[56].

[55]This would be an adequate strategy if the aim of the analysis would be to achieve the most parsimonious list of predictors, which was not intended here.

[56]This limited power in relation to the individual predictors also recommends against selecting predictors based on parameter estimates as a method to achieve a parsimonious solution.

Model RM-SIP		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	control										.16(.15)	.20 (.19)	.20 (.18)			
2	control+BEs										.27 (.24)	.39 (.36)	.36 (.33)	.11**	.18**	.16**
3	control+BEs+IPs										.52 (.48)	.60 (.57)	.56 (.52)	.25**	.21**	.20**
4	(Constant)	2.22	4.84	-1.63	3.59	3.91	4.00									
	gender	-.23	-.79	-.87	.67	.67	.74	-.02	-.06	-.07						
	spread of pain	.21	.39	.24	.09	.10	.11	.12*	.22**	.13*						
	comorbidity	.64	.11	1.47	.60	.64	.70	.06	.01	.12*						
	previous surgery	.74	.65	.76	.62	.64	.70	.06	.05	.06						
	anger	.46	.42	.67	.35	.35	.39	.10	.09	.14						
	sadness	-.21	.13	.33	.35	.33	.40	-.06	.03	.08						
	shame	.72	.62	.45	.29	.26	.33	.18*	.16*	.11						
	anxiety	-.02	-.32	-.02	.34	.31	.38	.00	-.07	.00						
	happiness	.02	-.28	.37	.31	.31	.36	.00	-.06	.08						
	consequences	1.28	1.21	.95	.23	.23	.24	.46**	.45**	.36**						
	time line	.21	.17	.07	.21	.20	.20	.06	.05	.02						
	identity	.29	.12	.55	.19	.21	.22	.10	.04	.18*						
	concern	-.31	-.38	-.29	.18	.17	.19	-.13	-.16*	-.12						
	emotional resp.	-.22	.03	-.18	.20	.20	.23	-.10	.01	-.07						
	CPA	-1.53	-1.18	-1.29	.48	.50	.57	-.26**	-.20*	-.22*	.54 (.50)	.61 (.58)	.57 (.53)	.02**	.01*	.01*

Table 7.55: Emotions, IPs and CPA as predictors of pain disability

Model T-PRI		B			SE B			β			R^2 (adj R^2)			ΔR^2		
	stage	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	control										.07 (.06)	.11 (.10)	.13 (.12)			
2	control+BEs										.21 (.18)	.27 (.24)	.33 (.30)	.14**	.16**	.21**
3	(Constant)	-2.07	-5.04	-15.16	6.69	6.19	5.89									
	comorbidity	-.82	1.23	2.05	1.21	1.27	1.15	-.04	.06	.10						
	age - pain onset	-.11	-.10	-.09	.05	.05	.05	-.13*	-.11	-.10						
	previous surgery	.30	1.30	.75	1.28	1.29	1.19	.01	.06	.04						
	anger	.40	-.08	.82	.71	.72	.67	.05	-.01	.10						
	sadness	.46	1.11	.87	.74	.68	.67	.07	.16	.12						
	shame	.29	.98	1.65	.60	.54	.55	.04	.14	.24**						
	anxiety	1.32	.96	.15	.67	.62	.61	.17	.12	.02						
	happiness	.24	.36	.63	.65	.63	.57	.03	.05	.08						
	consequences	1.58	.59	1.14	.45	.44	.40	.32**	.13	.26**						
	time line	-.31	-.14	.15	.44	.40	.35	-.05	-.02	.03						
	identity	.61	1.17	1.47	.39	.42	.38	.12	.23**	.28**						
	concern	.07	.88	.33	.37	.33	.32	.02	.22**	.08						
	emotional resp.	.08	-.64	-.42	.40	.40	.39	.02	-.16	-.10	.33 (.29)	.40 (.36)	.54 (.51)	.12**	.13**	.21**

Table 7.56: Emotions and IPs as predictors of pain intensity

To further clarify the relations between these variables, SEM models were next developed[57]. Figure 7.12 presents the structure of the main model tested. The five discrete emotions are each measured by two items selected based on the psychometric analysis of the BES (see Chapter 8) and form together a second order latent representing emotional distress. Illness perceptions, acceptance, pain intensity and pain disability are each represented as separate latents, measured by the 5 emotion-related BIPQ items and by 4 items of the CPAQ, MPQ and RM-SIP scales. The selection of items was necessary in order to limit the number of manifest variables in the model (thus ensuring an acceptable statistical power) and also due to the suboptimal GOF of CFA models for most questionnaires. Control variables were not included in the model for the same reason of limiting the number of variables in the model. Additional paths were modeled from the BIPQ item referring to the perception of concern and the CPAQ item 18 ("My worries and fears about what pain will do to me are true") to the anxiety latent. Also, the BIPQ item referring to emotional impact was considered as an indicator for all discrete emotion latents (as a secondary exploratory aim to identify which of the emotions, if not all, are related to the individual's perception of emotional impact). The 5 main latents were allowed to covary freely; no directional relations were modeled, since the data were cross-sectional, and any models stipulating causal relations would be equivalent to this measurement model (as long as all paths are free).

Results are presented in Tables 7.57 (GOF indices) and 7.58 (measurement parameters and correlations between the 5 latents). The model did not represent an optimal fit to the data according to the χ^2 test, although the indices could be considered by some as suggesting an acceptable approximation. Further examination of modification indices indicated residual covariances between various items measuring different concepts (in addition to dropping the non-significant paths included in Table 7.58), for example, the CPAQ item 1 ("I am getting on with the business of living no matter what my level of pain is") loaded on the happiness/joy latent in two of the stages[58]. As LM tests showed different patterns for the three stages and are less reliable in the case of less optimal fit, no modification was attempted. It is possible that an unknown factor structure is more adequate to this data set, although a simple 1-factor structure with covarying errors only for basic emotion synonyms

[57]Latent variable modeling is also a recommended solution to decrease in statistical power due to the use of sets of multiple overlapping variables in MRMs, according to Cohen et al. (2003, p. 186).

[58]Wald tests indicated only dropping the paths between IP of emotional response and sadness, shame and joy latents (in all stages), plus the path between the CPAQ item 18 and anxiety, and the disturbance term of the sadness latent (in stage 3).

stage	1	2	3
S-B χ^2 (302)	503.43 $p < .001$	473.36 $p < .001$	466.66 $p < .001$
NFI	.81	.81	.82
NNFI	.90	.91	.92
CFI, IFI	.92	.92	.93
MFI	.63	.64	.64
RMSEA	.06 (.05-.06)	.06 (.05-.06)	.06 (.05-.06)

Table 7.57: Goodness of fit statistics for model in figure 7.12

had a comparatively lower fit, e.g. S-B $\chi^2(319) = 768.4$, NFI= .71; NNFI= .79; CFI= .81, RMSEA= $.09(.08 - .10)$. A simpler structure with one third-order factor (pain severity) on which all 5 concepts load did not lead to an improved fit, e.g. S-B $\chi^2(307) = 500.2$, NFI= .81; NNFI= .90; CFI= .92, RMSEA= $.06(.05 - .07)$, χ^2 diff.$(5) = 33.6$, $p < .001$[59].

Parameter estimates suggested strong connections between all concepts, especially concerning CPA (although results should be treated with caution due to suboptimal fit). Emotion/distress had only medium sized associations with IPs at the latent level (although IPs of concern and emotional response were significantly related to anxiety in all three stages). Notably, perceptions of emotional impact were not related to sadness and (lack of) joy (not reported), only to anxiety and (inconsistently) to anger and shame.

7.7.2 Individual growth trends

Although the data set was not characterised by clinically significant changes at group level, these negative results might mask relevant variability of change patterns between individuals. It is possible that the psychological and health status of some participants improved, while others' status remained stable or aggravated; if this were the case, explaining these differences based on the information collected would be an important research goal. Multilevel modeling of longitudinal data offers the possibility to explore the individual parameters of linear change and potential predictors of both between-subjects and within-subjects variance (Singer and Willett, 2003). More specifically, it allows a search for answers to specific questions such as: do participants differ in their clinical trajectories (i.e. improvement

[59]The lack of optimal fit might be also due to not specifying method effects, and to misspecification of categorical status of PD items. However, as the questionnaires have distinct response formats, the differences due to method cannot be distinguished from substantive differences. Also, specifying categorical status led to estimation errors.

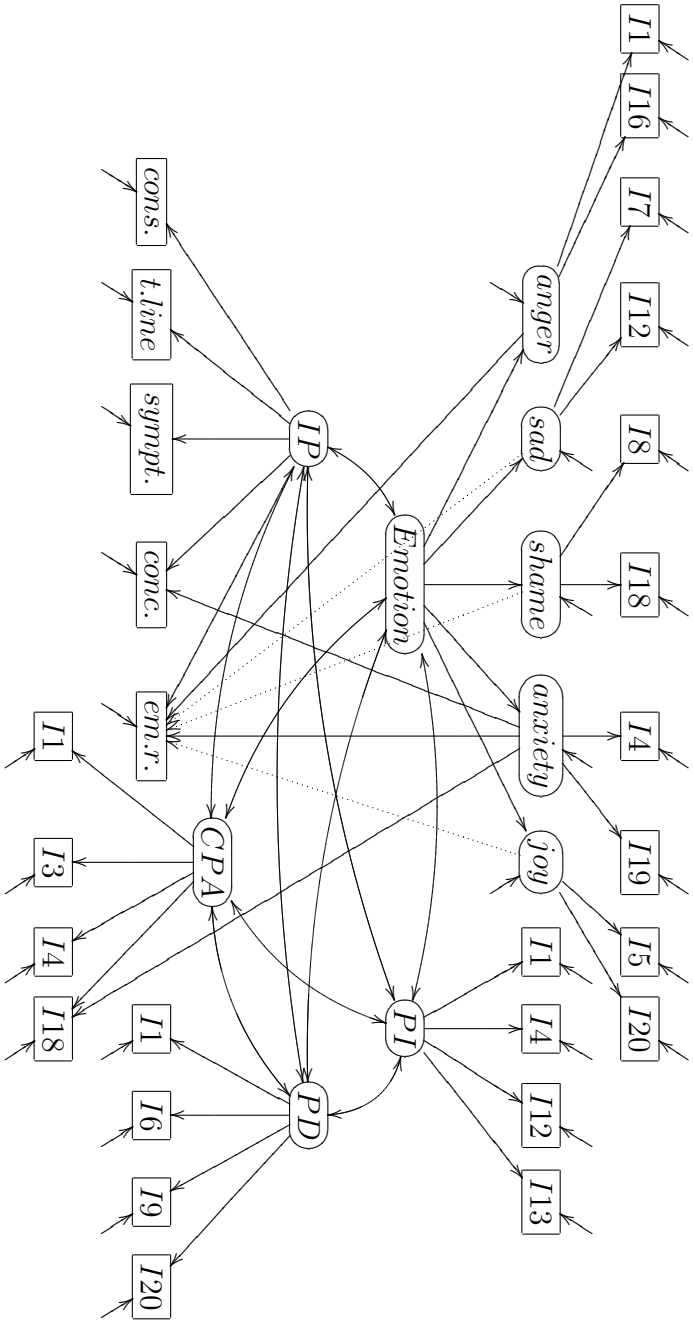


Figure 7.12: Relations between basic emotions, IPs, CPA, pain intensity and pain disability - SEM

observed/	1st/2nd order	β			E/D			R^2		
latent	latent	1	2	3	1	2	3	1	2	3
anger	Anger	.90	.85	.84	.43	.52	.55	.81	.73	.70
aggression	Anger	.80	.80	.81	.60	.60	.58	.64	.64	.66
misery	Sad	.90	.87	.90	.45	.50	.43	.81	.75	.81
gloominess	Sad	.81	.81	.79	.59	.59	.62	.65	.65	.62
guilt	shame	.70	.78	.79	.71	.63	.61	.49	.60	.63
blameworthy	shame	.79	.83	.79	.61	.56	.61	.63	.69	.63
anxiety	anxiety	.76	.72	.84	.65	.70	.54	.58	.51	.71
worry	anxiety	.79	.63	.86	.61	.78	.52	.63	.39	.74
happy	joy	.90	.92	.94	.43	.40	.34	.81	.84	.88
cheerful	joy	.88	.87	.89	.46	.49	.46	.79	.76	.79
anger	emotion	.72	.72	.79	.69	.70	.61	.52	.51	.63
sadness	emotion	.92	.92	.94	.40	.39	.34	.84	.85	.89
shame	emotion	.80	.73	.83	.60	.68	.56	.64	.53	.69
anxiety	emotion	.85	.85	.81	.54	.52	.59	.71	.73	.65
joy	emotion	-.71	-.74	-.72	.71	.68	.70	.50	.54	.51
consequences	IP	.94	.90	.92	.38	.44	.38	.86	.81	.85
timeline	IP	.47	.45	.50	.89	.89	.87	.21	.21	.25
symptoms	IP	.60	.75	.70	.82	.66	.72	.33	.56	.49
concern	IP	.55	.49	.64	–	–	–	–	–	–
concern	anxiety	.31	.39	.28	.71	.68	.64	.50	.53	.60
emot. resp.	IP	.34	.41	.54	–	–	–	–	–	–
emot. resp.	anger	.24	.26	ns	–	–	–	–	–	–
	shame	ns	.17	ns	–	–	–	–	–	–
emot. resp.	anxiety	.31	.45	.28	.62	.56	.56	.62	.69	.69
CPA 1	CPA	.60	.53	.56	.80	.85	.83	.36	.28	.31
CPA 3	CPA	.40	.32	.42	.92	.95	.91	.16	.10	.17
CPA 4	CPA	-.49	-.44	-.50	.87	.90	.87	.24	.19	.25
CPA 18	CPA	-.38	-.21	-.61	–	–	–	–	–	–
CPA 18	anxiety	.28	.55	ns	.80	.69	.76	.36	.53	.42
PI 1	PI	.38	.56	.63	.93	.83	.78	.14	.31	.40
PI 4	PI	.61	.55	.58	.80	.83	.82	.37	.30	.34
PI 12	PI	.58	.56	.58	.81	.83	.81	.34	.31	.34
PI 13	PI	.69	.65	.70	.72	.76	.71	.48	.43	.49
PD 1	PD	.69	.75	.75	.73	.66	.66	.47	.56	.56
PD 6	PD	.35	.37	.44	.94	.93	.90	.12	.14	.19
PD 9	PD	.53	.57	.59	.85	.82	.81	.28	.32	.35
PD 20	PD	.42	.48	.42	.91	.88	.91	.18	.23	.18

	Emotion (distress)			IP			CPA			PI		
r	1	2	3	1	2	3	1	2	3	1	2	3
IP	.34	.42	.41									
CPA	-.79	-.91	-.81	-.70	-.88	-.76						
PI	.39	.51	.56	.59	.67	.74	-.66	-.74	-.69			
PD	.48	.64	.62	.86	.79	.79	-.79	-.98	-.75	.60	.73	.79

Table 7.58: Parameter estimates for model in Figure 7.12

or worsening of psychological or health status)? If yes, what personal or contextual characteristics influence these differences? In addition, do the participants' psychological or health status trajectories fluctuate in time? If yes, what characteristics might influence these fluctuations? This exploratory analysis aimed to shed more light onto such issues.

Total scores on CPA, discrete emotions, emotion-related illness perceptions, pain disability and pain intensity were considered for this analysis, given their selection in the previous analyses. A first step was to identify if the variance at the individual level (especially in score changes across the study time frame) was statistically significant (and therefore worth explaining) for all time-varying concepts, based on unconditional means and unconditional growth models. Models were next developed to explore the role of contextual and psychological variables on pain intensity and pain disability.

The multilevel longitudinal modeling was performed using the SPSS MIXED procedure, following the recommendations of Singer and Willett (2003), Peugh and Enders (2005), Leyland (2006), and Hedeker and Gibbons (2006). A 3-wave data set is considered the minimum requirement for such analyses; although it is acknowledged that more waves of data collection would improve the accuracy of estimates. On the other hand, the sample size of the present study is considerably larger than some other similar studies (e.g. Affleck et al., 1999; Strand et al., 2006). Baseline models were fitted for all outcomes, and predictors were added incrementally to the model comparing differences in overall model fit and relevant parameters (selected models are presented, for brevity). Only the intercept and stage (where appropriate) were considered as random effects, to preserve the parsimony of the solution and the statistical power[60]. Residuals were examined for normality and heteroscedasticity and were found satisfactory[61]. Time was centered on the first wave values for ease of interpretation (since the study period represents a snapshot of the chronic illness trajectories and it is not characterised by any special event or treatment common to all participants). For time-varying variables, individual mean scores were computed and centered on the grand mean, and individual time-varying scores were centered on the person means, in order to distinguish trait-like characteristics from state-like changes. Two structures for level-2 covariance matrix (a more restricted first-order

[60]The limited number of measurement occasions for each individual does not offer sufficient data for estimating additional variance components (Singer and Willett, 2003, p. 169).

[61]A few exceptions were related to outcome variables with skewed distributions, such as shame and perceptions of timeline. However, as these variables were only explored in unconditioned growth models, no transformations were performed, for ease of interpretation.

autoregressive versus unstructured) were tested, the latter proving more adequate based on significant differences in deviance statistics and examination of normality and heteroscedasticity of residuals. Full maximum likelihood (ML) estimation was used for all models.

An important word of caution is necessary regarding the issue of causality in these models: the use of time-varying predictors does not allow causal inferences, especially when these predictors are endogenous, as in the present data set. The models only regress the outcomes on simultaneous (not prior) predictors. Unfortunately, in this multilevel framework the limited 3-wave data do not allow a time-lagged model, in which prior status of predictors is linked to current status on outcomes. Regressing time-2 outcomes on time-1 predictors would lead to only 2 waves with data for both types of variables (Hedeker and Gibbons, 2006, p. 70), which would not be sufficient for estimating intraindividual regression equations. Such model would be the method of choice in addressing problems of causal direction inherent in models that consider time-varying endogenous predictors (Singer and Willett, 2003, p. 177–81).

The results for the first step analyses (intra- versus interindividual changes) are presented in Tables 7.59, 7.60 and 7.61. All variables were characterised by significant variations at both intra- and interindividual levels; the proportion of the total outcome variation that lies between subjects was estimated by the intra-class coefficient (ICC) and varied between .83 for pain-related disability (participants had relatively stable levels of disability, however the differences between participants were comparatively large) and .52 for perceptions of illness identity (participants experienced illness-related symptoms to variable degrees, the variability being comparable with the differences between participants). Therefore, the attempts to explain variance at both levels is justified.

The unconditional growth models explored the degree to which time (i.e. the linear changes between the three stages) explained variance in scores at both levels. CPA, anger, sadness, disability and perceptions of consequences, concern and emotional impact were characterised by significant but limited changes at group level, but no differences between individuals regarding the change trajectories (the random parameter of time change was therefore eliminated in a third model (labelled Un.Gr.^{b}). In contrast, shame, anxiety, happiness, and perceptions of illness identity (symptoms) and timeline were stable across the three stages in terms of linear trends. An interesting case was pain intensity (T-PRI), which did not show change at group level but differences in linear trends were significant, and partly explained

		CPA			RMSIP			TPRI		
		Un.Ms	Un.Gr.	Un.Gr^b	Un.Ms	Un.Gr.	Un.Gr.^{b}	Un.Ms	Un.Gr.	Un.Gr.R
Fixed effects										
initial	intercept	2.729**	2.644**	2.644**	12.953**	13.388**	13.394**	19.112**	18.950**	18.950
status		(.055)	(.057)	(.058)	(.346)	(.351)	(.358)	(.558)	(.616)	(.617)
rate of	intercept		.100**	.102**		-.514**	-.523**		.179	.179
change			(.022)	(.020)		(.125)	(.117)		(.326)	(.326)
Random effects										
Level 1	within	.184**	.145**	.175**	6.106**	5.016**	5.856**	37.594**	28.465**	28.466
	person	(.012)	(.014)	(.012)	(.415)	(.482)	(.398)	(2.553)	(2.735)	(5.335)
ICC		.80			.83			.64		
Level 2										
initial	intercept	.746**	.742**	.744**	29.413**	28.609**	29.314**	67.663**	76.725**	77.104
status		(.071)	(.076)	(.070)	(2.760)	(2.875)	(2.741)	(7.299)	(3.128)	(8.780)
rate of	rate of		-.006 ns	–		-.205	–		-7.627*	9.388
change	change		(.021)			(.748)			(3.808)	(3.064)
co-			.030*	–		.853*	–		9.282**	$r = -.29$
variance			(.012)			(.405)			(2.677)	
Pseudo-	model		.01	.01		.01	.01		.00	–
R^2	within person		.21	.05		.18	.04		.24	–
GOF	-2LL	1450.16	1417.23	1425.28	3934.69	3908.18	3915.01	4995.827	4981.872	4982
	AIC	1456.16	1429.23	1433.28	3940.69	3920.18	3923.01	5001.827	4993.872	4994
	BIC	1469.82	1456.55	1451.50	3954.33	3947.47	3941.21	5015.489	5021.195	5021

Table 7.59: Unconditional means and growth models for CPA, disability (RM-SIP) and pain intensity (T-PRI)

		anger			sadness			shame		anxiety		happiness	
		Un.Ms	Un.Gr.	Un.Gr^b	Un.Ms	Un.Gr.	Un.Gr^b	Un.Ms	Un.Gr.	Un.Ms	Un.Gr.	Un.Ms	Un.Gr.
Fixed effects													
initial	intercept	4.346**	4.420**	4.421**	3.544**	3.632**	3.632**	2.879**	2.918**	4.648**	4.654**	4.807**	4.827**
status		(.077)	(.083)	(.081)	(.094)	(.099)	(.099)	(.092)	(.095)	(.079)	(.084)	(.089)	(.080)
rate of	intercept		-.086**	-.086**		-.102**	-.102**		-.044		-.007		-.023
change			(.033)	(.030)		(.036)	(.035)		(.037)		(.034)		(.032)
Random effects													
Level 1	within	.363**	.296**	.357**	.479**	.430**	.469**	.489**	.459**	.434**	.434**	.368**	.351**
	person	(.026)	(.030)	(.026)	(.035)	(.044)	(.034)	(.036)	(.047)	(.032)	(.032)	(.027)	(.036)
ICC		.76			.76			.77		.73		.77	
Level 2													
initial	intercept	1.166**	1.279**	1.162**	1.758**	1.806**	1.162**	1.670**	1.620**	1.186**	1.191**	1.219**	1.128**
status		(.126)	(.148)	(.125)	(.186)	(.210)	(.125)	(.179)	(.196)	(.130)	(.148)	(.131)	(.139)
rate of	rate of		-.084	–		-.038	–		-.019		-.003		-.049
change	change		(.047)			(.062)			(.060)		(.039)		(.042)
covariance			.062*	–		.040	–		.029		.000		.017
			(.027)			(.027)			(.035)		(.000)		(.027)
Pseudo-	model		.01	.01		.004	.01		.002		.0		.00
R^2	within person		.18	.02		.10	.002		.06		.00		.05
GOF	-2LL	1584.62	1570.86	1576.85	1776.87	1767.19	1768.58	1775.46	1772.28	1660.26	1660.21	1598.91	1594.97
	AIC	1590.62	1582.86	1584.85	1782.87	1779.19	1776.58	1781.46	1784.28	1666.26	1672.21	1604.91	1606.97
	BIC	1603.80	1609.22	1602.42	1796.05	1805.56	1794.15	1794.64	1810.64	1679.44	1698.57	1618.09	1633.33

Table 7.60: Unconditional means and growth models for discrete emotions

		Conseq.			T-line		Identity		Concern			Em.resp.		
		Un.Ms	Un.Gr.	Un.Gr^b	Un.Ms	Un.Gr.	Un.Ms	Un.Gr.	Un.Ms	Un.Gr.	Un.Gr^b	Un.Ms	Un.Gr.	Un.Gr^b
Fixed effects														
initial	intercept	7.340**	7.541**	7.543**	8.936**	9.000**	7.491**	7.587**	7.174**	7.427**	7.428**	6.996**	7.166**	7.166**
status		(.121)	(.122)	(.129)	(.090)	(.095)	(.103)	(.118)	(.134)	(.141)	(.147)	(.131)	(.139)	(.144)
rate of	intercept		-.233**	-.238**		-.075		-.111		-.292**	-.296**		-.196*	-.199*
change			(.055)	(.054)		(.054)		(.067)		(.072)	(.072)		(.070)	(.070)
Random effects														
Level 1	within	1.315**	1.251**	1.265**	1.159**	1.033**	1.922**	1.826**	2.352**	2.257**	2.281**	2.196**	2.137**	2.167**
	person	(.089)	(.119)	(.085)	(.079)	(.099)	(.129)	(.173)	(.158)	(.218)	(.154)	(.148)	(.142)	(.146)
ICC		.72			.59		.52		.62			.63		
Level 2														
initial	intercept	3.343**	2.898**	3.331**	1.665**	1.500**	2.041**	2.116**	3.846**	3.437**	3.818**	3.677**	3.312**	3.652**
status		(.334)	(.356)	(.331)	(.188)	(.224)	(.247)	(.350)	(.416)	(.493)	(.411)	(.396)	(.441)	(.392)
rate of	rate of		.223	–		.067		-.066	–	.181	–		.146	–
change	change		(.123)			(.102)		(.164)		(.203)			(.143)	
co-			.017	–		.123		.087		.010	–		.000	–
variance			(.088)			(.079)		(.124)		(.154)			(.000)	
Pseudo-	model		.01	.01		.00		.003		.01	.01		.01	.01
R^2	within prs.		.05	.04		.11		.05		–	–		.03	.01
GOF	-2LL	2719.17	2694.38	2700.10	2505.39	2494.78	2799.97	2796.63	3029.48	3011.37	3012.91	2986.50	2977.45	2978.56
	AIC	2725.17	2706.38	2708.10	2511.39	2506.78	2805.97	2808.63	3035.48	3023.37	3020.91	2992.50	2989.45	2986.56
	BIC	2738.84	2733.71	2726.32	2525.05	2534.10	2819.63	2835.95	3049.15	3050.69	3039.12	3006.16	3016.77	3004.77

Table 7.61: Unconditional means and growth models for emotion-related illness perceptions

by differences in initial pain levels[62]. Importantly, in all unconditioned models a significant amount of intra-individual variance (intra-individual fluctuations from their personal change trajectories) remained, justifying further attempts to search for possible time-varying predictors[63].

Further exploratory analyses aimed at identifying time-invariant and time-varying predictors of intra- and interpersonal variation in pain-related disability (RM-SIP) and pain intensity (T-PRI). Results are not presented here due to the similarity to the multiple regression models. Suffice it to say that, as in the previous MRMs, there was an extensive overlap between acceptance, basic emotions and illness perceptions in predicting health status. Thus, while all variables explained a significant proportion of the variance in disability and pain intensity when entered separately (controlling for the relevant contextual variables), entering several variables in a single model led to most variables explaining non-significant proportion of the outcome variance. Since selecting variables based on significance levels would have had no theoretical support and would have capitalised on change fluctuations in the sample, no further steps were taken to achieve a simplified prediction model. However the omnipresence of the overlap between measures of chronic pain impact in the analyses presented so far point to the relevance of the last exploratory step of this study: the issue of separating components of pain and emotion in the context of self-report.

7.7.3 Discrimination ability

This final exploratory analysis aimed at identifying inter and intraindividual differences in the ability to distinguish between different aspects of the chronic pain experience, such as between sensory and affective components of pain perception, or between pain and emotional distress, or between different emotional reactions. Following the methodology applied by Zautra et al. (2001, 2005), Strand et al. (2006) and Kratz et al. (2007), moderation analyses were performed in a HLM framework

[62]The model estimation by SPSS Mixed (both ML and REML) resulted in a negative variance of the level-2 rate of change, therefore the model was also estimated using the R(lmer) package, which provided similar estimates with the exception of the problematic parameter, now within adequate limits (labelled Un.Gr.R)

[63]A related research goal in this context could be to assess the relevance of patient clusters as predictors of interindividual differences in improvement or deterioration of health status. However, this question became meaningless for most variables due to the non-significant differences in change slopes. Only differences in T-PRI change trajectories were estimated, given that T-PRI change trajectories showed significant interindividual variation. Neither illness perception nor acceptance clusters accounted for any variance in pain intensity changes (non-significant interactions between categories and time), despite having a significant main effect on the average (stable) levels of pain intensity reported (results not reported).

in order to identify the factors that influence the strength of correlations between three main concept diads: affective and sensory pain, pain and emotion ratings, and happiness and negative emotions ratings. Without attempting a replication of any of the previous studies, the focus of these analyses was to identify whether similar relationships can be detected at wider time intervals, as opposed to those identified based on daily or weekly ratings.

Regarding the distinction between affective and sensory pain qualities, exploratory moderation analyses were performed with the following predictors: acceptance, discrete emotions, ERSs, illness perceptions, pain disability and several contextual variables. A-PRI scores were considered the outcome, while S-PRI was modeled as the main predictor, both as individual mean scores (labelled 'mean') at level 2 and as deviations ('dev') from personal trajectories at level 1. Stage number was included as an additional level 1 predictor to control for personal change trends (fixed and random effects). Comorbidity (high versus low scores) was included as an additional level 2 control variable, based on the previous MRM analyses. The potential moderators were added in separate models based on this baseline[64]. For example, the core model components tested for the mediation role of CPA are described in the following equations:

$$\begin{aligned}\text{Level 1: A-PRI} =& \beta_0 + \beta_1\text{S-PRI(dev)} + \beta_2\text{CPA(dev)} \\ &+ \beta_3\text{S-PRI(dev)} \times \text{CPA(dev)} + \beta_4\text{stage} + \epsilon \qquad (7.1)\end{aligned}$$

$$\begin{aligned}\text{Level 2: } \beta_0 =& \gamma_{00} + \gamma_{01}\text{S-PRI(mean)} + \gamma_{02}\text{CPA(mean)} \\ &+ \gamma_{03}\text{comorbidity} + \zeta_0 \qquad (7.2)\\ \beta_1 =& \gamma_{10} + \gamma_{11}\text{CPA(mean)} \qquad (7.3)\\ \beta_3 =& \gamma_{30} + \gamma_{31}\text{CPA(mean)} \qquad (7.4)\\ \beta_4 =& \gamma_{40} + \zeta_{4,} \qquad (7.5)\end{aligned}$$

where the level 1 equation describes the predictors of intra-individual fluctuations of A-PRI scores, plus random error (ϵ), and the level 2 equations describe the predic-

[64] In contrast to previous studies which started with an extended specification (e.g. Zautra et al., 2005 and eventually eliminated non-significant predictors in the final model (e.g. Zautra et al., 2001), these analyses were performed incrementally, as advised by Singer and Willett (2003), with a focus on the SPRI-moderator interactions. Other interactions, e.g. between the time slope or comorbidity and the existing predictors, were included only if significant.

tors of the interindividual differences in level 1 parameters[65]. The main parameters of interest are γ_{11} (the degree to which mean levels of CPA influence the strength of the relationship between fluctuations in SPRI scores and APRI scores), γ_{30} (the degree to which fluctuations in CPA influence the strength of the relationship between fluctuations in SPRI scores and APRI scores) and γ_{31} (the degree to which the moderating effect of fluctuations in CPA varies with mean levels of CPA).

The results for the moderating effects of CPA and selected emotions, ERSs and IPs are shown in Tables 7.62 and 7.63[66]. The relationship between sensory and affective pain reports was moderated by mean levels of chronic pain acceptance, anger, sadness, shame, happiness/joy (but not anxiety), dysfunctional emotion regulation strategies (but not functional strategies)[67], and perceptions of concern and emotional response. These moderation mechanisms are also potentially overlapping, but all together suggest that in individuals with higher mean levels of emotional distress the reports of sensory and affective pain are more correlated. Interestingly, only fluctuations in symptoms reports (illness identity perceptions) mediated this relationship, suggesting that when symptoms intensify, sensory and affective pain become more difficult to disentangle.

No such moderation effects were found for pain disability, perceptions of illness consequences and timeline, or for contextual factors such as gender, previous surgery, pain management or psychological therapies, visceral pain, back pain, head/neck pain, education, marital status, pain spread, age at pain onset, reported comorbid depression, age, pain duration, previous negative or positive events[68].

Another set of models assessed to what extent the correlation between pain and emotional distress was moderated by positive emotion (as suggested by previous research), or other variables. Anger, sadness, shame and anxiety were considered separately as outcomes, while T-PRI scores were modeled as the main predictors (as individual means and deviation scores). Additional control variables were added based on previous MRM and HLM analyses, which also informed the inclusion of

[65]Only the intercept and time slopes were modeled with random components (ζ_0, ζ_4), as explained previously.

[66]The model parameters presented were estimated using the R(lmer) package, due to a negative variance estimate of the level-2 rate of change given by SPSS Mixed. As previously for T-PRI, estimates were similar, with this single exception. Significance is therefore reported based on the SPSS Mixed results.

[67]Item level analyses confirmed the moderation effect of internal dysfunctional ERSs, but not external dysfunctional, and the lack of moderation by functional strategies.

[68]The only two exceptions were the minimal contextual moderating effects of the presence of pain in the hands/legs or of comorbid arthritis.

A-PRI		Baseline	CPA	Anger	Sadness	Shame	Joy
Fixed effects							
Level 1	intercept	-.734**	2.931**	-2.192**	-1.745**	-1.317**	1.413*
		(.411)	(.582)	(.549)	(.449)	(.449)	(.704)
	S-PRI(dev)	.264**	.416**	–	–	.134**	.568**
		(.019)	(.059)			(.047)	(.082)
	Moderator(dev)		-.955**	.809**	.414**	.306*	–
			(.118)	(.266)	(.128)	(127)	
	S-PRI(dev)		–	–	–	–	–
	*Moderator(dev)						
	stage	.175	-.127	-.222*	-.202*	-.206*	-.230*
		(.093)	(.094)	(.097)	(.097)	(.098)	(.094)
	Moderator(dev)		–	-.632**	–	–	–
	*stage			(.212)			
Level 2	comorbidity	.708**	.400	.600*	.521*	.563*	.612**
		(.230)	(.206)	(.240)	(.234)	(.242)	(.238)
	S-PRI	.320**	.277**	.309**	.299**	.315**	.326**
	(mean)	(.017)	(.016)	(.020)	(.019)	(.019)	(.019)
	Moderator		-.955**	.428**	.474**	.335**	-.411**
	(mean)		(.118)	(.113)	(.091)	(.093)	(.102)
Level1*	Moderator(mean)		-.059**	.057**	.066**	.039**	-.065**
Level2	*S-PRI(dev)		(.020)	(.004)	(.004)	(.014)	(.017)
	Moderator(mean)		–	–	–	–	–
	*S-PRI*Mod(dev)						
Random effects							
Level 1	within person	2.478**	2.458**	2.409**	2.357**	2.321**	2.456**
		(1.574)	(1.568)	(1.552)	(1.535)	(1.524)	(1.567)
Level 2	intercept	3.403**	3.029**	3.079**	3.009**	3.278**	3.006**
		(1.845)	(1.741)	(1.755)	(1.735)	(1.811)	(1.734)
	stage	.751	.700	.605	.648	.700	.515
		(.867)	(.837)	(778)	(.805)	(.837)	(.717)
	correlation	-.60**	-.74**	-.61**	-.64**	-.62**	-.61**
Goodness-of-fit							
	-2LL	3030	2966	2578	2562	2580	2574
	AIC	3048	2991	2601	2585	2605	2596
	BIC	3088	3045	2654	2633	2658	2644

Table 7.62: Moderation of the S-PRI and A-PRI relation by CPA and discrete emotions

A-PRI		Baseline	ID	ED	Sympt.	Concern	Em.Resp.
Fixed effects							
Level 1	intercept	-.734**	-2.185**	-.703	-1.711**	2.232**	.491
		(.411)	(.493)	(.410)	(.570)	(.450)	(.354)
	S-PRI(dev)	.264**	–	–	.265**	–	–
		(.019)			(.019)		
	Moderator(dev)		–	–	–	.407*	.200**
						(.166)	(.056)
	S-PRI(dev)		–	–	-.042*	–	–
	*Moderator(dev)				(.080)		
	stage	.175	-.173	-.200*	-.164	-.139	-.144
		(.093)	(.092)	(.092)	(.093)	(.094)	(.091)
Level 2	comorbidity	.708**	.643**	.703**	.602**	.498*	.502*
		(.230)	(.220)	(.230)	(.230)	(.215)	(.212)
	S-PRI(mean)	.320**	.294**	.320**	.292**	.280**	–
		(.017)	(.017)	(.017)	(.020)	(.017)	
	Moderator(mean)		.840**	–	.209**	.332**	–
			(.170)		(.080)	(.052)	
	Moderator(mean)		–	–	–	–	.035**
	*S-PRI(mean)						(.002)
Level1*	Moderator(mean)		.109**	.170**	–	.036**	.036**
Level2	*S-PRI(dev)		(.008)	(.012)		(.003)	(.003)
	Moderator(mean)		–	–	–	–	–
	*S-PRI*Mod(dev)						
	Moderator(dev)		–	–	–	-.230*	–
	*comorbidity					(.111)	
Random effects							
Level 1	within person	2.478**	2.533**	2.533**	2.450**	2.420**	2.400**
		(1.574)	(1.591)	(1.592)	(1.565)	(1.556)	(1.549)
Level 2	intercept	3.403**	3.127**	3.370**	3.418**	2.974**	2.706**
		(1.845)	(1.768)	(1.836)	(1.849)	(1.725)	(1.645)
	stage	.751	.662	.662	.746	.726	.645
		(.867)	(.813)	(.814)	(.864)	(.852)	(.803)
	correlation	-.60**	-.64**	-.61**	-.62**	-.65**	-.60**
Goodness-of-fit							
	-2LL	3030	3006	3028	3026	2990	2972
	AIC	3048	3027	3046	3048	3014	2992
	BIC	3088	3072	3087	3098	3069	3038

Table 7.63: Moderation of the S-PRI and A-PRI relation by selected ERSs and IPs

stage as a fixed effect in anger and sadness. Happiness, CPA, ERSs, IPs, pain disability and control variables were included as potential moderators in separate runs[69]. For example, the model for anxiety with happiness as moderator was stipulated as follows:

$$\begin{aligned}\text{Level 1: Anxiety} =& \beta_0 + \beta_1 \text{T-PRI(dev)} + \beta_2 \text{Happy(dev)} \\ &+ \beta_3 \text{T-PRI(dev)} \times \text{Happy(dev)} + \beta_4 \text{stage} + \epsilon \qquad (7.6)\end{aligned}$$

$$\begin{aligned}\text{Level 2: } \beta_0 =& \gamma_{00} + \gamma_{01} \text{T-PRI(mean)} + \gamma_{02} \text{Happy(mean)} \\ &+ \gamma_{03} \text{comorbidity} + \gamma_{04} \text{Negative events} + \zeta_0 \qquad (7.7)\\ \beta_1 =& \gamma_{10} + \gamma_{11} \text{Happy(mean)} + \gamma_{12} \text{T-PRI(mean)} \qquad (7.8)\\ \beta_2 =& \gamma_{20} + \gamma_{21} \text{Happy(mean)} + \gamma_{22} \text{T-PRI(mean)} \qquad (7.9)\\ \beta_3 =& \gamma_{30} + \gamma_{31} \text{Happy(mean)} + \gamma_{32} \text{T-PRI(mean)} \qquad (7.10)\\ \beta_4 =& \gamma_{40.} \qquad (7.11)\end{aligned}$$

The main differences from the previous analyses are the inclusion of a symmetric equation for the second predictor/moderator (here happiness), which tested whether mean levels of pain and happiness acted as a moderator on the strength of the association between happiness scores and anger scores[70], and the inclusion of pain mean scores as possible moderators of the Level 1 relations[71]. Also, the time trajectories were considered identical, based on previous growth models. The two-way and three-way interactions (parameters $\gamma_{11}, \gamma_{12}, \gamma_{21}, \gamma_{22}, \gamma_{31}, \gamma_{32}$) are the main focus of the analyses.

No interactions between pain and happiness were significant in relation to anger, sadness or shame scores. Only anxiety scores were predicted by significant interactions between fluctuations in pain and happiness and between mean scores of pain and fluctuations in happiness. Table 7.64 presents three consecutive models

[69]Due to the high number of the possible combinations, only analyses related to pain and happiness as predictors of emotional distress are presented. However it is interesting to note that, in contrast with Kratz et al. (2007), no moderation effect of mean levels of CPA was found between pain fluctuations and any negative emotions; only interactions between mean CPA and mean pain levels were identified for sadness and shame.

[70]This addition is related to the following set of analyses, which focused on exploring mediators for the positive-negative emotions distinction.

[71]These model components were also tested in the previous set of analyses, but were not considered a key element and were indeed found to be non-significant.

ANXIETY		Baseline	Model 1	Model 2	Model 3
Fixed effects					
Level 1	intercept	3.078**	5.429**	5.421**	5.379**
		(.242)	(.374)	(.374)	(.374)
	T-PRI(dev)	.021**	.018**	.018**	.018**
		(.005)	(.005)	(.005)	(.005)
	Happy(dev)		-.212**	-.165	–
			(.054)	(.127)	
	T-PRI(dev)		.024*	.020	–
	* Happy(dev)		(.012)	(.012)	
Level 2	Negative events	.079*	–	–	–
		(.031)			
	comorbidity	.384**	.340**	.340**	.343**
		(.143)	(.128)	(.128)	(.129)
	T-PRI(mean)	.048**	.038**	.038**	.038**
		(.008)	(.007)	(.007)	(.007)
	Happy (mean)		-.416**	-.415**	-.408**
			(.055)	(.055)	(.055)
Level1*	T-PRI(mean)			-.018**	-.012**
Level2	*Happy (dev)			(.005)	(.002)
Random effects					
Level 1	within person	.413**	.395**	.384**	.389**
		(.030)	(.029)	(.028)	(.028)
Level 2	intercept	.900**	.701**	.705**	.705**
		(.102)	(.083)	(.083)	(.083)
Goodness-of-fit					
	-2LL	1587.58	1523.61	1513.00	1517.84
	AIC	1601.58	1541.61	1533.00	1533.84
	BIC	1632.33	1581.15	1576.94	1569.00

Table 7.64: Moderation of the happiness and anxiety relation by pain

of these relationships in comparison with a baseline (pain only) model. Model 1 indicates a significant interaction between fluctuations of pain and happiness reports, suggesting that the (negative) correlation between anxiety and happiness increases when pain fluctuations increase (or viceversa). Model 2 suggests that including the interaction between mean levels of pain and happiness fluctuations renders non-significant both the previous interaction and the main effect of happiness fluctuations from the mean. Model 3 presents a simplified model with only the latter interaction, which indicates that the happiness and anxiety scores are in fact less associated (negatively) in individuals with higher mean pain levels.

A final set of models enquired the degree to which the correlations between happiness and negative emotions might be moderated by acceptance, ERSs, illness perceptions, pain disability and control variables[72]. The models were structured identically with the previous set of models, the only difference was the specification

[72]The pain-happiness interaction was explored in the previous set of models.

ANXIETY		Baseline	+ CPA
Fixed effects			
Level 1	intercept	6.175**	6.645**
		(.362)	(.357)
	Happy(dev)	-.254**	-.504**
		(.054)	(.150)
	CPA(dev)		-.304**
			(.076)
Level 2	comorbidity	.483**	.363**
		(.133)	(.129)
	Happy (mean)	-.469**	-.300**
		(.057)	(.064)
	CPA(mean)		-.406**
			(.082)
Level1*	Happy(dev)		.125*
Level2	* CPA(mean)		(.059)
Random effects			
Level 1	within person	.412**	.391**
		(.030)	(.028)
Level 2	intercept	.802**	.715**
		(.093)	(.084)
Goodness-of-fit			
	-2LL	1565.03	1522.71
	AIC	1577.03	1540.71
	BIC	1603.39	1580.25

Table 7.65: Moderation of the joy - anxiety relation by CPA

of happiness scores (mean and fluctuations from personal trajectories) instead of pain scores as the main predictor.

Interindividual differences were also identified in strength of association between these emotion diads. Due to the number of possible predictor combinations and the space limitations, only one example related to the moderating effect of CPA is presented here. Table 7.65 shows that mean CPA levels moderated the relation between changes in happiness and anxiety scores, mirroring the findings related to the pain-happiness interaction. Interestingly, anger, sadness and shame scores were not predicted by any interaction between happiness and CPA, which singles out anxiety as a central emotion for the ability to discriminate emotional aspects of living with chronic pain.

7.8 Summary

The analysis of the interrelations between acceptance, emotions and illness perceptions in the chronic pain adjustment was studied empirically on a 3-wave longitudinal survey based on a heterogeneous sample of chronic pain sufferers, selected

from the users of a NHS regional pain clinic and several pain support organisations. The statistical analysis was approached hierarchically, more theoretically complex analyses building on the results of testing more specific hypotheses.

The examination of the separate research areas led to several important results. Both CPA and illness perceptions were characterised by continuous trends, rather than categorical distinctions, as reflected in the low distinctiveness and stability of the clusters identified. CPA and PF showed overall little differences, especially based on the SEM analysis. Dysfunctional ERSs were related to increased emotional distress, while functional ERSs were related to less frequent negative emotions and more frequent happiness/joy. IPs had a heterogeneous structure, suggesting that separate subscale scores are more adequate than total scores. The HSIs also had heterogeneous relationships, reflecting at least 2 distinct aspects, pain experience and pain behaviours. CPA was associated with more frequent positive emotions and functional ERSs, and less frequent negative emotions and dysfunctional ERSs, but item level analyses revealed a less consistent relation to functional ERSs. Also, CPA was associated especially with emotion-related IPs, less consistently with perceptions of timeline, control and identity, and unrelated to perceptions of understanding. The emotion-related content was also the common element in emotion and IP variables.

The exploratory analyses targeted three main issues. First, emotion, IPs and CPA were all related to HSIs, controlling for the (small) influences of contextual factors, as shown by the separate MRMs and by the comparative MRMs. A measurement-focused SEM highlighted the significant relationships (or content overlaps) between the three concepts and two HSIs (pain intensity and pain disability) and the lack of unidimensionality of the measures used, which prevented further analyses at the structural level. As no clinically significant changes were identified overall at group levels, no longitudinal analysis was performed. Nevertheless, the second issue addressed was separating intra- and interindividual variance based on HLM analyses, which identified significant proportions of variance at both levels. HLM also revealed minor (albeit statistically significant) change trends at group level only for CPA, anger, sadness, disability and IPs of consequences, and differences between individual change trajectories only for T-PRI, despite stability at group level. A third set of analyses revealed moderation effects of CPA, selected emotions, ERSs and IPs for the relation between sensory and affective pain, and also moderation effects (of opposite sign) of CPA and T-PRI for the relation between happiness and anxiety.

Chapter 8

Analysis of the psychometric properties of several questionnaires in the context of chronic pain assessment

8.1 Introduction

This chapter presents a psychometric analysis of the questionnaires used in this study. This effort might at first seem unnecessary for a study that focuses on substantive issues rather than on measurement, therefore the reasons for such an analysis are detailed first. Each of the questionnaires is then examined based on a set of common criteria and where necessary on specific issues related to the particular theory and concept.

8.2 Why is a psychometric analysis necessary?

Although the evaluation of the psychometric properties of questionnaires for the study sample is rarely reported in the literature (Meier and Davis, 1990; PingJr, 2003), there are several reasons that recommend a thorough analysis prior to substantive theory testing.

First, scale reliability and validity are among the assumptions that justify the substantive inferences made based on the analysis of a dataset, therefore they can be regarded as auxiliary hypotheses amenable to testing (Meier and Davis, 1990). Reliability is not an intrinsic property of the scale, but rather of the specific use of the scale in the population represented by the study sample (Borsboom, 2005). From this perspective, reporting psychometric properties for the study sample in addition to reliability and validity estimates published by questionnaire developers

is recommended, both for providing sufficient information to readers for assessing the soundness of measurement and subsequent inferences and for offering an opportunity to correct any errors in the early stages (PingJr, 2003). Considering the psychometric support for the instruments used is recommended in ensuring the reliability and validity of any chronic pain taxonomy (Bonica, 2001, Ch. 2), especially given issues of criterion contamination present in chronic pain in comparison to the populations many of the measures were initially developed on (Pincus and Williams, 1999).

Second, some questionnaires have never been used in a chronic pain sample or adult sample before, and this dataset allows the testing of these questionnaires in this new context. Testing reliability is especially recommended if the study sample is sufficiently different from previous samples regarding various characteristics including homogeneity (Streiner, 2003). Third, some questionnaires (i.e. the Short Form McGill Pain Questionnaire) had unexpected response patterns and further analysis is an opportunity to understand to what extent the different patterns influence the total scores. Fourth, participants' comments on response forms on some items (i.e. items of the Sickness Impact Profile Roland Scale) revealed the need to reconsider these items and understand their impact on the overall score.

Fifth, and most important, it is necessary to ensure that the items and total scores are reflecting accurately the concepts, as conceptual relationships and differences are a central issue of the analysis and its interpretation. All concepts are particularly difficult to measure, and the inferences related to their associations need to take into account measurement issues. To a certain extent, the analysis of the interrelationships between emotions, emotion regulation, acceptance, illness perceptions and health related outcomes is also an analysis of the validity of the measurements used, which informs the critical analysis of the constructs used in describing the complex reality of living with chronic pain. Questionnaire analysis is one aspect of the necessary (but often neglected) role of instrument refinement in the ongoing process of theory development and increasing construct validity (Smith and McCarthy, 1995).

General guidelines for questionnaire evaluation refer to reporting means and standard deviations, sample reliability, and published estimates of reliability and convergent and discriminant validity (Meier and Davis, 1990; Wilkinson and the Task Force on Statistical Inference, 1999). Other authors recommend an even more detailed reporting, which includes unidimensionality, reliability, averaged variance extracted (AVE) as an indicator of the measurement error involved (similar to the

total variance extracted for a unidimensional scale), full matrix of correlations, together with conceptual definitions, items and measure development details for each instrument used, and in case of using structural equation models a detailed goodness-of-fit and parameter estimates reporting on a full measurement model (PingJr, 2003).

However the criteria for evaluating the psychometric properties of a questionnaire are controversial. From a general perspective, three main theoretical frameworks for test construction are available and each has different implications and applications in test evaluation, depending on the type of approach to measurement and theoretical assumptions related to the construct of interest: classical test theory (CTT), item response theory (IRT), and factor analytic tradition (both EFA and CFA; Nunnally and Bernstein, 1994). A detailed analysis of the strengths and limitations of these three frameworks and the related controversies is beyond the scope of this chapter. Suffice it to say that most instruments used in chronic pain research have been developed based on CTT (focusing mainly on ensuring internal consistency and testing convergent and discriminant validity in relation to relevant constructs), while newer validation efforts have adopted the factor analytic framework (focusing on ensuring structural validity via the use of EFA and more recently CFA). The use of CFA in assessing existing scales however often leads to suggestions for scale revision instead of support of their validity, as the strict criteria of CFA are usually introducing additional demands on scales built based on looser criteria (MacCallum and Austin, 2000). IRT has been seldom used in this research domain (e.g. Stroud et al., 2004).

Smith and McCarthy (1995) advise a few general criteria for test refinement (whether related to the process of test construction or ulterior changes): identification of hierarchical or aggregational structure, "establishment of internal consistency of unidimensional facets", "determination of content homogeneity of unidimensional facets", "inclusion of items that discriminate at the desired level of attribute intensity", replication on a new sample (p. 300). While determining content homogeneity by multiple raters and replicating the test modifications on a new sample are not possible in the present study due to the substantive focus of the study (as opposed to a measurement refinement focus), it was considered appropriate to aim for testing the structural properties, facet consistency and item discrimination properties.

The method and extent of evaluation also depends on several issues: whether constructs are homogeneous or heterogeneous, the response format, the relationship

between the current sample and previous questionnaire validation samples, the extent of prior evaluation by the questionnaire developers. Therefore in the present analysis each questionnaire was evaluated on some common criteria (good item and scale distributions, published estimates of reliability and validity, sample reliability, and scale structure) as well as specific criteria depending on the questionnaire's unique characteristics, if applicable.

In case the psychometric properties are not supported by current data, limited guidelines are given in the literature. Crocker and Algina (1986, p. 327) discuss the issue of item analysis in test revision and suggest that, in case the individual scores are not of interest by themselves and if enough good items are available for a meaningful content of the final questionnaire, the flawed items may be discarded; item revisions should be considered in this case. PingJr (2003) advises choosing between the available methods depending on various criteria: "the final itemization of a measure can be a trade off among consistency/unidimensionality, reliability, AVE and content and face validity" (PingJr, 2003, p. 132).

While these reporting requirements are difficult to comply with in journal articles due to space constraints, a detailed analysis of the instruments is nevertheless preferable. Summaries of the psychometric properties of the questionnaires used in the present study and related decisions are provided in the next sections (missing data analyses are presented in Appendix C).

8.3 Acceptance and Action Questionnaire

AAQ aims to measure experiential avoidance (Hayes et al., 2004a), as described in Chapter 3. Given the early stages of theory development in which this questionnaire version was developed, the estimates of internal consistency (Cronbach's $\alpha = .70$) and test-retest reliability ($r = .64$) reported by Hayes et al. (2004a) can be considered a good performance, although at the lower limit of the acceptable range (.70 to .80 for basic research, according to Nunnally and Bernstein, 1994). The items were selected from a pool of 32 items via iterative exploratory factor analysis (EFA) using SEM and tested via CFA on a new sample. Distribution properties for the selected items were not reported. The 1-factor CFA model for the 9 items was acceptable ($\chi^2(27) = 47.61$, $p = .0085$; GFI= .98; AGFI= .97; RMR=.054; range of item loadings .26 to .64). The authors reported low correlations ($r < .40$) with thought suppression, thought control, dissociative experiences, post-traumatic stress, impact of events as proof of discriminant validity and moderate to high correlations

Item no.	1	2	3	4	5	6	7	8	9	total
stage 1	4.9 (1.3)	4.2 (1.3)	3.6 (1.7)	3.9 (1.7)	4.7 (1.7)	4.6 (1.4)	4.0 (1.7)	4.6 (1.5)	4.8 (2.0)	3.9 (.9)
stage 2	4.9 (1.3)	4.2 (1.2)	3.6 (1.6)	3.9 (1.7)	4.8 (1.6)	4.5) (1.4)	4.3 (1.7)	4.7 (1.6)	4.8 (2.0)	3.9 (.9)
stage 3	4.9 (1.3)	4.1 (1.2)	3.5 (1.6)	4.0 (1.6)	4.8 (1.6)	4.6 (1.3)	4.1 (1.6)	4.5 (1.5)	4.5 (2.0)	3.8 (.9)

Table 8.1: Means (and standard deviations) for AAQ items and total score (possible range 1-7)

with measures of psychopathology to indicate concurrent validity. Several caveats were mentioned. First, the conceptualisation of psychological flexibility (experiential avoidance) as an underlying trait does not conform to contextual behavioral theory which describes this concept in terms of situated action (which might explain the lower test-retest reliability). Second, AAQ is a broad measure adequate for initial exploration of the domain, and additional scale development efforts are recommended in conjunction to theory refinement, as well as the development of situation-specific measures that would be more sensitive to context-related changes. Third, it is difficult for a self-report instrument to measure reliably a phenomenon that deals with issues of language entanglement, and behavioral measures need to be further developed.

In the present sample, inspection of item and scale distributions indicated good discrimination properties (means and standard deviations are provided in Table 8.1). The measure showed slightly better internal consistency (Cronbach's $\alpha = .76 - .78$ in the three stages) and test-retest reliability ($r = .76 - .72$ in intervals of 4 months to 8 months).

The 1-factor CFA model for the 9 items in the present sample was less acceptable. An EFA (PCA, oblimin rotation) indicated a 2-factor solution explaining 50.7% of the item variance, the second factor actually grouping the reversed items. Allowing reversed items to covary in a second 1-factor CFA model (Figure 8.1) led to a significant improvement in fit, although χ^2 reached non-significance only in one of the stages. Goodness of fit statistics for both models are presented in Table 8.2 (covariance matrix in Table D.2). Additional diagnostics (W and LM tests, residuals) suggested the existence of additional associations between some positively worded items, and weaker link between item 1 and the rest of the negatively worded items (which are also consecutive). Thus, even if the 1-factor solution is a parsimonious

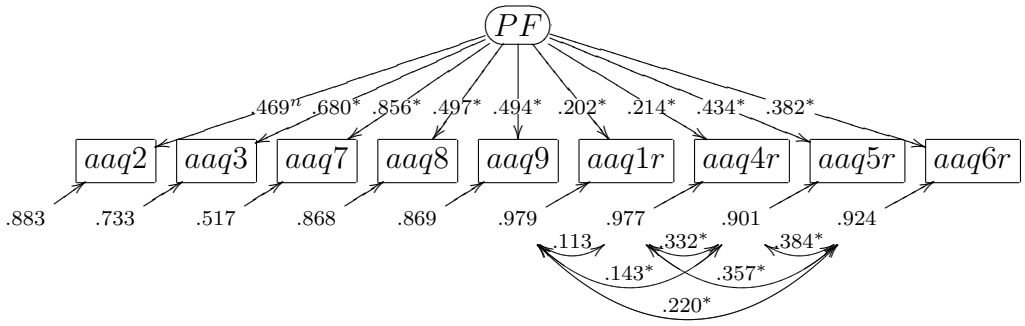


Figure 8.1: CFA model AAQ

	Model 1			Model 2		
Statistic	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
S-B χ^2 (df)	121(27) p<.001	76(27) p<.001	95(27) p<.001	57(21) p<.001	30(21) p=.08	42(21) p=.003
NFI robust	.73	.81	.75	.87	.92	.89
NNFI robust	.69	.82	.74	.85	.96	.90
CFI robust	.77	.87	.80	.91	.98	.94
RMSEA robust	.12 (.09-.14)	.09 (.06-.11)	.11 (.0-.13)	.08 (.06-.11)	.05 (.00-.08)	.07 (.04-.10)
range of item loadings	.25-.81	.34-.71	.34-.70	.20-.86	.24-.79	.34-.78
S-B χ^2 diff.	-	-	-	64, p<.001	46, p<.001	53, p<.001

Table 8.2: Goodness of fit statistics for AAQ models

model with adequate fit to the data, this additional information indicated the necessity of pursuing further research on the conceptual structure of PF, and on the wording of AAQ items.

These issues need to be taken into account in further SEM analyses and in the interpretation of the total score of the questionnaire. However, given the theoretical difficulties of defining psychological flexibility, AAQ scores do represent a useful approximation of an individual's level of flexibility until further theoretical developments lead to clearer concepts definitions and measurement tools.

8.4 Basic Emotions Scale

BES focuses on measuring self-reported frequency of experiencing five discrete emotions: anger, sadness, disgust, fear and happiness (Power, 2006). It has been developed based on a previous semantic analysis of emotion terms (Oatley and Johnson-Laird, 1987) which brought support to the proposal for five discrete emotions as

basic communicative signals[1]. As discussed in Chapter 4, basic emotions theories seem to converge on the issue of the adaptive value of categorising emotional experience via emotion terms, although the development and functioning of this categorisation is controversial.

The BES development consisted in selecting 30 items from the corpus used by Oatley and Johnson-Laird (1987) on theoretical grounds and applying them on a sample of 219 students. The items had satisfactory distribution properties. Internal consistency and related analyses were performed to reduce the number of items and improve scale reliabilities (resulting Cronbach's $\alpha = .81$ for anger, .84 for sadness, .84 for disgust, .89 for fear and .83 for happiness). Due to the cross-sectional design, test-retest reliability was not addressed. The remaining items were subject to CFA, where 6 alternative models were compared: 1-factor ('emotionality'), 2 independent factors (negative versus positive emotions), 2 correlated factors, 5 independent factors (basic emotions), 5 factors with an additional second order factor grouping the negative emotion latents, and finally 5 factors with an additional second order factor grouping all emotion latents. The last CFA model showed the best model fit statistics[2] ($\chi^2(162)=340.92$, $p < .001$ NNFI=.899; CFI=.912; AIC=16.92), a range of item loadings from .58 to .86 and a significant improvement from the previous model (difference $\chi^2(1)=24.44$, $p < .001$). The discriminant validity or concurrent validity of BES have not yet been studied. Power (2006) noted some limitations (the incapacity of retrospective self-report to distinguish the dynamic aspects of the emotional experience, the potential empirical or theoretical coupling of emotion terms, and the lower suitability of state-like ratings) and encouraged cross-validation on new data sets.

In the present sample, inspection of item and scale distributions indicated problematic discrimination properties only for 3 emotion terms: shame, humiliated and disgust[3] (means and standard deviations are provided in Table 8.3). The subscales showed adequate distributions, with the exception of the disgust/shame subscale (Table 8.4) and adequate internal consistency and test-retest reliability (Table 8.5).

[1]The analysis consisted in identifying one of the five emotions as a basic emotional component for all words from a corpus of 590 emotion terms in English, excluding generic emotional terms and a few exceptions. Basic (or primitive), in this context, refers to the linguistic property of lacking an internal structure (i.e. a set of distinct semantic components).

[2]Similar results were reported for BES including all initial items, and for state-like 'past week' ratings.

[3]It can be hypothesised that reporting low levels of shame in the responses to these terms was related to their particular linguistic use in this sample. Guilt and blameworthy were slightly more endorsed.

Item no.	1	2	3	4	5	6	7
stage 1	4.2 (1.6)	3.9 (1.8)	2.8 (1.7)	4.5 (1.6)	4.8 (1.3)	5.4 (1.4)	3.7 (1.8)
stage 2	4.1 (1.5)	3.8 (1.8)	2.8 (1.8)	4.7 (1.5)	4.7 (1.4)	5.2 (1.4)	3.7 (1.9)
stage 3	4.0 (1.6)	3.6 (1.8)	2.7 (1.8)	4.6 (1.5)	4.9 (1.4)	5.0 (1.4)	3.4 (1.8)
Item no.	8	9	10	11	12	13	14
stage 1	3.4 (1.8)	4.4 (1.7)	4.6 (1.4)	4.8 (1.4)	3.7 (1.7)	2.6 (1.7)	4.8 (1.5)
stage 2	3.2 (1.9)	4.3 (1.6)	4.5 (1.5)	4.8 (1.4)	3.8 (1.6)	2.7 (1.6)	4.8 (1.4)
stage 3	3.1 (1.7)	4.4 (1.6)	4.6 (1.4)	4.7 (1.4)	3.5 (1.5)	2.7 (1.7)	4.8 (1.4)
Item no.	15	16	17	18	19	20	21
stage 1	5.2 (1.4)	3.3 (1.9)	3.2 (1.7)	3.1 (1.8)	4.8 (1.5)	4.8 (1.4)	2.7 (1.7)
stage 2	5.0 (1.5)	3.2 (1.9)	3.2 (1.8)	3.0 (1.7)	4.8 (1.5)	4.7 (1.4)	2.7 (1.8)
stage 3	5.1 (1.4)	3.0 (1.8)	3.0 (1.7)	3.0 (1.7)	4.7 (1.5)	4.7 (1.4)	2.5 (1.7)

Table 8.3: Means (and standard deviations) for BES items and total score (possible range 1-7). Items order: anger, despair, shame, anxiety, happiness, frustration, misery, guilt, nervousness, joy, irritation, gloominess, humiliated, tense, loving, aggression, mournful, blameworthy, worried, cheerful, disgust (i.e. repulsion).

	anger	sadness	shame	anxiety	happiness
Stage 1	4.4 (1.3)	3.6 (1.5)	2.9 (1.4)	4.6 (1.3)	4.9 (1.2)
Stage 2	4.3 (1.2)	3.6 (1.5)	2.9 (1.5)	4.6 (1.2)	4.7 (1.3)
Stage 3	4.2 (1.2)	3.4 (1.5)	2.8 (1.5)	4.6 (1.3)	4.8 (1.3)

Table 8.4: Means (and standard deviations) for BES total scores (possible range 1-7)

	Cronbach's α			Stability		
Stages	1	2	3	1 to 2	2 to 3	1 to 3
Anger	.82	.79	.81	.80	.77	.73
Sadness	.87	.88	.89	.80	.80	.76
Shame	.88	.91	.91	.78	.78	.75
Anxiety	.85	.85	.89	.75	.71	.75
Happiness	.90	.92	.92	.76	.81	.76

Table 8.5: Internal consistency and stability for BES subscales

Statistic	Stage 1	Stage 2	Stage 3
S-B χ^2 (df=184)	346.0 $p < .001$	329.2 $p < .001$	272.4 $p < .001$
NFI robust	.88	.88	.90
NNFI robust	.93	.93	.96
CFI robust	.94	.94	.97
Robust RMSEA	.06 (.05-.07)	.06 (.05-.08)	.05 (.04-.06)

Table 8.6: Goodness of fit statistics for BES model

A CFA model for the 21 BES items specifying 5 basic emotion factors and one second order emotionality factor (Figure 8.2) showed acceptable, but not optimal fit (goodness of fit statistics are presented in Table 8.6, covariance matrix in Table D.6)[4]. Loadings on the second order factor ranged from $.95 - .97$ for sadness to $-.66 - -.70$ for happiness, indicating that this latent represents in essence 'negative emotionality', or perhaps degree of emotional distress[5]. This result contrasts with the model in Power (2006), where happiness had a positive loading (.39) on the emotionality factor, which indicates differences between a healthy adolescent sample and a sample of adults suffering from chronic pain. Model modifications suggested by the Lagrange Multiplier (LM) test indicated various emotion terms as being influenced by other emotion latents (such as 'frustration' or 'irritation' responses being related to anxiety, or 'mournful' responses related to shame). This is potentially indicating idiosyncratic uses of these emotion terms in the present sample, or the difficulties of labelling a particular emotional experience as strictly related to only a particular basic emotion[6].

Due to the lack of optimal fit, EFAs (PCA, oblimin rotation) were performed in order to search for alternative models. The EFAs extracted 4 factors with eigenvalues greater than 1, explaining 45–49%, 9%, 7–8% and 6–7% of the item variance. In the 4-factor rotated EFA solutions, shame, happiness, anxiety and anger items formed distinct factors, while the sadness-related emotion terms loaded in various degrees on all negative emotion factors but mostly on anger. The 5-factor rotated EFA solutions, sadness represented a distinct factor only in the last stage. In the first two stages, the anger items separated in 2 distinct factors (anger/ aggression versus frustration/ irritation), while the sadness terms loaded mostly on the frustration

[4]The 'disgust' item loaded appropriately on the shame latent ($.71 - .80$).

[5]The loadings for the other three emotion latents were $.82 - .85$ for anger, $.71 - .83$ for anxiety and $.71 - .78$ for shame.

[6]As was the case with 'disgust' understood as anger by the adolescent sample in Power (2006), 'irritation' (for example) for a chronic pain sufferer might be used in some situations to describe states for which labels such as 'nervousness' and 'anxiety' are synonyms, while in other situation its use would be a synonym of 'anger', and in some situation one might experience both anxiety and anger.

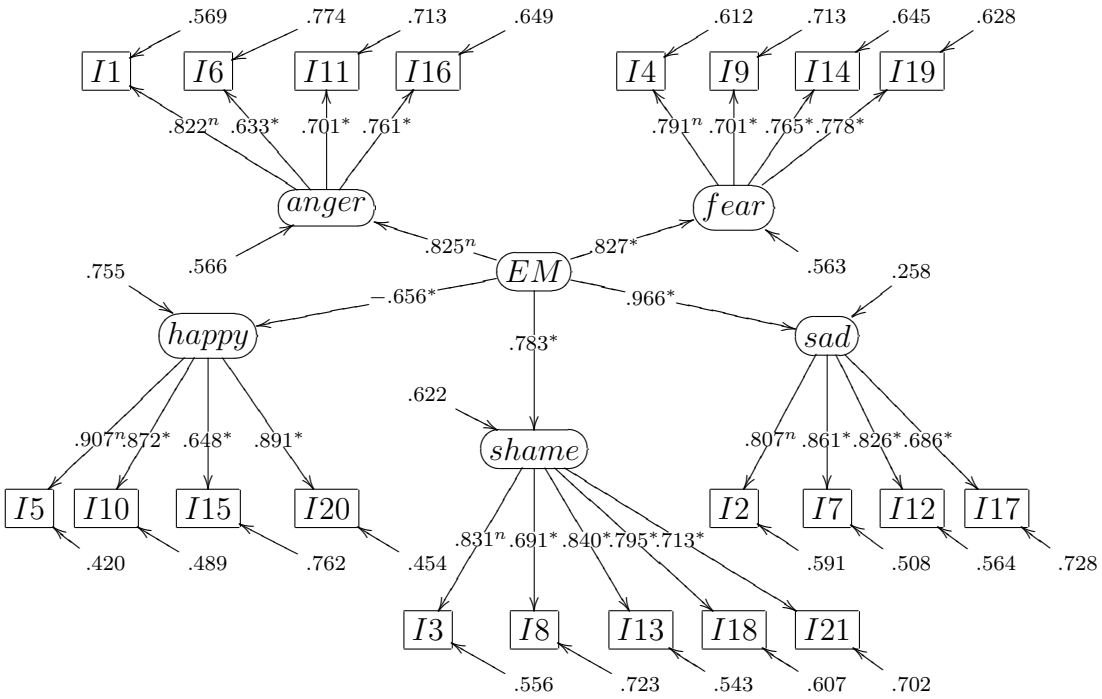


Figure 8.2: CFA model BES

factor. These results might indicate a coupling of sadness and anger (especially the 'anger-in' aspects) which is compatible with the SPAARS description of the process of anger generation via appraisal (Power and Dalgleish, 2008, p. 277–79)[7].

Attempts at model modification based on the LM test and on the EFA results did not lead to an optimal fit (i.e. nonsignificant χ^2), even if adding specifications such as error covariances for 'anger-in' and 'anger-out' items resulted in significantly improved fit (difference $\chi^2(2) = 45 - 10$, $p < .01$). Given that any alternative model might capitalise on sample variations and not be comparable across populations, the lack of the additional advantage of accurate description of the present sample recommends the adoption of the theory-based 5 correlated factors model in the present sample.

However, even if a correlated basic emotions model represents an acceptable and parsimonious approximation of the structure of the questionnaire, these results suggest it also masks to a certain degree the complexity emotional experience in chronic

[7]The emotional component of pain and the related disability can be processed via appraisals of goal incompatibility, and of goal thwarting by a perceived agent (either pain itself or the event that caused the pain onset, or medical care). But together with the appraisal of unavoidability, this processing may lead to blocking of anger associated with feelings of sadness.

pain (as detailed in Chapter 4). Therefore, while the subscale scores are undoubtedly useful in describing emotional life in the present study, the interpretation of any relationships with other concepts needs to consider this limitation of using emotion terms in self-report.

8.5 Regulation of Emotion Questionnaire

REQ was developed to assess emotion regulation strategies, defined as "processes involved in recognizing, monitoring, evaluating and modifying emotional reactions" (Phillips and Power, 2007, p. 145). Based on a review of the literature on emotion regulation, the authors have identified four categories of strategies: internal dysfunctional (ID; rejection of emotion via inhibition and the use of internal resources), external dysfunctional (ED; rejection of emotion via emotional expression or using other people and objects), internal functional (IF; acceptance of emotion via positive reappraisal, concentration, learning, etc.), and external functional (EF; acceptance of emotion via action or using social support). The theoretical stance behind REQ stipulates that tendencies towards using particular strategies might result in a general functional or dysfunctional style which impacts on mental and physical health, even if a specific strategy can be in itself adequate to particular situations and inappropriate in others (although the opposite causal direction could also be hypothesized, i.e. increased mental and physical distress can lead to increased use of particular strategies). A pool of 32 statements was selected based on existing literature and expert consensus on item classification according to the 4 categories and applied on a sample of 225 adolescents. Based on descriptive statistics, reliability analyses and EFA, 19 items were further selected and subject to a CFA on the same sample, which the authors reported as showing an acceptable fit (CFI= .91[8]). Estimates of internal consistency ranged from $\alpha = .76$ for IF and ED to $\alpha = .72$ for ID and $\alpha = .66$ for EF. Test-retest reliability could not be estimated in this initial study due to the cross-sectional design. Discriminant and convergent validity was supported by confirmation of expected correlations between the 4 subscales and other measures of emotional functioning (emotional and behavioral problems, psychological and somatic symptoms reporting, quality of life). The authors noted the necessity to further improve the questionnaire (especially the EF subscale) and the limitations related to the use of self-report in measuring emotion regulation, as many strategies are unconscious.

[8]No other fit statistics were reported, except average absolute standardized residuals (.05).

Item no.	1	2	3	4	5	6	7
Mean	2.7	2.4	2.7	2.9	1.4	2.4	3.0
(SD)	(1.0)	(1.0)	(1.2)	(.9)	(.7)	(1.1)	(1.2)
Item no.	8	9	10	11	12	13	14
Mean	2.6	2.9	1.3	3.1	3.0	1.4	2.0
(SD)	(1.0)	(.9)	(.7)	(.9)	(1.0)	(.7)	(1.0)
Item no.	15	16	17	18	19	20	21
Mean	3.0	3.0	1.2	1.3	2.1	2.9	2.7
(SD)	(1.2)	(.9)	(.5)	(.7)	(1.0)	(1.2)	(1.0)

Table 8.7: Means (and standard deviations) for REQ items and total score (possible range 1-5)

Subscale	Stage no.	ID	IF	ED	EF
Mean	all	2.3	2.9 - 3.0	1.5	2.6 - 2.7
(SD)		(.7)	(.6)	(.5)	(.7)
Cronbach's α	1	.75	.71	.73	.75
	2	.75	.72	.74	.78
	3	.77	.70	.77	.77
Stability	1 to 2	.74	.71	.72	.78
	2 to 3	.78	.65	.77	.84
	1 to 3	.76	.68	.72	.76

Table 8.8: Means, standard deviations, internal consistency and stability for REQ original subscale scores

Given that REQ includes strategies employed by the general population, that one of the subscales had lower reliability and that the questionnaire was validated only on an adolescent sample, it was necessary to explore the psychometric properties in the present sample. Item means and standard deviations are provided in Table 8.7 for stage 1 (there were no notable differences between stages). Distributions were highly skewed[9] in all three stages for 2 ID items (few chronic pain sufferers harm or punish themselves, or report things feeling unreal) and 4 ED items (fighting, being rude, bullying people or damaging objects were reported as infrequent events), indicating insufficient capacity to discriminate between chronic pain patients. Consequently, the ED subscale had a skewed distribution. Nevertheless, internal consistency and stability estimates were almost all above the acceptability threshold of .70 (Table 8.8[10]).

The CFA model of the original 19 item scale showed poor fit ($\chi^2(150) = 478$, $p < .001$; GFI= .84; AGFI= .80; RMR= .12; NFI= .68; NNFI= .72; CFI= .75, RMSEA= $.09(.08 - .10)$, range of item loadings .17 to .84 for stage 1), as did

[9]According to the criterion of less than 10% responses in two or more adjacent scale points, used by (Phillips and Power, 2007).

[10]The EF subscale scores are based on 6 items.

Subscale	Item
ID	7. I dwell on my thoughts and feelings
IF	9. I review (rethink) my goals or plans
	16. I plan what I could do better next time
ED	2. I take my feelings out on others verbally
EF	1. I talk to someone about how I feel
	3. I seek physical contact from friends or family
	8. I ask others for advice

Table 8.9: Best items for REQ subscales

the model based on 21 items ($\chi^2(187) = 608$, $p < .001$; GFI= .82; AGFI= .77; RMR= .13; NFI= .67; NNFI= .71; CFI= .74, RMSEA= $.09(.08 - .10)$, range of item loadings .25 to .75 for stage 1, Figure 8.3, covariance matrix in Table D.7). Additional diagnostics (LM, residuals) indicated multiple cross-loadings between items of different subscales, as well as significant increase in fit if opposite subscales were allowed to covary freely (IF and ED, EF and ID), suggesting a more complex structure of REQ. EFAs (PCA, varimax rotation) based on all 21 items indicated 4 factors[11], explaining 26–27%, 10–12%, 8–10% and 7–8% of the item variance. Several items loaded on different subscales[12], indicating that the subscales are not unidimensional.

Excluding all problematic items from further analyses was not possible due to the limited number of remaining items. Therefore, it was considered that even if a 4-factor solution did not fully represent the structure of the questionnaire, the analyses would need to be performed comparatively with subscale scores and best items (see Table 8.9). Due to the problematic psychometric properties of REQ in this sample, it is not certain which emotion regulation strategies are most relevant for chronic pain sufferers and any conclusions would have to be replicated using a more tailored measure.

8.6 List of Threatening Experiences

LTE is a measure of contextual threat (external stress or adversity) initially developed to assess external events aetiologically related to the onset of affective disorder. The authors recommended its use also in other populations for assessing the impact of adversity on emotional life, and noted that its brevity makes it suitable for

[11] 5 factors had eigenvalues above 1, but examination of the scree plots indicated that only the first 4 factors were distinct

[12] For example, doing something energetic (item 6) was more related to the IF scale not its original EF scale, while going out to do something nice (item 21) was related comparably to ID, IF, and its original EF scale.

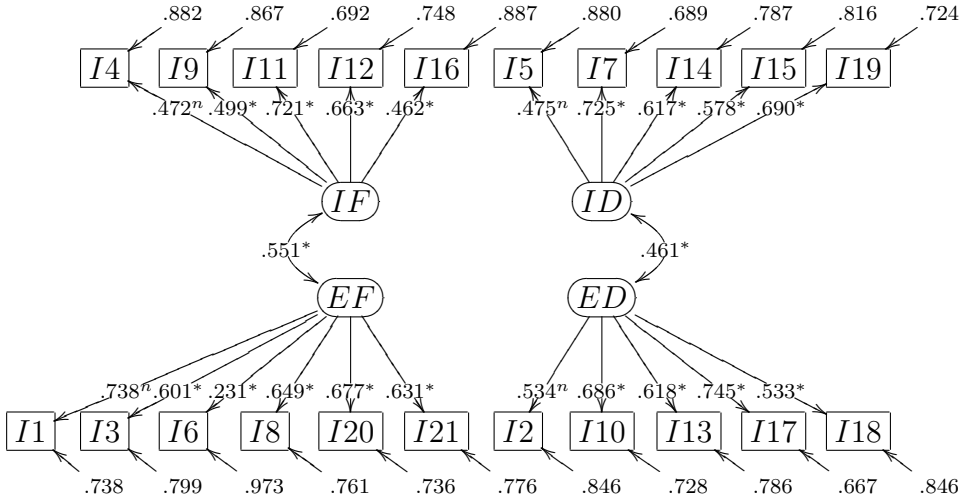


Figure 8.3: CFA model REQ

studies with multiple measures, in comparison with other lists available (Brugha and Cragg, 1990). It was developed based on interviews with both a sample from the general population and psychiatric outpatients with affective disorders by categorising events and rating them according to an earlier method (Brown and Harris, 1978). To eliminate bias due to post-hoc self-rating of the impact of an event and to lack of contextual information, Brown and Harris (1978) obtained ratings of the degree of short-term and long-term threat or unpleasantness from independent judges who were provided extensive background information about the event, excluding the individual's subjective reaction. The marked or moderate long-term threat ratings proved to be associated with increases in the likelihood of depression onset. Thus, another advantage of LTE is the proven relevance of the events for emotional distress. In a study of 50 psychiatric patients (Brugha and Cragg, 1990), test-retest reliability for the items ranged from .78 to 1 (with the exception of one item with low reliability which refers to stolen or lost valued property). In this sample, frequency of events ranged from 18 (unemployment) to 0 (death of a family member). Agreement between the ratings of the patient and those of a named informant (relative, friend, or confidant) ranged from .66 to .84. The questionnaire ratings were consistent with reports during an accompanying interview based on a similar structure (specificity and sensitivity were .74 and .89 for 6 months and .88 and 1.0 for 3 months respectively). The subjective report of long-term contextual threat assessed in the interview was also highly consistent with independent ratings

	Stage (interval)		
	1 (6 months)	2 (3-4 months)	3 (3-4 months)
Injury - self	26	18	28
Injury - family	29	22	28
Death - family	8	6	8
Death - close friend	26	24	19
Separation - marital	5	4	3
Breaking off - other	6	4	4
Relationship problem	19	19	16
Unemployment	7	6	6
Fired from job	2	2	2
Financial crisis	16	16	14
Problem - police	1	<.5	1
Stolen/lost property	5	2	6
At least one negative event	72	66	67
New/better job	6	6	6
Improved financial status	21	16	16
Birth of a child	24	17	19
Marriage - family	15	9	8
Relationship improvement	5	8	5
At least one positive event	52	43	40

Table 8.10: Percentage of reported occurrence for events in the LTE

of long-term threat based on contextual information (range $.63 - .90$). As the questionnaire assesses events of relatively rare occurrence, no distribution or structural properties would represent a validity test. To obtain a global indicator of adversity, Brugha and Cragg (1990) computed a dichotomous variable to differentiate participants that report at least one such event from those that report none.

In the present sample, most frequently reported threatening events were illness, injury or assault to the person or to close relatives, death of a close friend or relative, serious problems with a close friend, and financial problems. Most frequent positive events were improved financial status, and birth of a child. Percentages of reported occurrence for the events at each stage are presented in Table 8.10.

The high frequency of these events is notable. It reflects the significant amount of contextual stress that chronic pain sufferers face. Only part of it, such as health, interpersonal and financial issues are likely to be related to the condition, some are probably related to age (death or illness of family or close friends).

Reports from one stage were significantly[13] but moderately related with reports from other stages, indicating that probably some events lasted for longer periods, some respondents had experienced more similar events at short intervals and some respondents located events from one stage also in the time period of the next scale

[13]According to χ^2 tests, not reported.

	Stages		
	1 and 2	1 and 3	2 and 3
Injury - self	.17	.29	.38
Injury - family	.28	.41	.24
Death - close friend	.25	.12	.19
Relationship problem	.49	.47	.49
Financial crisis	.43	.51	.51
Improved financial status	.29	.16	.18
Birth of a child	.26	.23	.15

Table 8.11: Pearson correlations between stages for the most frequent events in the LTE

	Negative events			Positive events		
Stage	1	2	3	1	2	3
Median	1	1	1	1	0	0
SD	1.58	1.46	1.54	.93	.86	.91
Maximum	7	8	6	4	3	5

Table 8.12: Total LTE scores - descriptive statistics (possible range 0-12 for negative and 0-5 for positive events, excluding "other" responses)

(Table 8.11). As the first interpretations were realistic given the type of events, the third possibility does not pose major problems for the reliability of the reports.

The most frequent events were analysed separately to judge their impact on other psychological and health status aspects. This was especially important for the positive events that were newly introduced in the questionnaire. All the other events had a rare occurrence (and thus uneven distributions) and were not analysed separately. The degree of external stress was analysed both as dichotomous variables (as presented above) and by total scores (sum of events) in each stage, to take advantage of the quantitative information (Table 8.12).

8.7 Self-Administered Comorbidity Questionnaire

The SACQ measures the self-reported extent and severity of comorbid conditions (12 labels provided, with additional spaces for other conditions). On a sample of 170 patients admitted to general medical or surgical units, test-retest reliability was reported as .81 (ranging from .40 to above .90 for individual items), high agreement with a standard, chart abstraction-based measure ($.78 - .99$), medium to large correlations with medication use ($.32 - .57$), and good predictive validity in relation to several indicators of health status at 1-year follow-up (Sangha et al., 2003). As with LTE, due to low occurrence of some medical conditions, no structural and distributional properties of SACQ can be considered a test of its validity.

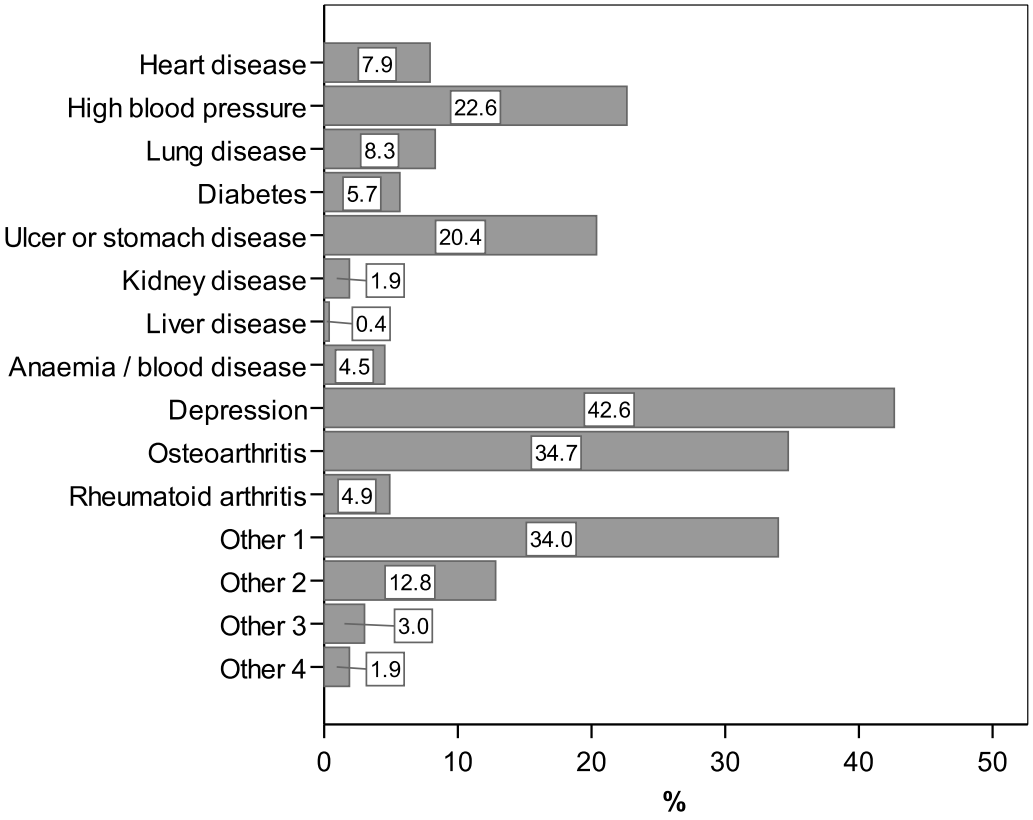


Figure 8.4: Comorbid conditions - percentages

In the present sample, the most frequent conditions were depression and osteoarthritis (see Figure 8.4). The medical conditions reported in the 'other' categories were asthma, irritable bowel syndrome and various other immune, circulatory, urinary or dermatological conditions. Most respondents (82%) reported one or more conditions. Total comorbidity scores were relatively low (median=4; SD=3.96; maximum=23 from a possible range 0-45). To improve distribution properties, total score was dichotomised by median split. Due to the high prevalence, depression and osteoarthritis were analysed separately to assess their impact on psychological and physical health.

8.8 Short Form McGill Pain Questionnaire

SF-MPQ was developed as a short measure of pain intensity and sensory and affective quality, based on the 78-item MPQ (described in Chapter 2). It includes 15 most commonly used sensory and affective terms, in addition to a Present Pain Intensity (PPI) scale and a Visual Analog Scale (VAS). SF-MPQ indices were reported to show high correlations with the original MPQ indices (range $.65 - .94$) and good

sensitivity to reductions in pain due to treatment in three samples (Melzack, 1987) and there is partial support for their capacity to discriminate between different pain syndromes (Melzack and Katz, 2001). It has been used for the assessment of various types of pain (Wright et al., 2001) and has been suggested to be adequate for use in elderly populations (Gagliese and Melzack, 1997) and to have good stability over a period of 4-5 weeks (Groenblad et al., 1990).

A 2-factor model of the pain descriptors (CFA) tested on the English version of SF-MPQ on a sample of chronic back pain patients showed a suboptimal fit (S-B $\chi^2(84) = 191.78$, $p < .001$; CFI-R= .86; AGFI= .81; SRMR= .08; RMSEA=.08) and a modified model (one sensory item moved to the affective scale, and 4 sets of error terms allowed to covary) provided a significantly better, but still suboptimal, fit (S-B $\chi^2(84) = 128.16$, $p < .001$; CFI-R= .94; AGFI= .87; SRMR= .06; RMSEA= .05; Wright et al., 2001). This study reported estimates of internal consistency of .78 and .76 for the sensory and affective subscales and item loadings from .30 to .80.

In another sample of patients undergoing lumbar magnetic resonance imaging for low back pain (Beattie et al., 2004), an EFA resulted in a 2-factor modified version of SF-MPQ: sensory and affective-sensory, with 3 and 5 items, respectively (only items with loadings greater than .50 were selected). This version had a better fit to the data in a new confirmatory sample of the same population ($\chi^2(19) = 22.0$, $p = .29$; CFI= .99; NNFI= .99; AGFI= .95; RMR= .05; RMSEA= .03) than the original Melzack model ($\chi^2(89) = 285.9$, $p < .001$; CFI= .83; NNFI= .80; AGFI= .85; RMR= .09; RMSEA= .08) and the Wright et al. (2001) model ($\chi^2(19) = 22.0$, $p < .001$; CFI= .96; NNFI= .95; AGFI=.94; RMR=.06; RMSEA= .04). Estimates of internal consistency for the sensory and affective subscales were in this study .70 and .73 for the original SF-MPQ and .75 and .77 for the modified version. While it can be argued that both Wright et al.'s (2001) and Beattie et al.'s (2004) models capitalise on sample fluctuations to improve fit of the modified versions, these studies expose the difficulties of confirming the original structure of the questionnaire on two separate samples (despite the similar chronic pain conditions) and justify the search for a better version and the evaluation of the questionnaire properties on any new sample, including the present one.

Questions have been raised regarding the suitability of factor analysis (FA) for testing the structure of the pain dimensions. Melzack and Katz (2001) acknowledged the mixed evidence from FA studies regarding the structure of MPQ, but cited Gracely (1992) for arguments against using FA to test the questionnaire structure. Indeed,

Gracely (1992) questioned the application of psychometrics in the evaluation of pain scales. He adopted an earlier distinction between semantic and associative meaning and argued that, while studies where subjects rate the similarity of the adjectives are good tests of the semantic meaning, studies that analyse the patients' responses to MPQ are tests of the associative meaning and therefore they study the characteristics of the patients, not of the questionnaire. Moreover, he argued that high correlations between subscales and low correlations between subscale items should not prevent the use of subscale scores, especially when subscales are differentially influenced by specific interventions.

These arguments are faulty, in my opinion. The psychometric properties of a questionnaire, whether a pain scale or ability or attitude test, are relevant to the extent that they tell us something about the respondent's experience via the respondent's answers. And they are adequate to the extent that the theory behind questionnaire construction stipulates specific relations between constructs that fit the structure of the reality it tries to measure. If the theory behind the SF-MPQ construction stipulates a distinction between sensory and affective dimensions of pain experience (and it is not a linguistic theory related to word meanings), the associative meaning should be the main focus of analysis. Any semantic analysis would be useful only to the extent that it guides the selection of relevant items in the context of the respondent's experience. Also, if the two dimensions show a lack of differentiation and subscale items do not correlate as expected, either the theory is faulty or incomplete, the questionnaire is not a reliable measure of the theoretical constructs or other data collection aspects have introduced significant amounts of error. Instead of rejecting the suitability of the statistical test, proponents of the theory would need to address these other possibilities. The fact that highly correlated constructs become less associated in different contexts would need to lead to theory refinements instead of rejection of other testing methods.

Nevertheless, as factor structure and other psychometric properties can vary between samples, using SF-MPQ in a heterogeneous sample (like the one in the present study) leads to another problem: the representativeness of the 15 descriptors for the whole sample and for all subsamples representing different diagnoses[14], which is another reason for examining the psychometric properties in this new case.

[14]This problem was overcome in some studies by developing tailor-made scales, such as the Neuropathic Pain Scale, with different descriptors relevant for the specific group (Jensen and Karoly, 2001).

Stage	Descriptor no.														
no.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	2.41	2.34	2.21	2.42	2.01	2.22	2.11	3.06	2.17	2.45	1.60	3.07	1.97	1.74	2.05
	(1.13)	(1.25)	(1.23)	(1.28)	(1.16)	(1.15)	(1.21)	(1.02)	(1.21)	(1.17)	(.99)	(1.08)	(1.11)	(1.03)	(1.22)
2	2.40	2.36	2.27	2.42	2.00	2.40	2.24	3.12	2.27	2.50	1.67	3.12	2.00	1.70	1.98
	(1.17)	(1.19)	(1.23)	(1.23)	(1.07)	(1.18)	(1.20)	(.96)	(1.15)	(1.16)	(1.00)	(.97)	(1.09)	(.98)	(1.16)
3	2.38	2.33	2.21	2.32	1.97	2.35	2.25	3.21	2.35	2.58	1.57	3.02	1.91	1.68	1.97
	(1.12)	(1.18)	(1.19)	(1.22)	(1.09)	(1.13)	(1.17)	(.86)	(1.14)	(1.08)	(.96)	(1.04)	(1.04)	(.98)	(1.16)

Table 8.13: Means (and standard deviations) for SF-MPQ items (possible range 0-3)

	Indexes														
	S-PRI			A-PRI			VAS			PPI			T-PRI		
Stage no.	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Mean	14.01	14.36	14.51	4.83	4.80	4.58	96.03	93.50	92.98	3.18	3.23	3.03	18.83	19.43	19.09
Std. Deviation	7.52	7.48	7.63	3.46	3.32	3.28	33.31	30.88	31.32	1.16	1.15	1.10	10.28	10.17	10.32
Skewness	.29	.25	.23	.40	.52	.58	-.74	-.60	-.36	-.01	.01	.05	.27	.26	.32
Kurtosis	-.74	-.71	-.89	-.87	-.67	-.55	-.14	-.16	-.42	-1.01	-1.08	-.95	-.87	-.68	-.78

Table 8.14: Descriptive statistics for SF-MPQ indexes (possible range: 0-33 for S-PRI, 0-12 for A-PRI, 0-144 for VAS, 1-5 for PPI, 0-45 for T-PRI)

Index	Stage		
	1	2	3
S-PRI	.81	.82	.85
A-PRI	.78	.80	.78
T-PRI	.87	.88	.89

Table 8.15: Internal consistency for SF-MPQ scales

Index	Stages		
	1-2	2-3	1-3
S-PRI	.63	.68	.56
A-PRI	.62	.66	.45
T-PRI	.66	.71	.56
VAS	.58	.71	.54
PPI	.64	.60	.49

Table 8.16: Test-retest stability of SF-MPQ indexes

Stage	Index	S-PRI	A-PRI	VAS	PPI
1	A-PRI	.72**			
2		.69**			
3		.75**			
1	VAS	.59**	.54**		
2		.62**	.57**		
3		.68**	.64**		
1	PPI	.62**	.55**	.71**	
2		.54**	.55**	.74**	
3		.51**	.59**	.72**	
1	T-PRI	.97**	.86**	.61**	.64**
2		.96**	.85**	.64**	.57**
3		.98**	.87**	.71**	.56**

Table 8.17: Correlations between SF-MPQ indexes

In the present sample, the item distributions were expectedly uneven for descriptors, as they were not designed to discriminate at a specific level of attribute intensity. The distributions of indexes (i.e. sensory, affective and total pain rating indexes, VAS and PPI) only slightly deviated from normality (means and standard deviations are provided in Tables 8.13 and 8.14). The measure showed slightly better internal consistency (Table 8.13). Test-retest reliability estimates showed a less stable measure (see table 8.16). Pain rating indexes were highly correlated in all three stages and moderately correlated with VAS and PPI.

This high correlation between sensory and affective components confirmed earlier concerns about the two scales being distinct dimensions of pain quality. An EFA (PCA, oblimin rotation) showed the existence of a single main factor that explained 36.4, 37.7, and 40.2% of total variance (in the three stages), while a 2-factor solution did not produce a meaningful result in terms of separation of descriptors. The 2-factor CFA model for the 15 descriptors in the present sample (Figure 8.5,

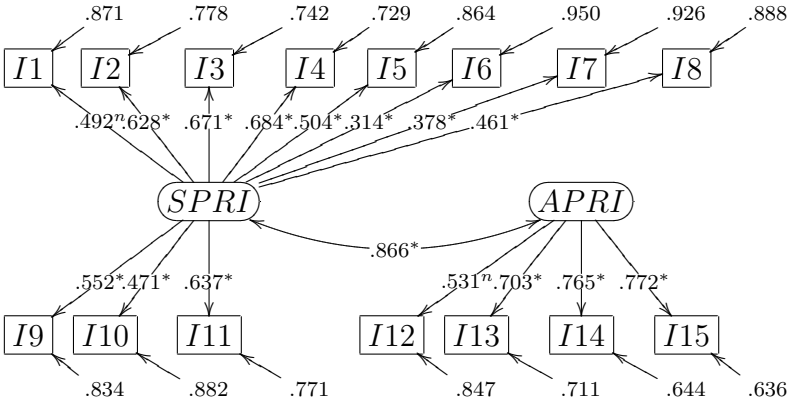


Figure 8.5: 2-factor CFA model SF-MPQ

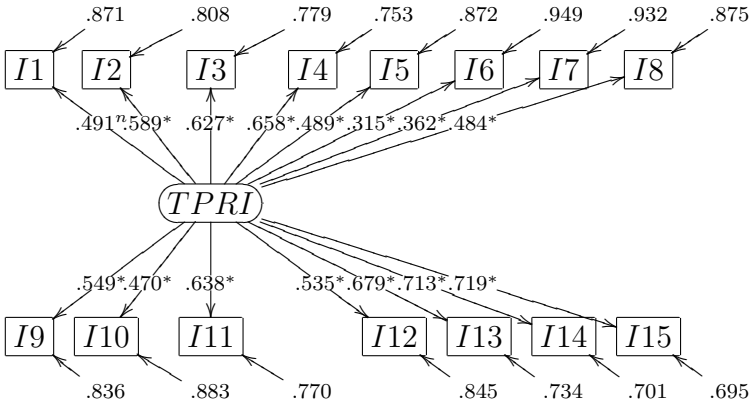


Figure 8.6: 1-factor CFA model SF-MPQ

covariance matrix in Table D.8) presented an acceptable, but not optimal, fit, with high correlations between the two factors (additional diagnostics suggested cross-loadings of several items on the opposite scale). A second (1-factor) model (Figure 8.6) showed a comparable fit. Goodness of fit statistics (ML, robust) are presented for both models in Table 8.18.

However the high correlations between the two subscales in the general sample might not replicate on specific subsamples (i.e. might depend on other patient characteristics, such as total pain intensity, emotion frequencies, emotion regulation, acceptance, illness perceptions). Also, previous research has indicated the two subscales are distinct in multiple contexts (Melzack and Katz, 2001). Therefore both the S-PRI and A-PRI were used in subsequent analyses together with the T-PRI,

	Model 1			Model 2		
Statistic	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
S-B χ^2 (df)	166(89) p<.001	177(89) p<.001	232(89) p<.001	189(90) p<.001	216(90) p<.001	246(90) p<.001
NFI robust	.87	.85	.82	.95	.82	.81
NNFI robust	.92	.90	.86	.90	.87	.84
CFI robust	.93	.92	.88	.92	.88	.97
RMSEA robust	.06 (.04-.07)	.06 (.05 - .08)	.09 .(.07-.10)	.06 (.05-.08)	.08 (.07 -.09)	.09 (.08-.10)
range of item loadings	.31-.77	.38-.85	.42-.74	.32-.72	.36-.74	.45-.71
Subscales correlations	.87	.80	.89			

Table 8.18: Goodness of fit statistics for SF-MPQ models

VAS and PPI scores, with particular emphasis on identifying variables that influence the relationship between S-PRI and A-PRI.

8.9 Chronic Pain Acceptance Questionnaire

CPAQ was developed to measure acceptance in chronic pain sufferers. The development and continuous improvement of the questionnaire is described in detail in Section 3.6. It is now considered that chronic pain acceptance (CPA) "entails that an individual reduce unsuccessful attempts to avoid or control pain and focus instead on participation in valued activities and the pursuit of personally relevant goals" (McCracken et al., 2004b, p. 159). CPA has two components: pain willingness (PW) and activities engagement (AE), with estimates of internal consistency of .82 (AE), and .78 (PW). No data for the internal consistency of a combined scale, test-retest reliability and range of loadings were reported. As a test of discriminant validity, the authors presented regression analyses showing unique contribution to pain-related functioning (controlling for age, gender, education, pain intensity and duration). A CFA model of the correlated 2-factor model (Vowles et al., 2008b) showed acceptable, although suboptimal, fit ($\chi^2/df = 2.2$, RMSEA= .06 (.05–.07), GFI= .89, AGFI= .86, CFI= .90).

However, in my opinion, the present version of CPAQ has two major weaknesses. First, given the issue of content contamination mentioned in Section 3.6, it is likely that the selection of the factors based on their relationship with measures of patient functioning (method applied by McCracken et al., 2004b) leads away from the efforts to measure the mechanisms of acceptance as described by ACT and biases the results towards conceptual overlaps with these measures, interpreted as

clinical utility. These overlaps can lead to confusing results if the instruments are used in assessing interventions. For example Vowles et al. (2009) reported that CPAQ scores improved after a CBT intervention. This might not indicate "that acceptance is a key process by which many therapies work" (as the authors proposed, p. 55), but that CPAQ shows content contamination with outcome measures (or perhaps the lack of practical distinctions between ACT and CBT, or the fact that reappraisal leads to acceptance). This example highlights the importance of concept clarity and distinctiveness (as reflected in measurement tools) for testing substantive hypotheses.

Second, all items of PW are reverse-scored. They represent strong statements related to emotional distress, efforts to control pain and related thoughts viewed as a life priority, even avoidance of activities, which conceptually would belong to the AE subscale ("I avoid putting myself in situations where my pain might increase"). AE contains only positive statements of a well-managed life despite pain, including acceptance of pain, which should actually measure PW ("It's ok to experience pain"). An inspection of items content suggests a second interpretation: the two factors are (at least partly) an artifact of a strong impact of the method (i.e. wording items in a positive or negative manner). It might be possible that AE and PW are distinct dimensions of CPA in addition to the differences due to item format. Indeed, the two subscales differ to a certain degree in their relationship with measures of functioning (McCracken et al., 2004b), although the analysis supporting this claim does not control for measurement error, as it relies on multiple regression equations. Some differences have also been identified in our study, e.g. the higher correlations between AE and work status, joy, and functional ERSs[15]; moreover, the distinction between experience and behaviour is mirrored in the analysis of health status indicators. Nevertheless, this overlap between content and method poses a major conceptual and practical problem: it cannot be appreciated to what degree the differences between PW and AE are due to substantive aspects or differences in responses to similar issues due to item wording.

The effect of item format is well known in the psychometric literature (Tourangeau et al., 2000; Podsakoff et al., 2003). The use of both positive and negative wording has several advantages: prompts respondents to pay more attention to item content, widens the sampling of the concept's content domain, counters bias due to acquiescence. However, there are associated problems: lower internal consistency, and

[15] Although BES and REQ might also be influenced by acquiescence bias, as there are no reverse-coded items included, due to the type of response format, i.e. requesting frequency estimations.

inadequate solutions if differences in item format are not accounted for (Weijters et al., 2009). Using CPAQ total scores would be a form of correcting for this bias, as is using an equal number of positive and negative items. Balanced scales are often recommended as an adequate method to offset acquiescence bias, although the assumption that the bias is equal for positive and negative items does not always hold (Billiet and McClendon, 2000).

The role of item wording in questionnaire development is more clearly explained in the belief-sampling model of survey response (Tourangeau et al., 2000), which stipulates four sets of cognitive processes that respondents perform in these situations: comprehension of the particular question, retrieval of relevant information from memory, judgement (integration of information) and response (mapping the judgement on the response format and editing it according to additional criteria). In this framework, item wording (as part of the broader item context) particularly influences the type of information accessible during survey responding, which determines the specific beliefs the respondent samples in answering a particular question (Weijters et al., 2009).

In contrast to CFA, where substantive and method-related common sources of item variance can be specified a priori[16], EFA cannot distinguish between these two and may therefore lead to biased solutions. The situation where EFA indicates two separate factors for positively and negatively worded items is rather frequent in the psychometric literature and inevitably leads to a concern about whether the factors are substantive or artefactual (Marsh, 1996, p. 810). Ideally, items intended to describe each of the hypothesised CPA components (or processes) should be balanced (similar number of reversed and nonreversed items). In this situation, the use of SEM (in this case CFA) allows for modeling of method biases related to acquiescence (Billiet and McClendon, 2000). Unfortunately when method and content based relations overlap, no modeling strategy is able to disentangle the two influences upon responses. However, the alternative hypothesis of a 1-factor model with correlated errors between items of similar format can be tested (Marsh, 1996).

Given the considerations above, it is very likely that the responses to CPAQ are influenced by item format especially due to the paradoxical nature of pain. Applying the belief-sampling model, lower correlations between reversed and non-reversed items are due to the respondents accessing contradictory beliefs. Acceptance is by design a paradoxical concept, as extensively discussed in Section 3.4. It is easily

[16]Although model identification is a concern in certain conditions.

imaginable that a person in pain endorses both statements referring to trying to lead a satisfying life and statements describing pain as a priority: they simply access opposite beliefs as prompted by consecutive reversed and non-reversed items.

These two major limitations have not been addressed until now, even if the psychometric properties of the CPAQ have been a subject of controversy recently. Nicholas and Asghari (2006) reanalysed the factorial structure of CPAQ via EFA (PCA) and identified 5 factors: the first almost overlapping with AE, factors 2 to 4 representing largely unrelated groups of PW items, and the fifth being a single-item factor representing one AE item with ambiguous wording. Another critique refers to not controlling for catastrophizing, fear-avoidance beliefs and self-efficacy beliefs which, when entered in the multiple regression analyses, decrease the predictive power of PW and AE below the level of significance, with the exception of AE in relation to depression. In their opinion, a remedy would be the use of multiple instruments to measure the different components of acceptance. This critique suffers from the same weaknesses. First, without considering item format, an EFA can produce misleading results. Second, unless the problem is addressed via an adequate longitudinal analysis, it will always be possible to reword this argument as 'which concept has the most substantial content overlap with measures of functioning?'. Their study was also criticised (McCracken et al., 2007b) for relying on EFA rather than CFA, not considering the improved predictive power of the total CPA score compared with AE or PW alone and ignoring the broader theoretical framework that guided the initial research[17]. A recent CFA analysis of the CPAQ in a Swedish version (Wicksell et al., 2008b) reported confirmation of the 2-factor model (even if the χ^2 test showed significant differences between the model implied covariances and the data), without considering the impact of item format.

A more recent study by Vowles et al. (2008b) provided relevant information for understanding the potential influence of method bias. The authors analysed the CPAQ via EFA, CFA and cluster analysis. Using a combination of criteria (eigenvalues, percentage of variance accounted, deviation from simple structure of the resulting solutions), they compared factor solutions from 5 to 2 factors and selected the 2-factor solution which confirmed the previous division into PW and AE subscales. CFA models were tested on a new sample. Although indices of fit were within the

[17]They responded by stating that CFA was considered premature given the contradictory results of the EFA and that their main argument against CPAQ variables remains their decreased association with outcome measures when controlling for other relevant concepts.

commonly used guidelines, none of the models[18] had optimal model fit based on the χ^2 test[19]. As discussed above, a CFA model that specifies both method and substantive factors is impossible as long as there is a perfect overlap.

The cluster analyses in Vowles et al.'s (2008b) study reached a 3-cluster solution, which was replicated in our study (see Section 7.6.1). Due to the low distinctiveness and stability of the clusters, I will adopt a more conservative view of considering the 3 groups as a way of dissecting the data, rather than an indicator of the existence of discrete categories. The groups (high AE and PW, low AE and PW, and discrepant, i.e. low PW and high AE) differed on nine measures of functioning, with the discrepant group falling in between the low and high acceptance groups. The authors interpreted these data as indicative of the usefulness of the two scales, but an alternative interpretation in light of the possible method bias is that the three groups represented different response patterns to CPAQ depending on the severity of their pain. The high acceptance group described persons whose pain severity was low, therefore they endorsed most statements in the direction of functionality, irrespective of item format. The low acceptance group included persons whose levels of pain affected their lives to such an extent that they endorsed most items in the direction of disability, again irrespective of item format. The middle group were those for whom pain was of medium severity (or maybe fluctuating) and therefore they endorsed both types of statements. Thus, the third group participated more to the overall distinction between these two factors [20]. Applying the belief-sampling model (Tourangeau et al., 2000), it is easily conceivable that a person with medium pain intensity would be more able to access opposite beliefs regarding their approach to pain, as they have stored in their episodic memory a balanced sample of situations where pain can be overwhelming or more easily ignorable[21].

Due to the two issues described above (content contamination with measures of patient functioning and method-content overlap in items), I would argue that the CPAQ needs further improvement in order to be able to support further research on CPA. Most importantly for the present study, a thorough analysis of the questionnaire is imperative.

[18] An initial 2-factor model, one model modification based on modification indices, another eliminating 2 items with low loadings and one 4-factor model.

[19] For a summary of the recent debate regarding χ^2 and fit indices see Appendix B.

[20] In this context, it is notable that there was no group of patients that reported high PW with low AE.

[21] Indeed, this method bias can be moderated by other variables, also depending on the substantive content of the items. For example, in items measuring self-esteem, the size of the negative-item effect varies with age and verbal ability (Marsh, 1996).

Item no.	1	2	3	4	5	6	7
stage 1	3.96 (1.62)	3.21 (1.56)	2.34 (1.52)	3.75 (1.63)	2.20 (1.80)	2.85 (1.71)	3.80 (1.57)
stage 2	4.15 (1.45)	3.35 (1.51)	2.68 (1.67)	3.64 (1.68)	2.32 (1.69)	2.88 (1.81)	3.48 (1.52)
stage 3	4.11 (1.51)	3.44 (1.56)	2.65 (1.64)	3.33 (1.71)	2.45 (1.65)	3.05 (1.86)	3.321 (1.56)
Item no.	8	9	10	11	12	13	14
stage 1	3.20 (1.87)	2.66 (1.84)	2.32 (1.70)	2.74 (1.67)	3.38 (1.59)	4.01 (1.61)	3.75 (1.67)
stage 2	3.05 (1.87)	2.76 (1.95)	2.76 (1.70)	2.46 (1.64)	3.45 (1.59)	3.97 (1.53)	3.54 (1.75)
stage 3	3.23 (1.82)	2.91 (1.90)	2.51 (1.64)	2.29 (1.61)	3.50 (1.65)	3.87 (1.60)	3.43 (1.76)
Item no.	15	16	17	18	19	20	total
stage 1	3.29 (1.63)	3.30 (1.73)	4.00 (1.62)	3.29 (1.79)	2.57 (1.63)	4.67 (1.40)	2.63 (.94)
stage 2	3.25 (1.70)	3.01 (1.78)	3.92 (1.61)	3.02 (1.80)	2.79 (1.66)	4.50 (1.44)	2.77 (1.00)
stage 3	3.46 (1.60)	3.14 (1.74)	3.99 (1.59)	3.00 (1.77)	3.97 (1.60)	4.43 (1.46)	2.87 (1.00)

Table 8.19: Means (and standard deviations) for CPAQ items and total score (possible range 1-7)

In the present sample, item distributions were only moderately skewed, indicating a good capacity to discriminate, with the exception of item 1 and 20 (most respondents reported that they get on with the business of living despite their pain, and struggle when they are in pain). The means and standard deviations are provided in Table 8.19. The subscales and total score distributions were normal. Internal consistency was $.90 - .91$, and test-retest reliability was $.81 - .87$ for the 4 month intervals and .79 in 8 months.

EFA (PCA, oblimin rotation) indicated a similar solution to Vowles et al. (2008b): 4 factors with eigenvalues above 1 explaining 34–38%, 10–14%, 6–7% and 5% of the total variance (in all three stages). A 2-factor solution showed the expected loadings on the two factors. However given that the first factor explains a substantially bigger percentage of variance and that the second factor contains the reversed items, an alternative conclusion could be that the most adequate solution is a 1-factor structure, even if the remaining unexplained variance suggests that the items reflect additional phenomena (including the influence of the method, as detailed above).

The 2-factor CFA model in the present sample (Figure 8.7) showed an acceptable, yet suboptimal fit, and so did a 1-factor model with specification of method bias (correlated covariances between reversed items, as modeled in Marsh, 1996; Figure

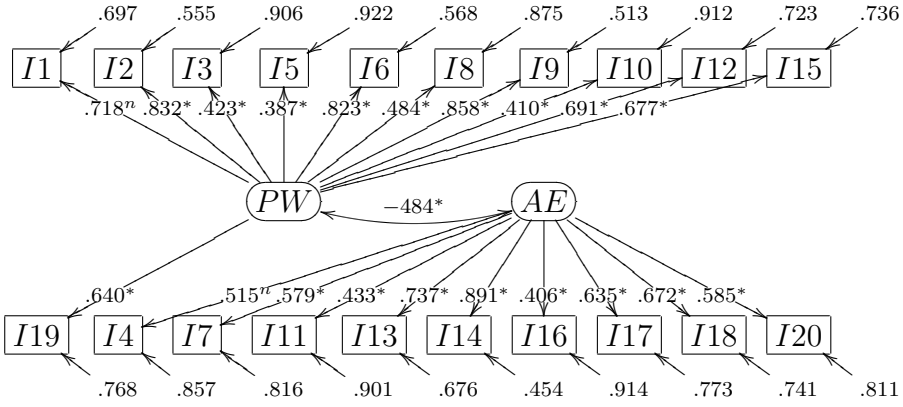


Figure 8.7: 2-factor CFA model CPAQ

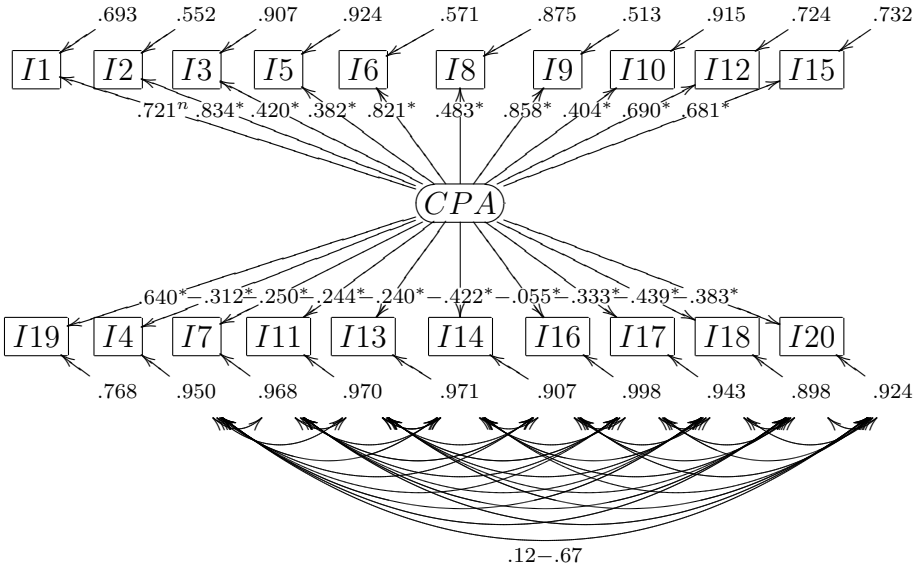


Figure 8.8: 1-factor CFA model CPAQ

8.8). Goodness of fit statistics are presented in Table 8.20 (covariance matrix in Table D.9). The comparable fit of both models suggested that both models are equally plausible (supporting the alternative interpretation of the two subscales as method artifact), although the lack of perfect fit together with the additional diagnostics (LM test suggesting cross-loadings of several items on the opposite scales) indicated that the data possibly have a different unknown structure. This reflects the theoretical difficulties with defining CPA (discussed in Chapter 3).

	Model 1			Model 2		
Statistic	Stage 1	Stage 2	Stage 3	Stage 1	Stage 2	Stage 3
S-B χ^2 (df)	392.35 (169)	314.07 (169)	358 (169)	298.67 (134)	221.92 (134)	266.34 (134)
NFI robust	.81	.82	.81	.85	.87	.86
NNFI robust	.87	.90	.88	.87	.92	.89
CFI robust	.88	.91	.89	.91	.94	.92
RMSEA robust	.07 (.06-.08)	.06 (.05-.07)	.07 (.06-.08)	.07 (.06-.08)	.05 (.04-.07)	.07 (.06-.08)
range of item loadings	.39-.89	.41-.89	.40-.90	.06-.86	.17-.88	.09-.90

Table 8.20: Goodness of fit statistics for CPAQ models. Model 1 - 2 factors, Model 2 - one factor with method bias specifications. All χ^2 tests significant ($p < .001$).

In conclusion, due to the overlap between method effects and possible substantive differences between the two subscales and their low distinctiveness and stability, a more cautious approach was adopted. Both subscale and total scores were analysed, with particular interest in the substantive differences between AE and PW.

8.10 Sickness Impact Profile Roland-Morris

SIP-RM is a short measure of pain-related physical disability first developed for use in low back pain (Roland and Morris, 1983), based on the longer SIP (briefly described in Chapter 2). The authors reported good short-term test-retest reliability ($r = .91$), more sensitivity compared to doctor ratings of physical signs and significant associations with several clinical features recorded on patient examination. Deyo (1986) reported high correlations with the original SIP Physical Disability subscale ($r = .89$), good test-retest reliability in a 3-week period ($r = .83$ for patients with no clinical change and $r = .76$ for patients with modest improvement), medium correlations to clinical measures of disability (spine flexion, pain severity, straight leg raising; $r = .28 - .42$), and sensitivity to clinical change. Jensen et al. (1992) applied SIP-RM (excluding the last item on pain duration) on patients with and without low back pain and concluded that its stability, its degree of association to SIP and other related clinical measures, and its sensitivity to change are not influenced by the presence of low back pain. Therefore they recommended the use of SIP-RM for other pain sites as well, although they advised replication on other patient samples. Stroud et al. (2004) developed an 11-item short form based on an IRT analysis of a heterogeneous sample of chronic pain sufferers, the new measure showing similarly sized associations with measures of depression and pain intensity.

Item	Stage 1	2	3
1. stay at home most of the time	45	41	38
2. change position frequently	89	86	87
3. walk more slowly	77	75	71
4. not doing any of the jobs that I usually do	47	41	36
5. use a handrail to get upstairs	53	53	56
6. lie down to rest more often	62	56	56
7. difficulty getting out of an easy chair	53	50	48
8. try to get other people to do things for me	37	35	32
9. get dressed more slowly	62	57	52
10. only stand up for short periods of time	63	59	58
11. try not to bend or kneel down	62	59	56
12. difficult to get out of a chair	49	48	44
13. difficult to turn over in bed	62	56	55
14. appetite is not very good	34	32	31
15. trouble putting on my socks	56	53	51
16. only walk short distances	68	66	59
17. sleep less well	83	77	75
18. get dressed with help from someone else	19	14	17
19. sit down for most of the day	29	31	28
20. avoid heavy jobs around the house	79	71	75
21. more irritable and bad tempered	50	45	42
22. go upstairs more slowly	62	61	57
23. stay in bed most of the time	10	11	7
24. in pain almost all of the time	85	79	80

Table 8.21: Percentages of reported pain-related disability in the three stages

In the present sample, items proved to cover a wide range of disability levels, as shown in percentages of item endorsement in Table 8.21. The measure showed good internal consistency (Cronbach $\alpha = .89 - .90$, item-total correlations $.30 - .70$) and stability ($r = .83 - .86$). Total scores covered the whole range and show normal distribution (mean$= 12 - 13$, SD$= 6$, possible range 0-24).

The authors did not discuss scale unidimensionality as support for validity, although it could be argued that the suitability of a total score would be supported by this property. A 1-factor CFA with more than 20 binary variables would require a larger sample size (Bentler, 2004, p. 70), and led to problems with matrix imputation in the present sample. Considering the items as reflecting a continuous normal distribution led to suboptimal GOF indices for a 1-factor model (e.g. Sattora-Bentler $\chi^2(252) = 594$ $p < .001$, CFI$= .81$, NFI$= .72$, NNFI$= .79$, RMSEA$= .07(.06 - .08)$, range of item loadings $.27 - .70$ for stage 1), although lack of fit might be due to not accounting for the categorical status of the variables. Selecting half the items for a 1-factor CFA with specification of categorical status led to similar lack of fit (e.g. Sattora-Bentler $\chi^2(54) = 558$ $p < .001$, CFI$= .84$, NFI$= .81$, NNFI$= .80$,

RMSEA= $.12(.10 - .13)$, range of item loadings $.32 - .87$ for stage 1), suggesting lack of unidimensionality for the selected items.

The existing support for the validity and reliability of the scale can be considered sufficient given no additional requirements for validity stipulated by the theory. Therefore the measure was considered adequate, despite limited face validity for particular items (such as 'using a handrail to get upstairs' for respondents that do not live in environments with multiple storeys, which several participants noted). The comparative use of selected items and total scores in structural models is however recommended given the likely heterogeneity of the questionnaire items.

8.11 Brief Illness Perception Questionnaire

The BIPQ assesses 9 categories of cognitive and emotional representations of illness (chronic pain) by single items: consequences, timeline, personal control, treatment control, identity, concern, understanding, emotional response, causation (Broadbent et al., 2006b). It was developed as a shorter version of the Revised Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002), for application in situations that prohibit the use of a long (80 item) version, such as multi-measure, population-based or repeated-measures studies. The psychometric properties of the BIPQ were assessed on samples of patients with myocardial infarction, renal disease, type-2 diabetes, asthma, minor illnesses and chest pain prior to diagnosis. The authors reported good test-retest reliability (on the renal disease sample; see Table 8.23), and moderate to good correlations with corresponding subscales of IPQ-R (Pearson's $r = .32 - .63$). Items were further validated by associations with self-efficacy (only for personal control), metabolic control in diabetes, and asthma morbidity and beliefs about medication. The predictive validity was supported by associations with key outcomes following myocardial infarction (attendance of rehabilitation classes, return to work, cardiac anxiety, quality of life). The discriminant validity was supported by significant differences in responses to items between the samples assessed (Broadbent et al., 2006b). The causal attributions item was validated by the similarity of factors identified with causes identified by IPQ-R. The data (means and SDs were reported) showed rather skewed distributions for some items in some patient groups, which indicated that these items might discriminate less well within particular illness categories (e.g., the mean timeline perception in diabetes patients is understandably 9.2, while concern perceptions for minor illnesses are on average 2.5, for an 11-point scale). BIPQ has had a limited application in

	items							
stage	1	2	3	4	5	6	7	8
1	7.6 (2.0)	9.0 (1.6)	4.4 (2.6)	5.3 (2.7)	7.6 (2.0)	7.5 (2.4)	7.2 (2.6)	7.2 (2.4)
2	7.1 (2.2)	8.9 (1.7)	4.8 (2.5)	5.2 (2.5)	7.4 (2.0)	7.0 (2.6)	7.0 (2.4)	6.8 (2.5)
3	7.0 (2.3)	8.9 (1.8)	5.1 (2.5)	5.5 (2.6)	7.4 (2.0)	6.8 (2.5)	7.4 (2.4)	6.7 (2.5)

Table 8.22: Means (and standard deviations) for BIPQ items (possible range 0-10)

chronic pain research to this date: a group of patients with chest pain in Broadbent et al. (2006b), and two migraine groups (French version) in Radat et al. (2009).

In the present sample, with the exception of personal and treatment control, all items were negatively skewed, especially those referring to timeline. Around 60% of respondents considered, probably with good reason, that their pain will last forever, and most of them were significantly affected by their condition, experienced many symptoms, were very concerned, understood rather clearly their condition and were rather affected emotionally. The distributions of the items raise questions regarding their capacity to discriminate between the respondents in this sample (descriptive statistics are presented in Table 8.22).

Stability estimates are presented in Table 8.23, in comparison to estimates in the original study by Broadbent et al. (2006b). It can be noted that the stability of BIPQ items in the chronic pain samples and for intervals between 4 and 8 months were obviously reduced in comparison to the estimates on shorter intervals in patients with renal disease. They were also overall lower than the 3-week test-retest reliability for the equivalent scales of IPQ-R in a rheumatoid arthritis sample, as reported in Moss-Morris et al. (2002). As seen in Chapter 7, the low stability is not due to substantial change trends, but rather to fluctuations from the individual trajectories, partly related to the substantive associations between IPs and emotions, acceptance and health status. Other causes might be a lower reliability in our sample due to respondent burden (BIPQ was presented last) or a genuine lack of stability of illness perceptions at such intervals[22].

The open-ended responses of the causality item were grouped in categories relevant to the illness studied. The great majority of causal attributions were similar in all three stages, therefore only one coding was performed for all. Categories were

[22]As detailed in Chapter 5, the SRM predicts fluctuations in IPs, as the individual continuously reappraises the condition in light of the coping strategies outcomes and environment changes. However, the extent of these fluctuations in chronic and acute conditions is not specified.

	stages			Broadbent et al. (2006b)	
items	1-2	2-3	1-3	3 weeks	6 weeks
1 consequences	.61**	.61**	.63**	.70**	.71**
2 timeline	.49**	.56**	.49**	.67**	.73**
3 personal control	.35**	.29**	.34**	.63**	.42**
4 treatment control	.38**	.33**	.22**	.55**	.70**
5 identity	.45**	.47**	.43**	.65**	.75**
6 concern	.52**	.57**	.53**	.66**	.66**
7 understanding	.43**	.48**	.43**	.48**	.61**
8 emotional response	.52**	.57**	.54**	.65**	.72**

Table 8.23: Stability estimates for the BIPQ ordinal items (Kendall τ in present study, Pearson's r in Broadbent et al., 2006b)

developed based on the IPQ-R classification (Moss-Morris et al., 2002). The immunity category was excluded, and two more categories were included: unknown cause, and medical attributions (anatomical/ physiological descriptions, comorbid conditions, and 'wear and tear', an explanation often given in medical contexts for chronic pain, especially due to osteoarthritis). It is important to note that medical attributions are not causes per se, only labels; nevertheless, the existence of such a diagnosis-related explanation may have an important impact on the sufferer's life. In the chronic pain context, the word 'cause' can also be interpreted as 'pain trigger' (e.g. 'it hurts when standing') or 'pain location' (e.g. 'my legs hurt'), but these meanings are not intended by the question. The few responses that implied such an interpretation and some unclear responses (e.g. 'stupidity', 'daily living') were considered missing data. Responses which would fit in more categories were included in all (e.g. obesity, alcoholism, anorexia were considered both comorbid conditions and behaviourally determined conditions). Only 11 participants (4%) did not respond to this question in any stage.

Percentages of occurrence are presented in Table 8.24. Most frequent causal attributions were related to medical treatment, the respondent's own behaviours, and a specific external event such as accident, injury, assault. It is notable that medical attributions were used by more than half of the respondents, and very few stated unknown causes. Most respondents (78%) stated up to 3 causal attributions, and 72% used more than 1 of the broad categories. Cause variables with higher frequencies were further analysed on their own, as were the broader categories.

The analysis of the BIPQ structure was performed as part of the main analysis in Chapter 7.

Category	Subcategory	Examples	%
Psychological attributions			44.4
	Stress/ worry/ emotions	anxiety/worry, stress, emotional state, depression, sadness	13.5
	Family / social conflict	childhood abuse, stress over family worries, up-bringing	5.8
	Own behaviour	bad posture, don't get my base levels right, excess weight, careless life style/work practices, diet, too much sport, overdoing	36.7
Risk factors			53.7
	Heredity	genetic predisposition to arthritis	5.8
	Poor medical care/ medical intervention	failed surgery, long waiting times, orthotics caused phlebitis and nerve pain, crap doctors	39.4
	Age	getting older, old age	5.8
	Height/ Weight	excess weight, my size (small)	5.8
External events/ conditions			52.5
	Chance	unlucky, bad luck	3.5
	Accident/ injury/ assault	whiplash, someone stuck a knife in my face, previous accidents	34.4
	Pregnancy	pregnancy, childbirth	3.5
	Working conditions	working in the fields when young, injury at work, my job	21.6
Unknown		no idea, nobody has explained the reason for the pain, don't know, never been told	9.3
Medical attributions			59.8
	Anatomical/ physiological descriptions	prolapsed disc, hip fracture, narrowing of spinal canal, chronic inflammation, nerve damage, slipped disc, trapped nerve	36.3
	Diagnoses/ comorbid conditions	spina bifida occulta, arthritis, illness, amputation, osteoporosis, chronic fatigue syndrome, fibromyalgia	33.6
	'Wear and tear'	wear+tear	6.9

Table 8.24: Percentages of causal attributions

8.12 Health care visits, medication and self-help variables

The selection of the categories used to assess health care use was made following a description of a set of questions with a similar purpose in a study of the impact of individual and social factors on health care seeking, self care and medication (Andersson et al., 1999). Percentages of reported health care utilisation in the present sample (irrespective of frequency) are shown in Table 8.25[23].

Unfortunately, the medication categories used in the present study proved to be rather technical, unclear, and incomplete. They did not contain examples (such as brand names), used generic terms (such as 'hypnotics' which includes opioids, benzodiazepines, etc.), and did not include antidepressants (such as imipramine and amitriptyline) and GABA analogues (such as gabapentin and pregabalin) which are increasingly prescribed in various chronic pain conditions. The difficulty of responding to these questions was apparent in response patterns (most responses were within the 'combined analgesics' category, which might have been interpreted as a generic 'pain relief' category) and respondent comments about not knowing which categories their medication belonged to. As no direct access to the original questionnaire format was available at the time of study design and the description available was very brief, the original study might have worded the questions differently to ensure reliability. Therefore the data regarding medication categories per se were considered unreliable and were not used in further analyses. No difficulties were noticed in responding to questions on health care visits and self-help.

Although the analysis of general groups of health care visits, medication and self-help were reported in the study by Andersson et al. (1999), the method of computing general scores was not described. Sums or means of the item values do not lead to meaningful scores, as percentages of overall reporting and patterns-of-use frequencies differ between health care categories. Therefore, decisions needed to be informed by the analysis of the descriptive statistics.

For medication, the great majority stated they were taking their selected type of medication daily or several times a day. Thus, the medication schedule might depend more on the doctor's prescription rather than the patient. Since there was

[23]Examples of most frequently reported other therapies were aromatherapy, massage, reflexology, psychology/counselling, osteopathy and visits to a pain clinic consultant. Other medications frequently specified were: amitryptiline, imipramine, lidocaine patches, gabapentin, pregabalin, tramadol. Other self-help methods included: use of TENS equipment, use of ice packs, baths, massage, meditation, relaxation, distraction, yoga, physiotherapist-recommended exercises.

type		stage 1	2	3
visits	GP	74	69	64
	emergency	11	9	12
	hospital	13	13	10
	physiotherapist	26	24	21
	acupuncturist	12	10	13
	chiropractor	8	6	6
	homeopathist	5	4	4
	naturopathy	4	2	2
	other therapist	20	18	19
medication	Aspirin / paracetamol	54	58	56
	Combined analgesics	60	58	63
	Combined muscle relaxants	29	33	31
	Tranquilisers/sedatives	28	27	26
	Hypnotics	8	8	9
	Ointments	26	30	35
	Natural medicines	19	20	16
	Other 1	20	18	18
	Other 2	6	8	6
	Other 3	2	1	1
Self-help	Use heat	56	58	58
	Rest	90	89	91
	Physical activity	64	69	70
	Other 1	29	29	23
	Other 2	8	5	7
	Other 3	3	2	1

Table 8.25: Percentages of reported health care use in the three stages

little variation in this respect, dichotomous variables would be more suitable to represent individual categories and the total number of categories selected (including 'other') would differentiate relatively reliably between low and more intense medication use. Still, the possibility that some might take more medicines from the same category and the fact that different categories don't have equivalent therapeutic effects lower the reliability of a total number of categories chosen as an indicator of medication use. To improve distribution properties of medication use, the use of 5 or more types of medication was considered a single ordinal level[24] (resulting percentages are presented in Table 8.26). As perfect stability is not to be expected for medication use in intervals of 4 and 8 months due to possible changes in the medication regimen, the relatively high correlations between the number of categories selected in the three stages (Table 8.28) was considered as indicating acceptable test-retest reliability.

Concerning health care visits, a certain degree of variation in responses existed only in the frequency of visits to GP, which was the most reported type of visit (Table

[24]The transformed variables were used only for analyses which assume normality.

	Stages		
No. categories	1	2	3
0	6.0	5.8	5.2
1	20.0	16.4	18.5
2	29.4	25.7	28.0
3	20.0	30.1	23.7
4	14.7	11.5	14.2
≥5	9.8	10.6	10.4

Table 8.26: Percentages of degree of medication use in the three stages

8.25). This variable was dichotomised in a 'never or once' category (44–55%) and a '2 times or more' category. The other types of visits were more related to specialist treatment, conventional or complementary, therefore were considered as belonging to a single distinct category[25]. These variables were characterised by stable frequency patterns (for example, physiotherapy involved usually 2 to 5 visits in 3 months, while a visit to the emergency service was a single event) and thus were better represented by dichotomised variables. Given the low percentages of occurrence, these types of visits were added to compute the total 'number of specialist treatments used'. Results showed that 41–45% of the respondents did not visit any specialist in the last 3 months, 33–35% visited only one type of specialist, the rest have visited more (only between 3–4% contacted 4 to 6 types of therapists). Thus, this variable was transformed in a 3-categories ordinal variable ('no treatment', 'one type of treatment', 'two or more types of treatment') to best represent the degree of professional help use. Stability estimates for visits to GP and professional help use are presented in Table 8.28.

Among the self-help methods, 90% of the respondents used rest to ease their pain, therefore this variable did not differentiate and was not considered further. There was more variation in the frequency with which people used heat and physical activity, however the highest percentage reported daily use (29–35% and 33–34%, respectively). For consistency, the same method of computing a global self-help score was applied here, resulting in a 4-level variable (Table 8.27). It is important however to note the limited reliability of this proxy of self-help use: there were few categories offered, and relying on other examples from respondents probably underestimated the number of methods they actually used. Still, to the extent that respondents that were preoccupied with using self-help tended to report more

[25]It must be noted that in the NHS Pain Centers the distinction between conventional and complementary is less clear-cut, as acupuncture and aromatherapy for example are practised within the multidisciplinary pain management service.

	Stages		
No. categories	1	2	3
0	16.2	11.9	13.7
1	30.9	31.4	29.4
2	35.8	42.0	45.0
≥3	17.0	14.6	11.8

Table 8.27: Percentages of degree of self-help use in the three stages

	number of	Stages		
Variable	levels	1 to 2	2 to 3	1 to 3
Visits to GP[a]	2	.42**	.46**	.37**
Professional help use[b]	3	.38**	.44**	.40**
Medication use[b]	6	.55**	.60**	.55**
self-help use[b]	4	.51**	.51**	.50**

Table 8.28: Stability estimates for health care use variables (a = Pearson's r, b = Spearman's ρ)

examples, this variable is acceptable as a proxy. Stability estimates for self-help use are presented in Table 8.28.

8.13 Conclusion of the psychometric analysis

The development of substantive theories is best undertaken in coordination with the development of related instruments; results of a substantive study, whether confirming predictions or not, should be assessed from the perspective of both the theoretical statements and measurement issues and lead to improvements in one or both of them (Nunnally and Bernstein, 1994, p. 107). This approach has been adopted in this study, and the present chapter has shown both the strengths and limitations of the instruments used, to better assess the accuracy of the inferences at the level of the substantive theories compared in Chapter 7.

Chapter 9

Discussion and conclusions

The present thesis focused on building an integrative account of behaviour, emotion and cognition in chronic pain. This effort requires a thorough examination of the existing theories and the alternative frameworks that have the potential to accommodate multiple aspects of the phenomenon. An important concern is avoiding a simplistic overview, which would lose the power to explain such a complex reality. The theoretical chapters (2 to 6) have therefore attempted to present a detailed picture of the efforts to understand the psychological aspects of living with chronic pain and have proposed that further research can benefit from being formulated and interpreted within the Cognitive-Affective Model of the Interruptive Function of Pain, and the wider framework of the Self-Regulatory Model. Many specific research questions can be formulated within these larger frameworks, and some of them were pursued empirically in the present study, as described in Chapter 7.

The present chapter attempts to bring together theory and empirical findings into an integrative view of the psychological aspects of chronic pain adjustment. First, the empirical results are interpreted within the theoretical perspectives that generated the associated hypotheses and exploratory goals. This interpretation is next summarised in a brief overview of the main findings. The present study is then critiqued considering both its strengths and limitations, which inform potential trajectories for future research. Finally, a brief summary of the main conclusions is presented.

9.1 Discussion and interpretation of the empirical results

Our longitudinal study of chronic pain adjustment has covered many substantive and measurement issues. The four areas of interest (acceptance, emotions, illness

perceptions, health status) were first examined separately, to test hypotheses related to each substantive theory. A next step towards theoretical integration was the study of each diadic relationship, followed by a comparative analysis of the psychological aspects in relation to health status. A further step was the exploration of time-related trends and fluctuations in all four areas, both at the intra- and interindividual levels. The identification of wide areas of overlap between concepts led to the exploratory investigation of intra- and interindividual differences in the strength of association between different aspects of self-reported pain experience; these differences were interpreted from the perspective of the proposed concept of "discrimination ability". In addition to these substantive analyses, a detailed psychometric analysis of the questionnaires used was performed in order to ascertain the degree to which the substantive inferences are supported by the reliability and validity of the measurement components. The sections below briefly discuss each of these empirical results.

9.1.1 Separate examination of the four areas

Chronic pain acceptance and psychological flexibility

One important question recently examined in relation to CPA is whether the responses would be better characterised by a categorical approach (distinct patient groups) rather than a dimensional one (scores on continuous variables). Cluster analyses were therefore performed in the present study following Vowles et al.'s (2008b) procedures and reached a comparable 3-cluster solution (k-means): high acceptance, low acceptance and discordant (activities engagement scores higher than pain willingness) groups, with significant differences in gender, education, presence and severity of comorbid conditions, vocational status, presence of depression, number of GP visits, pain intensity (VAS, PPI, T-PRI), pain related disability (RM-SIP), and medication use. However, the hierarchical cluster analyses did not suggest clearly distinct patient groups. Moreover, the clusters did not show stability in time, contrasting with the stability of the continuous scores, indicating a sizeable fluctuation of the solution given by the aggregation algorithm based on slight variations in the data.

Our results indicate that differences in CPA are better described as differences 'of degree' rather than 'of kind', to paraphrase Meehl (1992, p. 121). However, the use of data dissection in relation to CPA might also have its place in chronic pain assessment and research, as long as it is acknowledged that the categorisation is

based on relative cut-offs and that membership in one of the categories may be a temporary characteristic. The present analysis certainly does not offer a final response, as many other statistical procedures are available to answer the question of whether acceptance is more adequately described as a category or a continuum, and results need replication of different data sets and alternative measures.

The distinction between taxa and continua is particularly important in clinical psychology. If a trait reflects a latent continuum, locating individual scores on continuous variables and describing changes as small increments determined by a combination of additive changes is appropriate, while if it reflects latent categories, assigning individuals to the appropriate category by means of cluster analysis or other statistical methods and explaining causation in terms of single dichotomous factors might be more adequate (Haslam and Kim, 2002). The use of taxonomic analyses in chronic pain research has a long history, from initial attempts to identify patient groups based on psychosocial characteristics measured by WHYMPI (Rudy et al., 1989; Turk and Rudy, 1988), to recent studies that use taxometric techniques to study the relations between patients characteristics (e.g. pain intensity, pain interference and depression; Wilson et al., 2005), to identify distinct profiles related to the patients' readiness to adopt a self-management approach (Kerns et al., 2005), or in investigating the taxonicity of specific concepts, such as fear of pain (Asmundson et al., 2007). The issue of patient classification still awaits a comprehensive approach that would take full advantage of the various statistical methods available. Developing tailored pain management programmes for distinct groups would need to be based on their stability and distinctiveness, for which limited support exists as yet.

Another important question for the ACT model itself is whether CPA and PF are the same or different concepts. As discussed in Chapter 3, PF is described in ACT theory as a phenomenon that characterised the individual's attitude to internal events in general (feelings, thoughts, sensations). Also, illness specific acceptance measures have been developed starting from the initial item pool of the AAQ. Thus, it can be hypothesised that CPA is actually equivalent to general PF in the specific context of chronic pain, which would reflect in high correlations between the total scores of the two measures[1].

[1]Although an exact cut-off value for discriminant validity is not commonly agreed, Tabachnick and Fidell (2001, p. 84) state that variables with $r > .70$ are potentially redundant (except in repeated measures or structure designs) and weaken the analysis as they inflate the size of error terms, while variables with $r > .90$ cause multicollinearity and singularity and lead to statistical problems with matrix inversion.

On the other hand, ACT theory stipulates differences in how the six core processes apply to separate content domains in individual cases. For example, values can be applied differently in domains of living such as family, work, education and so on. Individuals may rate some domains as more important or rate living their life more in tune with their values in these domains, while other categories of life experiences might be less important or harbouring conflicts of values. Therefore, it can be hypothesised that accepting chronic pain is distinct from adopting a flexible attitude towards other mental content. In this situation, some individuals might be accepting of their pain, but not of other issues in their life, or viceversa, which would reflect in lower correlations between total scores.

The correlational analyses of total scores indicated substantial differences between the two concepts, and cross-lagged longitudinal path analyses indicated an asymmetric relationship in which only general PF scores are influenced by prior CPA scores. However accounting for measurement error (via both correction for attenuation and SEM) led to higher stability estimates which suggested no longitudinal change. Cross-sectional SEMs with selected items suggested a possible lack of distinctiveness between CPA and PF.

It is important to interpret these contradictory results from the perspective of ACT theory. The results based on the total scores and the cross-lag panel model would suggest that only CPA has a causal influence on later psychological flexibility in other domains of the patient's life. Therefore, pain management interventions would possibly need to focus on changing the patient's attitude towards pain, and expect changes in other domains as well, as the patient generalises the principles of acceptance to other situations.

However this conclusion would be premature, given the still early stages of theory development and the methodological issues involved. AAQ has lower internal consistency compared to CPAQ (given the broader definition of PF), and this leads to lower stability as well; one of the drawbacks of cross-lagged panel models is that concepts "that are measured unreliably will [...], when modelled as effects, likely appear to be influenced by more variables than actually influence them" (Maruyama, 1998, p. 115). Thus, the pattern identified at a total score level is likely to be a method artifact. The item-level SEMs indicate a more substantial overlap between CPA and PF to the extent that the boundaries between the two concepts are blurred both from the point of view of manifest indicators and external influences. These results would recommend a more integrated approach to pain management, in which issues of general flexibility would be addressed. This second

approach also has drawbacks, such as the limited representation of the content areas of both concepts by the items selected[2].

Given the theoretical difficulties of defining the concepts (detailed in Chapter 3) and the limitations of the current measures (examined in Chapter 8), these results are far from conclusive. In summary, the research findings in relation to acceptance reflect a psychological characteristic possibly better described in terms of a continuum and describing the sufferer's attitude not only towards pain, but towards various other life domains. The difficulties of delimiting the content area in a single unidimensional construct are apparent in this analysis as well as in the psychometric analysis of the CPAQ in Chapter 8. They reflect the still incipient stage of theory development in ACT and justify further substantive and psychometric research.

These conclusions, as well as the interpretation of the results concerning acceptance discussed in the next sections, are however relying on realist assumptions as dictated by the statistical methods applied and have limited consequences in the application of ACT within a functional contextualist framework, or at the level of individual or therapeutic models of pain management.

Emotions and emotion regulation strategies

The relationship between basic emotions and emotion regulation strategies (ERSs) in chronic pain patients has not yet been specifically described in the literature, given the recent development of the categorisation selected for this study (based on criteria of functionality and resource type). However the general literature on emotion regulation on which the REQ is based (Phillips and Power, 2007) suggests that dysfunctional ERSs are related to experiencing negative emotions (anger, fear, shame, sadness) more frequently (as reflecting chronic amplification of emotional distress) and happiness less often (reflecting lower levels of well-being and consistency with personal goals and plans). On the contrary, functional ERSs are related to more frequent happiness and less frequent negative emotions.

The correlational analyses confirmed the expected relationships. Certainly, these results cannot clarify whether dysfunctional ERSs lead in time to an increase in negative emotions, or are simply a behavioural expression of increased distress. The high stability of the measures did not allow a longitudinal examination. The complexity of the relations between ERSs and emotions is only starting to be explored, and this particular categorisation of emotion regulation is intended as a starting

[2]The SEMs were run with another set of items, with similar results.

point, rather than a definitive map of this territory (Power, 2008). However the differences in the magnitude of correlations indicate a non-homogeneous pattern. For example the limited relation between internal functional strategies and shame and anxiety in comparison with the strong connection between internal dysfunctional strategies and the same emotions, suggests a more important role of strategies such as rumination in these emotional states.

A few remarks about the results of the psychometric analyses of the two questionnaires are necessary. First, frustration was reported as the most frequently experienced emotion in our sample, confirming previous findings by Wade et al. (1990) who reported frustration as the most intense emotion experienced as concomitant of their pain and Fernandez and Milburn (1994) who found anger as the most intense emotional experience. Second, among all BES emotion terms, problematic discrimination properties were identified only for 'shame', 'humiliated' and 'disgust'. Reporting low levels of shame in responses to these 3 terms was probably related to a particular use of these terms in this sample, as guilt and blameworthy were slightly more endorsed. Third, in contrast to the acceptable structure of the BES (the more valuable given the difficulties of measuring emotional experience with emotion labels), the suboptimal psychometric properties of the REQ indicate that the strategies relevant for chronic pain sufferers differ from those frequently used by a healthy population. As living with chronic pain usually limits one's behavioral choices, due to both physical limitations and avoidance motivation (or, in CAM terms, interruption associated with escape motivation), it is understandable that patients use less external dysfunctional strategies (physical aggression is often not an option for a person with limited physical strength who depends on the others' support), and perhaps use strategies not included in the REQ, for example antecedent-focused emotion regulation. In this context, investigating the relations between acceptance and emotion becomes even more salient.

Illness perceptions

The examination of the BIPQ subscales focused on two main issues: the adequacy of data reduction methods (total scores and clustering), and the exploration of the causal attributions.

The possibility of computing total scores for BIPQ (or IPQ) implies that all illness perceptions are reflecting a single dimension of functionality versus dysfunctionality. Radat et al. (2009) reported using total scores for BIPQ and support this with proof of limited internal consistency (Cronbach $\alpha = .68$). As detailed in Chapter

5, this practice is questionable on theoretical and statistical grounds. Given the previous empirical results (Moss-Morris et al., 2002), it was hypothesised that the BIPQ subscales do not form a unidimensional construct. The inspection of the correlation matrix between BIPQ subscales revealed a non-homogeneous pattern that questioned the validity of a total score approach. IPs of consequences, timeline, identity, concern and emotional response were relatively closely related, while IPs of understanding and control formed a separate group. These observations were confirmed by a PCA analysis which resulted in the two groups of IPs being differentiated by 2 principal components: low cognitive control – high emotional impact, and high cognitive control – moderate emotional impact. These results accurately reflect the theoretical assertions of the Self-Regulatory Model and the parallel-processing model, which describes both convergent and divergent interactions between cognition and emotion in relation to the health threat, as described in Chapter 5.

As with CPA, a taxonomic analysis is also relevant for IPs. Following the methodology described by Hobro et al. (2004), two well-represented groups of patients were identified which had similar characteristics with the initial 'adaptors' and 'non-adaptors' groups identified by Hobro et al. (2004): one of the groups reported significantly less consequences, shorter timeframe, higher personal and treatment control, more understanding, less emotional impact, less symptoms and less concern, and also less pain intensity, pain-related disability and frequency of GP visits. No differences were found in terms of gender, age, vocational and marital status, education, comorbidity, pain location, duration and other health care use variables. However, the distinctiveness and stability of the groups identified was not found adequate. The distance coefficients of the hierarchical clustering algorithms did not support a two cluster solution, and visual exploratory methods revealed no separate groups, but rather a continuous distribution of scores on which the clustering algorithm applied relative cut-offs. The solution also showed low stability, with a considerable number of cases changing category between the three stages. The stability is likely due to both lack of cluster distinctiveness and low test-retest stability of subscales scores (unsurprising due to the single-item format).

We can conclude that, similar with CPA, differences in illness perceptions are more adequately described as differences 'in degree', in this case on several distinct continua (minimum two, cognitive and affective). This clarification is important given the possible consequences in practice of using these groups. Both Hobro et al. (2004)

and Clatworthy et al. (2005) discussed classification as being useful for tailored interventions. Given the evidence, only 20% of the patients would be reliably labelled as adaptors or non-adaptors in an 8-month period. If 60% are unreliably classified or excluded from these groups (excluding the 20% due to attrition) the errors of allocation in the situation of unstable groupings may have important practical consequences. If the delay between diagnosis and intervention is around 4 months (which is conceivable considering waiting times for pain management programmes), a sizeable proportion of patients that have been classified as non-adaptors at the diagnosis stage will have changed their illness perceptions until the intervention and would receive an intervention that would not address their current beliefs. Moreover, labelling patients as 'adaptors' or 'non-adaptors' can be detrimental to therapeutic progress if applied inflexibly. As ACT theory explains, reifying language is an important barrier to psychological flexibility. Especially when labels do not reflect stable characteristics (such as gender), they can work against the purpose for which they were created, i.e. to enhance the efficiency of behaviour change interventions.

This danger of inflexible application is illustrated by the following assertion of Hobro et al. (2004, p. 281). In the context of discussing possible links between illness perceptions and fear of pain and catastrophizing (identity, controllability, consequences), they proposed that diagnosing illness perceptions could help select suitable intervention strategies. For example, they suggested, self-management strategies are less valid for persons with perceptions of low personal control. Targeting maladaptive belief patterns would be a first option in this case. However this recommendation conflicts with both the SRM and ACT theory, which underline the role of context in the adequacy of individual beliefs, and do not view the belief of low personal control as invariably maladaptive.

Thus, using continua and taking into consideration the lack of stability of illness perceptions are therefore more adequate in this context. Using continua also offers a way of dealing with the excluded cases. The respondents belonging to other less represented clusters are ignored in the two-cluster solution. Clatworthy et al. (2005) do state that grouping all individuals is not a criterion for a good categorisation, however they also state that the excluded cases can prove to be interesting extreme cases worth studying[3]. However, if dealing with continua rather than categories allows for dealing with all existing cases, while reducing the classification errors due to a questionable clustering solution, then it is preferable (Everitt et al., 2001).

[3]Also, in practice a clinician needs to deal with all cases, which might lead to automatically and erroneously using the same categories for all patients.

The lack of stability of IP scores might partially depend on changes in the severity of the condition. The comparison between the selected groups does not control for illness severity assessed by methods independent of self-report, to exclude the alternative interpretation that the two groups might reflect extremes of a pain severity continuum (as reflected in the other measures of pain, mood and functioning). Hobro et al. (2004) do not discuss this possibility and build interpretation alternatives only in terms of personal attitudes (ambivalence about treatment control, negative affect, catastrophising). As discussed in Chapter 5, this is a common interpretation bias in SRM research.

The analysis of the causality-related IPs in chronic pain revealed no consistent relation with other IPs, except the association between anatomical/ physiological descriptions (and the wider category of medical attributions) with perceptions of understanding, which suggests that having a causal explanation in medical terms is considered an important aspect of understanding one's illness. These results contrast with Moss-Morris et al.'s (2002) findings on a mixed health problems sample, in which various associations were identified, for example between psychological and change attributions and emotional IPs, risk factor attributions and personal and treatment control. Such relations make causal attributions more relevant for other conditions (such as diabetes, asthma, or HIV), than for chronic pain conditions.

Health status indicators

The relationship between indicators of health status such as pain-related disability, perceived pain intensity, health care utilisation, vocational status and also to other related characteristics (such as pain spread, comorbidity, time since pain onset) is an important issue for chronic pain management, even if it has been surprisingly rather neglected in the literature. As most are used to assess the severity of the condition, it is important to identify to what degree they are all interchangeable measures. If the answers to all such indicators would be influenced by a single latent cause (i.e. health status), it would be suitable to target treatment programmes towards improving this causal variable, which would reflect in better patient scores in the relevant indicators. If on the contrary the various indicators are distinct and loosely related, it is possible that each is influenced by different factors in the therapeutic process (and illness progression) and different causal mechanisms need to be identified. In this case, situations in which interventions might result in contradictory results (for example decreasing pain intensity while increasing pain-related disability) would need to be given special consideration.

In the first case, one would expect strong associations between health status indicators, and similar relationships with psychological variables such as acceptance, emotions, illness perceptions. Von Korff and Miglioretti (2005) referred to work on the validation of the Chronic Pain Grade (CPG) questionnaire (Smith et al., 1997)[4] to support the assertion that indicators such as pain intensity, interference with activities, and pain-related role disability can represent an underlying dimension of pain severity, even if they represent distinct constructs. Therefore it can be hypothesised that the indicators of pain-related disability (SIP-RM), perceived pain intensity (SF-MPQ indices), health care utilisation, and vocational status form a unidimensional construct.

However, some evidence speaks against this model. For example, Jensen et al. (1992) reported that pain-related disability was significantly correlated with pain intensity ($r = .27$) and number of pain areas ($r = .28$) only for low back pain sufferers[5]. Also in the case of CPG scores, the relationship between pain intensity and pain-related disability changed depending on total pain severity levels, with pain intensity being more discriminative at lower levels of severity, while disability being more relevant at high levels (Von Korff et al., 1992; Von Korff, 2001; Smith et al., 1997). The low correlations between these indicators in certain cases is one of the reasons for recommending the assessment of multiple outcomes in evaluating results of treatment programmes, as discussed in Chapter 2. Based on these considerations, it can be hypothesised that the health status indicators do not form a unidimensional construct.

Correlation analyses indicated a heterogeneous pattern of relations. The number of specialist treatments and the number of self-help methods used were excluded from further analyses due to lack of association with other measures of health status. Pain intensity and disability indicators showed medium correlations, medication had small to medium sized correlations to all other variables, while GP visits and work status were associated with most other variables. Two alternative CPA models were tested to identify whether the distinction between pain experience and pain behaviours would be justified, and the results provided support for this distinction.

[4]This study was conducted on a general practice population in Scotland and reported an internal consistency of .91, item-total correlations of $.69 - .83$ and factor loadings greater than .75 for all 7 items measuring pain intensity (present, worst and average) and pain related disability (interference with activities, including work).

[5]Correlations with depressive symptom severity were significant for all patient groups ($r = .40 - 47$).

This analysis suggests that at least two outcome categories exist and are possibly influenced by different factors, and particular attention needs to be paid in pain management interventions to factors that influence these two aspects differentially. As discussed in Chapter 5, strategies that aim to diminish pain intensity at the expense of increasing pain disability have actually been the target of behavioral interventions for decades, although behavioural control runs the danger of focusing on the decrease of pain disability via at least temporary increases in pain experience. From this perspective, the more recent acceptance-based interventions attempt to strike a balance between the two outcome categories by reformulating achievable aims to change activity levels and testing the constraints of the personal pain perception levels[6].

9.1.2 Interrelations

Emotions and acceptance

Correlational analyses indicated that both CPA and PF are related to more frequent positive emotions and functional ERSs, and less frequent negative emotions and dysfunctional ERSs. At the item level however, only dysfunctional strategies showed consistent associations, while external functional strategies were only weakly related to acceptance and the two internal functional subscale items (both related to reviewing future plans) were significantly related only to the activities engagement subscale.

These results confirm the theoretical similarities between emotion and acceptance approaches to chronic pain adjustment. The magnitude of the correlations between emotions and acceptance are similar to the results of McCracken et al. (1999) on pain-related anxiety. Since the BES has not targeted pain specifically, it is unsurprising that both PF and CPA show correlations of similar magnitudes, with PF consistently more associated to emotions and internal dysfunctional ERSs. Unlike Kratz et al. (2007), who found pain acceptance related only to positive affect and not to negative affect in weekly ratings, focusing on specific negative emotions led to significant associations, which highlights the need to differentiate discrete emotions in chronic pain research.

[6]In this context, the CPAQ distinction between activities engagement and pain willingness is supported by this similar distinction at the level of health outcomes. This substantive difference would however need to be clearly separated from method effects in future research.

There are several possible interpretations of these relationships. As discussed in Section 6.2.1, CPA (or psychological flexibility and its opposite, experiential avoidance) may be viewed as a set of meta-emotional representations that guide both antecedent-focused and response-focused emotion regulation, an emotion regulation strategy in itself (Sloan, 2004; Kollman et al., 2009), a broader construct that encompasses emotional distress and emotion regulation (Kashdan et al., 2006; McCracken and Keogh, 2009). In CAM, experiential avoidance is viewed as a natural result of chronic pain stimulation (which motivates escape) and partially overlaps with worry as a pain-focused problem-solving effort, while acceptance is equalled to reformulating the pain problem in terms of reducing disability and distress (Eccleston and Crombez, 1999). In the SRM, acceptance can be considered as reflecting cognitive beliefs about pain, emotional responses to pain and pain behaviours (coping strategies), constituting rather an emergent pattern in the cognitive-affective interactions in responding to health threats.

Alternatively, both acceptance and emotional responses might be viewed as at least partly determined by health status, or even to a certain extent as indicators of illness severity: a more severe condition would lead to (and reflect in) lower acceptance, increased emotional distress and lower positive emotions. Selecting between these alternative interpretations is only partly an empirical problem (and due to the different formats of the questionnaires used, only partly answerable based on this data set). Clearly, emotional issues can be reformulated in ACT terminology, and vice versa, and it is unclear if the relations identified here are structural (in the sense of causal relations between distinct phenomena), measurement-related (i.e. indicating an overlap in content between the two questionnaires), or both. This issue will be further detailed in the next sections.

An important observation is the comparatively stronger association between the AE subscale and happiness and functional ERSs at the total scores level, and the significant correlations between internal functional ERSs and AE only. These differences indicate that the method bias identified in CPAQ (see Chapter 8) functions also in BES and REQ, and it is possible that the lower correlations between the items of the happiness subscale and the other emotions, and also between functional and dysfunctional ERSs might also be due to similar mechanisms; when prompted to report positive or negative behaviour, respondents tend to agree to both, as they access opposite examples from their previous experience, thus lowering the correla-

tions between the two types of items[7]. On the other hand, the differences are not present in the case of negative emotions and dysfunctional ERSs, suggesting that either the positive items are in this case more prone to such method bias, or the substantive differences between AE and PW interact with the method bias to produce this pattern. This observation highlights the importance of distinguishing between substantive and method effects in questionnaire construction, where possible.

Illness perceptions and acceptance

Non-adaptors (as identified by cluster analysis of illness perceptions) had as expected significantly lower scores of acceptance (CPA and PF) compared with adaptors. Beyond these simple differences, exploring the relationships between specific IPs and acceptance was more informative (which underlines the necessity of working with continua instead of using data reduction if data are not homogeneous).

All IPs except perceptions of understanding were significantly related to CPA and PF, the association being stronger for CPA, which is to be expected, as both CPA and IP subscales refer to pain specifically, while PF addresses more general issues (and thus is more related to the emotional impact of the condition on other life domains, as shown by its associations with perceptions of emotional response, concern and consequences). The stronger associations with emotion-related perceptions and the lack of association with understanding (as a cognitive aspect) support the view that all three domains, acceptance, emotions and illness perceptions, share a common focus on emotional aspects of living with chronic pain, described in the CAM as perception of threat. This is also in accord with the central (although rather neglected as yet) role of emotion in the SRM. As stated in the previous section, it is unclear whether these relationships are structural, measurement-related or both.

Importantly, perceptions of personal and treatment control are positively associated with CPA. This result is relevant for the controversial issue of the role of control in acceptance. As detailed in Chapter 3, acceptance is defined as opposite to effortful control, and ACT has been developed in a sense as a reaction to the control-based approach of OBT and CBT. Based on factor analyses of an early version of the CPAQ, McCracken (1999) eliminated items referring to cognitive control due to low correlations with the rest of the questionnaire. On the other hand, Risdon

[7]One difference is that, while the CPAQ subscales were derived based on EFA, both BES and REQ included these distinctions as a theoretical choice from the design phase. Given the format of the questionnaires, at least for BES, accounting for acquiescence bias by using reversed format would not be possible.

et al.'s (2003) study on the meanings of acceptance in pain-related discourse in British culture identified one account that equates acceptance with taking control. Also, perceptions of personal and treatment control may indicate lower severity of the pain condition, which is understandably easier to accept, in the sense of getting on with one's life at lower levels of pain intensity. Thus, even if control is not a representative aspect of CPA, these results indicate that it is not an antonym either. Both control and acceptance have many meanings, which encumber definitional clarity.

Among the causal attributions, only reports of external causes were related (negatively) to CPA and PF. This broader category includes two main causes: accident/ injury/ assault (whiplash, someone stuck a knife in my face, previous accidents) and working conditions (working in the fields when young, injury at work, my job). These types of events are understandably less likely to be accepted, since they involve conflictual relationships which are difficult to come to terms with (in comparison to situations where the pain is considered due to comorbid conditions, or own behaviour).

Illness perceptions and emotion

Non-adaptors consistently reported more frequent negative emotions and use of internal dysfunctional ERSs and less frequent positive emotions. However the differences in the use of external dysfunctional and both functional ERSs were not consistent across the three stages. Since illness perception subscales measures specifically the individual's response to health threats, the lack of consistent associations may be due to the fact that the unrelated ERSs are not highly relevant to regulation of the emotional impact of a health threat. As already seen, external dysfunctional ERSs are underreported in this group, and some functional ERSs are weakly related to CPA and PF. These results highlight the need to further explore emotion regulation in the context of chronic pain, particularly antecedent-focused, as opposed to the response-focused strategies included in REQ.

The relationships between specific IPs and specific emotions were also informative. In addition to perceptions of emotional impact (concern and emotional response), specifically designed to measure emotion and therefore unsurprisingly related to all emotion variables, most other IPs showed significant associations, with the exceptions of perceptions of timeline and understanding. As detailed in Chapter 5, the content of the IPs was developed within the SRM based on the content of Stimulus Evaluation Checks (SECs) described in Scherer's component process model

of emotion. While SECs include appraisals of novelty, pleasantness, goal significance, ability to cope (including causation and control) and compatibility with internal and external standards, the SRM's Prototype Assembly and Appraisal Checks (PCs) refer to both emotional and cognitive interpretations. Even if perceptions of identity, causes, timeline, consequences and controllability are described as defining the health threat at the cognitive level (Leventhal et al., 1992), the SRM stipulates multiple interactions between parallel cognitive and affective processes. Cameron et al. (1993) noted that emotion may be elicited by the symptoms, by perceptions of identity and consequences, by external events or by appraisals of coping outcomes, while Leventhal (2008) described them as 'embedded' in the cognitive-affective-behavioural framework. In the CAM, both IPs and emotion measures can be described as individual perceptions of threat, but also as the worry-laden problem-solving efforts (which may reflect in IPs of concern, emotional response, consequences and in dysfunctional ERSs or reports of frequent emotional distress).

These theoretical considerations suggest that perceptions of consequences, control and identity are related to emotion, while no theoretical statements referred to perceptions of timeline and understanding. The correlational analyses fit this description: timeline is not consistently related to any emotion and ERS, while understanding is consistently related only to external functional ERSs. As this ERSs category includes talking to others and asking for advice, acquiring an understanding of the condition may be the result of obtaining information from friends and health professionals, which highlights the importance of communication skills in acquiring a coherent illness model. Coherence is central to the SRM, and although the 'understanding' item of the BIPQ is only an approximate indicator this association suggests the usefulness of further investigating the role of external functional ERSs (and perhaps communication skills broadly speaking) in achieving and maintaining coherence (and thus taking adequate action). The consistent association between treatment control and external functional ERSs supports this conclusion.

While emotional response was associated to all emotion variables, concern appeared a more limited term, related only to negative emotions and internal ERSs. Perceptions of identity (symptoms) were only related to anger, sadness and anxiety, confirming research that showed the three emotions as the most frequently experienced in relation to pain perception (Fernandez and Milburn, 1994; Fernandez and Boyle, 2001; Clark et al., 2002; Price et al., 2001). Both personal and treatment control were positively related to happiness/joy, while personal control was also associated (negatively) with sadness, confirming controllability as both a cognitive

and emotional appraisal. Perceptions of consequences were associated with all emotions (except shame) and internal ERSs, suggesting that the impact of the chronic pain condition is perceived as highly emotional. These results also confirm findings regarding the structure of the BIPQ, presented in a previous section.

Shame and external dysfunctional ERSs were largely unrelated to illness perceptions. A possible explanation is the predominantly interpersonal and conflictual nature of both concepts which may be independent of the mostly intrapersonal and coping-oriented role of the illness model and its emotional components. Also, both characteristics are underreported, likely because they are less socially acceptable and less relevant for this population (as discussed above, antecendent-oriented ERSs might be used more frequently by chronic pain sufferers in order to avoid situations in which they might feel ashamed, or be aggressive towards others. In future studies, the use of different measures might be necessary in assessing these concepts.

This pattern of relationships may support various interpretations, for example illness perceptions as sources of frequent emotional reactions, or emotional distress as a result of illness severity generating both reports of frequent emotional states and perceptions of increased illness impact. It is important to note that participants' responses to the BIPQ, especially given the relationships between BIPQ items and emotion measures, cannot be viewed simply as 'maladaptive cognitions' independent of illness severity and its emotional aspects. As detailed in Chapter 5, this frequent misunderstanding can lead to inaccurate causal interpretations. Alternative interpretations will also be considered in the next sections.

Only external causal attributions were related to emotional variables (increased anger, sadness and internal dysfunctional ERSs, and decreased happiness and external functional ERSs), reproducing the associations to CPA and PF. Thus, chronic pain resulting from accidents, injuries or working conditions is reported as having a significant emotional impact.

9.1.3 Health status and psychosocial variables

Contextual factors and health status

Each health status indicator was related to a slightly different set of contextual variables, as were the indicators of the three psychological factors considered. Without discussing in detail the possible explanations for each single relation, it is important

to note that some of them might reflect unsophisticated causal processes (such as the relation between work status and financial status, or between education and work status), others more likely suggest conceptual overlaps, for example between increased negative emotions and reports of comorbid depression. The magnitude of the associations was overall small to medium, suggesting a minimal influence of demographic and medical characteristics, and confirming previous studies (e.g. Viane et al., 2003). Obviously, it is likely that other contextual factors not measured in this study have more impact on health status in chronic pain, especially more proximal indicators of the physical and social environment[8]. Therefore, controlling for these factors in further regression models to examine the unique contribution of psychological variables to statistical prediction of health status cannot be interpreted as eliminating all contextual differences that might influence health status reports.

To identify the individual roles and total predictive power of contextual variables for health status, regression models were run with selected variables (from consistently significant associations, eliminating overlapping and similar ones). Gender, spread of pain, comorbidity and previous surgical treatment significantly participated in explaining 19–23% of the variance in pain-related disability scores. This result contrasts with the limited predictive power of contextual variables for medication use (previous physiotherapy and total number of recent negative events, 7–9%), work status (comorbidity, 7–10%), S-PRI (age at pain onset and previous surgery, 6–7%), A-PRI (comorbidity, 4–7%), T-PRI (comorbidity, age at pain onset and previous surgery, 7–13%), and VAS (education and previous surgery 7–9%)[9]. The number of GP visits and the present pain intensity were not predicted reliably by any contextual variable. One possible interpretation is that, while disability is a relatively stable characteristic more influenced by gender roles and stable constraints of the medical condition, the rest of the variables depend on more circumstantial and idiosyncratic factors, in addition to factors such as comorbidity and previous treatments.

The fact that different factors influence sensory and affective pain quality brings support to the theoretical viewpoint that the two pain perception components, even if they correlate substantially in self-reports, are however independent in the sense of being subject to different influences.

[8]One limitation of the study is the limited coverage of social support, discussed in Section 9.4.
[9]The stability of the estimates between the three stages in notable.

Acceptance and health status

Confirming the study hypotheses, participants who reported more CPA reported better health status in terms of disability, vocational status, healthcare utilisation (medication, GP visits) and pain intensity (all indices), controlling for the relevant context variables. The PF scores did not consistently increase prediction when controlling for CPA, which reflects either a mediation mechanism, or, in light of the previous analyses, the lack of a clear distinction between the two. Some contextual variables also became nonsignificant predictors when CPA was included in the equation, possibly reflecting a mediation role of CPA.

It is important to note that the CPA subscales did not relate differently to pain perception and pain behaviour indicators (with the exception of work status, which was more strongly related to AE). Thus, although both CPA and health status may be characterised by the experience - behaviour distinction, this similarity is not apparent in the associations between the two. These results only partially confirm McCracken et al.'s (2004b) study, which also found AE more related to work status, but found PW comparatively more related to pain intensity, medical visits, pain medication and physical disability[10].

CPA explained a sizeable additional proportion of the variance in disability (21–24%), affective pain quality (15–31%), VAS (14–17%) and PPI (12–24%), and a comparatively lower percentage in the other HSIs (e.g. 3–4% for medication use, 7–17% for S-PRI). These results are consistent with the description of acceptance as focusing mostly on valued living and emotion regulation[11].

These models are not to be interpreted as a causal effect of CPA on health status; other equally possible interpretations may describe CPA as a result of health status, or even as a different aspect/indicator of pain severity (both in terms of pain perception and pain behaviours). The previous research on CPA reviewed in Chapter 3 has not achieved a definitive test of these alternative hypotheses. The only study approaching the design requirements for such a test is Vowles et al.'s (2007a) study of the role of pain, acceptance and catastrophising in explaining outcomes of an acceptance-based intervention. However, the simultaneous measurement of changes

[10]Their findings were based both on correlational analyses and MRMs controlling for pain intensity where appropriate, therefore an exact replication would be necessary for a full comparison.

[11]General pain indices such as VAS and PPI are arguably more influenced by affective aspects. For example, PPI is known to fluctuate considerably depending on momentary psychological factors such as mood, attention, etc., and correlates stronger with the affective and evaluative dimensions (Melzack, 1975).

in acceptance and outcome scores does not allow a definitive test of a mediation effect (for a detailed methodological explanation of causal inference using regression see Chapter 9 in Gelman and Hill, 2007).

Emotions and health status

Correlational analyses indicated that reports of lower distress and more frequent happiness/joy were overall associated with reports of better health status. However not all parameters were consistently significant, e.g. the relations between work status and shame and anxiety, and between medication use and anger, shame and joy. Happiness/joy was the least associated with HSIs (only with disability, work status, affective and total pain quality), which confirms previous accounts of positive emotions as more related to well being than with the emotional impact of chronic pain (Clark et al., 2003).

In contrast, only the use of internal dysfunctional ERSs was significantly related to all HSIs. All ERSs showed consistent relations only with pain-related disability and affective pain quality. Item-level analyses showed only dysfunctional ERSs (rumination and verbal aggression) as related to health status. The functional ERSs were not consistently related to any HSIs. In light of previous analyses of ERQ data in this chronic pain sample, these results further highlight the need to identify ERSs that are specific to this population. The controversies related to the effectiveness of pain coping strategies (discussed in Chapter 5) warn about the difficulties in identifying generally applicable ERSs, especially given their highly contextual nature. The importance of context is similarly expressed in relation to emotion regulation (Gross, 1999), and by the notion of "workability" in ACT (Gillanders, in press).

Controlling for contextual factors, basic emotions accounted for a significant additional proportion of the variance in most HSIs, except medication use: 11–18% for disability, 14–21% for T-PRI, 10–22% for S-PRI, 20–23% for A-PRI, 09–16% for VAS, 14–15% for PPI. These substantial associations highlight the importance of emotions in chronic pain adjustment, although the data support several alternative explanations, as discussed in the context of acceptance. The different emotions explained a common amount of variance in HSIs, as reflected in the non-significant unique contributions for most variables. This suggests that research on individual emotions needs to consider this considerable overlap with other discrete emotions. Not necessarily to control for other emotions or depression (e.g. Burns and Bruehl,

2005) as this might unnecessarily decrease parameter estimates, but to experimentally differentiate between the effects of distinct emotional states (e.g. Quartana and Burns, 2007; Quartana et al., 2007; Burns et al., 2008b) and to acknowledge that, beyond the distinctions between momentary, brief emotional experiences, self-reports using emotion labels may actually refer to the same emotional episode and reflect emotion regulation strategies. The concept of 'discrimination ability' (and the more general affect-related 'emotion granularity', Barrett, 2006b) build upon this particular function of verbal labels, as explained in Chapter 6.

Emotion regulation strategies did not add a significant unique contribution to prediction of HSIs, supporting the previously stated suggestion regarding the limited relevance of the response-focused ERSs for chronic pain sufferers.

Illness perceptions and health status

Adaptors reported significantly better health status compared with non-adaptors, although differences in work status and medication use were not significant in all stages. As stated previously, the heterogeneous structure of the BIPQ recommends examining the individual subscales rather than relying on the inexact data dissection based on the two groups. Thus, correlational analyses of the distinct IPs and HSIs indicated that only emotional IPs (concern and emotional response) and identity IPs were related to most HSIs. One exception was medication use, which was related only to perception of symptoms (identity). While personal control and understanding were not related to any HSI, perceptions of treatment control were related only to affective pain intensity and VAS and perceptions of timeline were related only to pain disability and pain intensity.

This pattern of relations might support various interpretations. In the case of IPs of illness identity, the most obvious interpretation is an overlap with health status, as the question is worded as a self-report of health status: 'How much do you experience symptoms from your chronic pain?'. This is a good example of the interpretation caveats in SRM research, and warns against viewing IPs as 'good' or 'bad' cognitions, independent of the individual's medical condition. All other IPs (except personal control and understanding) may overlap (maybe to a lesser extent) with health status, as the respondents might interpret the questions as referring to the severity of their condition. In particular, treatment control and pain intensity as indicated by the VAS scale are meaningfully related in the sense that, as treatment is directed mostly at reducing pain, respondents access the same information to respond to both questions ('is the pain controlled/reduced?'). It is important to

note that emotion-related IPs (as identified in previous analyses) were also most related to HSIs, which confirms results related to acceptance and emotions, and may also support various alternative explanations, from directional relationships to construct overlap.

None of the causal attributions categories were related consistently with disability, work status, HCU or pain intensity indicators, confirming the limited relevance of causal models in adjustment to chronic pain, as discussed previously.

The results of the MRMs indicated that IPs also increased significantly the amount of variance in HSIs explained: 28–31% for disability, 4–7% for medication use, 20–30% for T-PRI, 5–21% for S-PRI, 24–36% for A-PRI, 33–43% for VAS, 34–41% for PPI. In addition to the substantive interpretation of these relationships within the SRM (regarding the role of the illness model in coping with the condition and the subjective assessment of the health status based on the illness model), the relatively large effect sizes might also be due to similarities in question format particularly for VAS and PPI, which required an overall estimation of the respondent's perception regarding the pain severity (the difficulties of differentiating pain perception from perceptions of related aspects are discussed in detail in Chapter 2).

Comparative analysis

The MRMs exploring the comparative contribution of CPA, discrete emotions and IPs in predicting pain-related disability (RM-SIP) and total pain intensity (T-PRI) indicated that, while emotions and IPs participated significantly in both equations, CPA contributed significantly only to disability when emotion and cognition are controlled for. Also, the percentage of variance explained in disability was higher, compared to pain intensity. Similar results were given by the SEM parameter estimates, which showed all psychological variables more related to disability. The covariance estimates between the main latents indicated the CPA was strongly related to all other latents (all estimates $> .66$), while the relation between emotions and IPs showed the lowest magnitude (.34–.42).

However these estimates need to be treated with caution due to the suboptimal fit of the models (i.e. significant χ^2). This lack of fit might indicate that a different structure could represent the data more adequately. The theoretical issues of content overlap discussed and the previous empirical results also indicate that these constructs are not unidimensional. The GOF indices suggest that the additional paths specified between the model latents are not sufficient to account for

these conceptual overlaps. Moreover, as the questionnaires have distinct response formats, the influence of the self-report method used cannot be distinguished from substantive differences, which highlights the necessity of a multi-trait multi-method study in order to distinguish method and substantive effects based on new measures in which the same response formats are used to measure emotion, cognition and behaviour.

Although this comparative analysis can be considered a limited exploration of the relationships between these concepts, it speaks clearly of the measurement issues involved in studying chronic pain adjustment within an integrative model. Identifying unidimensional constructs and the structural/functional relationships between them is still a matter of future research. According to the CAM, the threat value of the stimuli, the personal characteristics influencing this value and the escape behaviours and affect-laden problem solving that result from the emerging problem definition may be more useful distinctions which the present data cannot differentiate. Further details on the cognition-emotion interactions in health threat (symptom) processing and problem-solving are offered by the SRM (see Chapter 5). Integrating these distinctions in future measurement tools may result in unidimensional constructs in specific assessment settings.

Nevertheless, as discussed in Chapter 2 in relation to pain measurement, the distinctions identified via research and clinical practice are difficult to measure via self-report, as the respondents access a variety of information from different domains when answering a question as simple as 'how painful is it?'. Questions about pain intensity may be answered by accessing information about its emotional impact, about the disability it causes, or about a variety of beliefs related to its consequences, timeline, etc. These issues blur the line between measurement and structure and represent a real barrier to model testing in chronic pain. They also highlight the need to approach pain measurement from the respondent's perspective, and to continue research (both qualitative and quantitative) on pain reporting.

An important step in approaching pain from the sufferer's perspective is identifying intra- and interpersonal variance in chronic pain reports, which was explored in the last two sets of analyses.

9.1.4 Individual growth trends

All psychological and health status indicators showed significant between and within-subject variance during the study period, which underlines the necessity to develop

models that would explain variance at both levels. It is important to note that the actual variance proportions depend also on the test-retest reliability of the measures at 4 to 8-month intervals, therefore RM-SIP values show most variance at the interpersonal level while IPs (single item scales) show comparatively more variance at the intrapersonal level than multi-item scales.

Except pain intensity, no differences in growth trajectories were found at the interpersonal level. Only CPA, anger, sadness, disability and IPs of consequences showed limited changes at group level (increase in CPA and decreases in the rest). These changes can be due to slight clinical improvements, but equally to a tendency to report higher levels of pain severity at first assessment (Strand et al., 2006). In contrast with similar multilevel analyses (Affleck et al., 1992; Zautra et al., 2001, 2005; Kratz et al., 2007) which focused on daily or weekly fluctuations, the present data refer to 4-month intervals. The magnitude of these changes is below what could be considered a relevant clinical improvement, which is to be expected in a heterogeneous sample to which no specific common intervention was applied.

9.1.5 Discrimination ability

In light of the considerable overlap between the concepts used, it is relevant to examine the inter- and intraindividual variation in the ability to distinguish between different aspects of the chronic pain experience, such as between sensory and affective components of pain perception, or between different emotional reactions, or between pain and emotion. A detailed theoretical account of the relevance of this ability is presented in the final section of Chapter 6.

The concept of 'discrimination ability' was supported only in relation to sensory and affective pain reporting. Mean levels of CPA, anger, sadness, shame, joy, dysfunctional ERSs and IPs of concern and emotional impact mediated the associations between sensory and affective pain quality, suggesting that for participants with lower levels of acceptance, and reporting more emotional distress (partly due to chronic pain), the distinction between sensory and affective pain becomes less clear. Moreover, when symptoms are perceived as increasing (IPs of illness identity increase), the distinction also becomes more blurred at an intrapersonal level. These mediation effects are an addition to the main statistical effects of the included moderators and sensory pain quality on affective pain quality.

Interestingly, anxiety levels do not have a moderating effect on this distinction. However, in terms of the ability to differentiate between pain and emotion or between positive and negative emotion, moderating effects of mean pain intensity and mean CPA levels were found only in relation to anxiety, which singles out anxiety as an emotion label for which different mechanisms likely apply. These interactions indicated that, at higher levels of pain intensity and lower levels of CPA, the negative relation between happiness and anxiety is weaker (not stronger, as the 'discrimination ability' concept would predict), which requires a different explanation.

Further investigation of 'discrimination ability' and other possible interpretations of the interaction effects identified here is necessary, and replication with different timeframes, measures and samples is essential. An additional interpretation of the moderation effects related to pain and positive and negative emotion was given by Zautra et al. (2005), who considered two alternative mechanisms, in addition to the role of mood clarity as an interindividual difference: positive affect as a source of resilience weakening the impact of pain on negative emotion, and the role of pain increases in decreasing the differentiation between positive and negative affect. All mechanisms may be valid, however the 'discrimination ability' in our results applies only to the sensory-affective pain distinction, while the interaction between changes in positive affect and changes in pain in predicting negative affect became nonsignificant when mean levels of pain were considered. This difference is likely explained by different time intervals (daily/weekly versus 4-month); differentiation between emotional experiences might be apparent only when shorter time-frames are considered.

The contrasting pattern identified for anxiety may reflect the special meaning of worry as a problem-solving effort in addition to a threat perception label, which is a central tenet of the CAM. In this context, the moderating role of pain intensity and CPA mean levels in the association between happiness and anxiety might actually reflect an increase of the impact of positive affect on reducing problem solving efforts in persons with lower pain severity and higher acceptance. These results are similar to the interaction found by Strand et al. (2007) between pain readiness to change and positive emotion in predicting pain reports, which they interpreted as a synergistic effect of the two predictors. Similarly, the present finding might reflect a vicious cycle effect: higher pain severity (and higher avoidance) levels combined with low frequency of rewarding events lead to more feelings anxiety, worry and fear. A positive interpretation suggests that obtaining even slight decreases in pain severity (and increases in acceptance) might help the positive events have more

impact on decreasing anxiety. These relationships are certainly not unidirectional, as the selection of anxiety as outcome variable was random. Further research is necessary to clarify these alternative mechanisms.

Another important aspect is to distinguish 'differentiation ability' from the concept of 'coherence' used both in the SRM and in emotion regulation literature. These theoretical perspectives assert that lower coherence might be maladaptive, for example that dissociation of response systems might be a mechanism underlying the harmful effects of emotional suppression (e.g. Mauss and Gross, 2004), or in the concept of 'desyncrony' whithin the fear-avoidance model (Lethem et al., 1983). The main theoretical difference between these two concepts is that, while coherence refers to the match between different levels of intrapersonal (e.g. propositional versus schematic) or interpersonal (e.g. patient versus doctor) representations, or between self-report versus physiological processes in emotional responding, the differentiation ability is a uniquely propositional (i.e. linguistic) ability. It refers to the regulatory function of language in relation to behaviour, as a flexible and nuanced labelling of external and internal events allows theoretically the selection of more contextually adequate responses.

If these mechanisms will be reliably confirmed in future research, they might indicate new therapeutic approaches to pain management by increasing the complexity of emotion processing (Zautra et al., 2005) and by focusing equally on accessing sources of positive affect to catalyse the effects of relatively more stable clinical improvements. Separating the overwhelming 'pain problem' into distinct, manageable problems has been a central component of CBT interventions (Turk et al., 1983). However the affective aspects of this differentiation mechanism (i.e. between sensory and affective pain, and between positive and negative affect) are certainly underdeveloped, although acute pain models and animal models of counterconditioning have been considered in the context of the SRM (Leventhal, 1993) and the CAM (Eccleston and Crombez, 1999).

9.1.6 Questionnaire analysis

The main focus of this thesis was to bring together three strands of research based on a detailed conceptual analysis, both theoretically and empirically. In this context, the psychometric analysis of the measures used was instrumental in assessing conceptual clarity in a practical sense. Measurement links theory to its empirical

testing and, although usually addressed separately in the literature, cannot be fully evaluated independently of substantive findings, and vice versa.

Chapter 8 has explored the properties of several validated measures as they apply to the chronic pain population sampled for the present study. Without reiterating the implications of each analysis here, it is important to underline the necessity of further improving the instruments used based on their psychometric performance and the substantive results obtained based on their application. Pain reporting is a difficult domain to assess, and the format of the instruments used participates to a considerable extent in the co-construction of pain experience. The intersubjectivity of pain measurement needs to be acknowledged at both substantive and psychometric levels.

9.2 Overview of the main findings

The above sections briefly discussed the various issues that the present study attempted to examine in the broader domain of psychological adjustment to chronic pain. The study of the specific domains revealed the continuous nature of the constructs (CPA and IPs), attempted to clarify conceptual distinctions (i.e. between CPA and PF) and tested theoretical relations (i.e. between emotions and ERSs) and structural hypotheses (i.e. regarding health status indicators). Together with the psychometric analyses in Chapter 8, these results constituted the foundation on which the interrelations between the psychological concepts and their relations to health status were examined.

This analysis revealed the central role of emotion in all concepts, including acceptance and illness perceptions, which were interpreted within the CAM's description of threat perception and affect-laden problem-solving as a special case of cognitive-affective interactions in responding to health treats (stipulated by the SRM). The role of emotion in health status reporting was emphasised in the subsequent comparative analyses, as was the substantial overlap between acceptance, emotions and illness perceptions. This analysis raised important measurement issues and clarified directions for future research in pain reporting.

The multilevel models confirmed the need to study variation in chronic pain adjustment variables at both intra- and interpersonal levels. They also highlighted the stability of pain experience, also identified in previous analyses. The multilevel

moderation analyses supported the 'differentiation ability' in relation to sensory-affective pain at 4-month intervals, and suggested a different mechanism for the anxiety-happiness relation, interpreted within the CAM as a catalysing effect of lower pain intensity and higher pain acceptance levels on the role of positive feelings in decreasing affect-laden problem-solving efforts.

9.3 Strengths and implications

The comprehensive theoretical review of behaviour, emotion and cognition as reflected in the three main research areas targeted can be considered a main strength of this study, as it offered a broad interpretation framework that guided empirical research and its interpretation. Certainly, the present thesis was not a systematic review, but a narrative literature review which focused on the comparative analysis of these domains. However the detailed account of the complexity of chronic pain adjustment revealed a diverse range of issues that were further tested and explored in the empirical study. Another strength is considering both measurement and substantive issues within the same analysis, which helped assess the accuracy and limitations of the possible interpretations. I would argue that the importance given to empirical evidence in research often leads to a neglect of theoretical and measurement issues, and the approach adopted in this study attempted to balance these three equally important aspects. A third strength of this thesis is the diversity of empirical issues explored. Given the complexity of the phenomenon studied, a more flexible approach to research and data analysis (both in terms of hypotheses and statistical methods used) was considered more adequate.

The implications for research and clinical practice of each of the findings reported have been briefly discussed in the appropriate sections. As a general comment, it is relevant to mention that the various results of this study may further guide research on pain reporting, but also lead to new approaches to pain management if the mechanisms identified are replicated reliably in future studies. The analysis recommends at least two main future directions for research within the CAM (and SRM). First, the threat value of the pain signal and the emotional aversive response to pain can be considered a common aspect of CPA, emotion and IPs, and further investigation of their possible distinct components may be useful. Second, the contextual variability of chronic pain adjustment is highlighted by all frameworks studied, and suggests that further research should focus on moderation mechanisms and clearly delimit specific contexts for which theoretical predictions are tested. The clinical implications of these results are also related to the link between emotion and

pain, and suggest the possible usefulness of including basic emotion in pain adaptation work, not only in terms of depression and anxiety, but also anger, shame and happiness. Combining acceptance approaches and research on emotion regulation strategies might also prove helpful, given the conceptual similarities identified in this study.

9.4 Limitations and directions for future research

The results of the present study need to be interpreted within the limits set by the philosophical and methodological assumptions detailed in Chapter 6. The realist stance adopted and the higher level of generalisation targeted by this integrative model imply that the results have a limited appplication at the level of individual or therapeutic models of chronic pain adjustment. They identify areas of conceptual overlap, clarify distinctions and structural relations and suggest areas of further research and possible mechanisms for pain-affect relations. However they are not informative regarding treatment options in particular situations, which are better addressed by individual or therapeutic models or by the specific theoretical models considered here. As previously stated, an integrative attempt inevitably loses vast amounts of information by focusing on a limited number of elements, and examines different models based on a single set of criteria which may not represent all the models considered with equal accuracy. Therefore, it is not intended to replace, but to complement these models.

Another important caveat regarding the results of the present study refers to the limits of generalizability regarding population sampling, choice of measured variables and measurement tools, and occasions of measurement. First, the sampling strategy used has selected participants that were willing to respond to a relatively long questionnaire, especially in the first stage, and might therefore differ from the general chronic pain population in terms of medical status or personality traits; the influence of these characteristics could not be assessed. Although the response rate is comparable to other studies in the field, it raises the question of the generalizability to chronic pain populations which include persons for whom illness severity, familial conditions or personality traits might have led to a refusal to participate. The users of NHS and support organisations services, despite showing no difference regarding relevant measured variables, might have differed in other, unmeasured, aspects thus biasing results.

Second, the present study, although it has measured a broad range of variables, did not include psychological concepts such as readiness to change, pain coping strategies, self-efficacy which are considered relevant concepts for chronic pain research (Keefe et al., 2004). Some of them partly overlap with the included concepts, but it is likely that some omissions led to limited description of the dynamics of adjustment to chronic pain.

One of the main limitations is the lack of thorough consideration of the social and environmental factors that participate in shaping the experience of chronic pain. Although present in an indirect way through participants' self-reports regarding socio-economic status, external events, perceived support, these external factors would need to be measured independently of the individual's perceptions in order to be considered independent indicators of the third aspect of the biopsychosocial model of chronic pain. Without such data, the models discussed still have a 'correlated error' component due to the method used (self-report) and to the possibility that other sources of variance might influence the report on which all variables included here were based. As both CAM and SRM are to a considerable extent contextualist approaches, the present study does not cover the full extent of these models.

Another limitation is not including measures related to personal goals. Hamilton et al. (2004) noted that, apart from the event-person interactions, an often neglected aspect includes the personal goals, which influence both the impact of stressful events on the person's emotional state, and the overall adjustment strategy in chronic pain. From this perspective, research on reinforcement sensitivity (and the behavioural inhibition and behavioral activation systems; Gray and McNaughton, 2003) may be relevant for emotional adjustment to chronic pain. One related limitation is not considering the intentionality ('aboutness') of emotions. As Consedine and Moskowitz (2007) stated, the role of discrete emotions on health status depends considerably on the specific situation that elicits them. For example fear of pain might lead to avoidance of painful medical examinations, but also to seeking medical diagnosis and treatment as a means to prevent increases in pain. Okifuji et al. (1999) distinguished between several targets of anger in chronic pain patients (from self-directed anger to anger towards health care providers and persons responsible for the onset of pain) and highlight the distinct implications each might have for adjustment to chronic pain. This distinction would be important in future studies.

On the other hand, such wide coverage is not feasible due to methodological and logistic reasons. Respondent burden is also an important ethical consideration which recommends limiting the length of the assessment and the effort required from the study participants (Ulrich et al., 2005). One would need to choose one level of explanation and place each study within a bigger framework. Skevington (1995, p. 184) describes four levels of analysis of the psychosocial influences on chronic pain experience: individual processes affected by social processes, interpersonal behaviours, group and intergroup behaviour, and higher order factors affecting psychological processing (health culture, history, ideology, politics etc.). This study is located at the first level, however with a limited consideration of social factors.

Third, there are several limitations due to the data collection methods used. All measurement tools selected rely on self-report, and the bias due to retrospective assessments and to the social aspects inherent in pain reporting (Skevington, 1995) could not be controlled for. The use of measurement methods other than retrospective self-report, while increasing the difficulty of data collection, would have added different perspectives free of these sources of bias. Moreover, the wording of some items (e.g. the categories chosen for reporting current vocational/employment status, current and past treatment methods, or the subjective rating of annual income) might benefit from further improvements in future studies.

Fourth, the results of this study are obviously limited to 4-month intervals. In this context, it is important to compare with other studies that have selected different measurement intervals. As seen in relation to the multilevel moderation analyses, distinctions between positive and negative affect are apparent in daily and weekly data (Zautra et al., 2001, 2005), but do not replicate when discrete positive and negative emotions are considered at 4-month intervals. On the other hand, back pain patients did show different trajectories of improvement or chronicity at intervals of 1, 2 and 5 years in a recent study by Von Korff and Miglioretti (2005), contrasting with the stable 1-year trajectories of our heterogeneous chronic pain sample. Therefore, different dynamics of chronic pain adjustment characterise different populations and time frames, recommending against unfounded generalisations.

Many additional analyses may be performed, such as exploring other possible moderation roles of the contextual factors (gender, pain location, etc.), controlling for pain intensity in predicting disability, achieving a simplified prediction model for HSIs, testing alternative directional models based on the cross-sectional data, exploring growth models at a latent level. However the conceptual and theoretical focus of the thesis, together with the space limitations, prevented a more detailed

exploration of the data set. Also, although several alternative models were considered where appropriate, the possibility of untested alternative models with better data fit cannot be excluded. Many SEMs presented had suboptimal fit, which on one hand is expected as SEM is well-known to represent a more stringent test (see Appendix B), but on the other hand highlights the need to continue the search for better explanatory models.

An important issue of which this study offered only a limited account is the issue of causality. Although this can be considered a limitation of the study and analysis, it was to a certain extent a deliberate choice. Given that no substantial change was found between stages, the data can be considered in essence cross-sectional. Directional relations in cross-sectional designs are justified only if the causal influence is considered instantaneous, or if the causal variable does not change from the moment of causal influence to the moment of concurrent measurement (MacCallum and Austin, 2000), conditions which do not apply to the relations between psychological variables and health status. On the contrary, these variables are endogenuous, in the sense that they are all describing the individual's status from different perspectives and are susceptible to reciprocal causation, as explained in Chapter 6. In contrast to the frequent use of cross-sectional data to support theories proposing directional claims (even if the difference between correlation and causation is usually mentioned in reports), the present study intended to limit statistical modelling within the constraints of the data properties, in order to avoid any unfounded inferences.

Moreover, the measurement issues identified in the comparative analysis of acceptance, emotions and illness perceptions in predicting health status prevented the analysis from testing other structural relationships. If suboptimal fit is obtained for the measurement model, some authors recommend against pursuing model testing at a structural level (Mulaik and Millsap, 2000). Although other analysis strategies are available, in our case the results obtained regarding the overlaps between the concepts analysed are by themselves informative and in my opinion they suggest that acceptance, emotions and illness perceptions cannot be considered distinct concepts linked by a yet unknown structural network, but complementary perspectives on a similar substantive issue. This interpretation recommends further research that would distill the content of these concepts into one or several unidimensional aspects of chronic pain adjustment and would improve measurement of these aspects. Only at this stage would testing of alternative structural relations be appropriate, and SEM methodology could help both measurement and structural research.

Another related limitation is assuming causal homogeneity, which underlies model development in MRM and SEM. However, Turk and Okifuji (2001) highlight the danger of considering chronic pain adjustment as a phenomenon with similar manifestations in all sufferers, which they name "the patient uniformity myth". The intra- and interpersonal moderation analyses presented in this study are a step towards unveiling the variability of chronic pain adjustment. Further exploration of moderation mechanisms is however necessary, and might invalidate the current explanatory models.

9.5 Conclusion

The findings of this study can be considered a diverse range of empirical and theoretical components converging towards an integrative account of chronic pain adjustment, which gives emotion a central position and in the same time describes its multifaceted nature. Rather than aiming for a parsimonious model at a general level, the approach here was to characterise the complexity of the phenomenon and interpret its various aspects within an overarching framework based on Eccleston and Crombez's (1999) CAM and Leventhal et al.'s (1992) SRM.

Selecting between models and concepts based on the strength of association with health status indicators is a frequently applied strategy, especially based on cross-sectional data. It does not account for common theoretical elements and also might exclude important unique contributions of the excluded variables or possible functional relationships. A different approach was adopted here, which consisted in a detailed conceptual analysis and comparison. The concepts studied offered complementary information and represented parallel tests of similar mechanisms. Importantly, the analysis highlighted the need to further test other possible distinctions based on the CAM and SRM.

The emotional aspects of chronic pain adjustment proved to be a common theme for acceptance, emotions and illness perceptions. In this context, emotion regulation becomes an important research direction, especially given that the response-oriented strategies included in the present study were only partially related to health status. Also, clinical applications would benefit from the integration of the three perspectives and the focus on regulating the emotional impact of chronic pain via various antecedent- or response-focused strategies and perhaps by developing the ability to distinguish between various emotional experiences, or between pain and emotion.

The role of context was another important issue that linked the three domains and is also integrated in the CAM and SRM. Distinctions that cannot be detected at a general level might be essential in specific situations, and research at both levels is valuable. The moderation analyses presented represented a step in this direction, and the results justify further moderation research (and also experimental and longitudinal studies with shorter time intervals) and underline the necessity of a flexible application of research findings in clinical practice.

Appendix A

Data collection documents

The next 31 pages contain the following documents in their original format:

a) the "Living with chronic pain" questionnaire (first stage);

b) the letter of invitation and patient information sheet;

c) the consent form;

d) the pain location questionnaire as used in the online version and for chronic pain support organisations;

e) the final report sent to participants;

f) the ethics approval documents.

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

GENERAL INFORMATION:

People adapt in different ways to their health condition, depending on their personality, life experience and habitual ways of dealing with everyday events.

The goal of the present research is to identify what makes your experience of chronic pain unique, in order to develop ways of customising treatment according to each patient's needs.

Your participation in this survey is very important for the ongoing efforts of improving health care services for people with chronic pain.

The present survey intends to measure several aspects of your emotional life and ways of thinking and dealing with your health condition and asks for a few details on your current health status and treatment history.

This study will work best if you attempt to answer all questions. However, if you prefer to omit any question for any reason, please feel free to do so.

Thank you for taking part in this survey!

Notes:

1. All information you are providing by answering this questionnaire will be treated as confidential. Anonymity will be ensured by storing contact details separate from answers, which will be identified by an individual code.

2. If you have any questions related to your participation in this study at anytime, please contact Alexandra Dima at: e-mail a.dima@sms.ed.ac.uk, tel. 07903385294, or the following address:

Clinical and Health Psychology
School of Health in Social Sciences
University of Edinburgh
Medical School
Teviot Place
Edinburgh
EH8 9AG

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

ABOUT YOU

Before you begin, we would like to ask you some questions about yourself.
Please tick the box corresponding to the answer that fits best or fill in the space provided.

What is your **gender**? ☐ Male ☐ Female

What is your **date of birth**? _____/ _____/ _____
Day / Month / Year

What is the highest **education** you received?
☐ None at all
☐ Primary school
☐ Secondary school
☐ College / University

What is your **marital status**?
☐ Single ☐ Separated
☐ Married ☐ Divorced
☐ Living as married ☐ Widowed

What is your current **vocational** status?
☐ Full-time working ☐ Full-time training
☐ Part time working ☐ Part time training
☐ unable to work because of pain
☐ unable to work due to other reason (e.g. housewife/person, retired)

Is your household's total **annual income** ☐ Below Average ☐ Average ☐ Above Average?

What is your **ethnic origin?** ☐ White ☐ Asian ☐ Afro-Caribbean
☐ Other (please specify_____)

What is your **nationality**? _____

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

The following questions will enquire about your life in general.

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Please tick the box corresponding to the answer that fits best.

	Never true	Very rarely true	Seldom true	Sometimes true	Often true	Almost always true	Always true
1. I am able to take action on a problem even if I am uncertain what is the right thing to do.	☐	☐	☐	☐	☐	☐	☐
2. I often catch myself daydreaming about things I've done and what I would do differently next time.	☐	☐	☐	☐	☐	☐	☐
3. When I feel depressed or anxious, I am unable to take care of my responsibilities.	☐	☐	☐	☐	☐	☐	☐
4. I rarely worry about getting my anxieties, worries, and feelings under control.	☐	☐	☐	☐	☐	☐	☐
5. I'm not afraid of my feelings.	☐	☐	☐	☐	☐	☐	☐
6. When I evaluate something negatively, I usually recognize that this is just a reaction, not an objective fact.	☐	☐	☐	☐	☐	☐	☐
7. When I compare myself to other people, it seems that most of them are handling their lives better than I do.	☐	☐	☐	☐	☐	☐	☐
8. Anxiety is bad.	☐	☐	☐	☐	☐	☐	☐
9. If I could magically remove all the unpleasant experiences I've had in my life, I would do so.	☐	☐	☐	☐	☐	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

We all experience lots of different feelings or emotions.
For example, different things in our lives make us feel happy, sad, angry and so on…
The purpose of the following scale is to find out about HOW OFTEN you experience certain emotions.
We would like to know about how you feel IN GENERAL.
For each question please circle ONE number only between 1 and 7 to indicate how you feel.

IN GENERAL, I FEEL THIS EMOTION:

	never			sometimes			very often
ANGER	1	2	3	4	5	6	7
DESPAIR	1	2	3	4	5	6	7
SHAME	1	2	3	4	5	6	7
ANXIETY	1	2	3	4	5	6	7
HAPPINESS	1	2	3	4	5	6	7
FRUSTRATION	1	2	3	4	5	6	7
MISERY	1	2	3	4	5	6	7
GUILT	1	2	3	4	5	6	7
NERVOUSNESS	1	2	3	4	5	6	7
JOY	1	2	3	4	5	6	7
IRRITATION	1	2	3	4	5	6	7
GLOOMINESS	1	2	3	4	5	6	7
HUMILIATED	1	2	3	4	5	6	7
TENSE	1	2	3	4	5	6	7
LOVING	1	2	3	4	5	6	7
AGGRESSION	1	2	3	4	5	6	7
MOURNFUL	1	2	3	4	5	6	7
BLAMEWORTHY	1	2	3	4	5	6	7
WORRIED	1	2	3	4	5	6	7
CHEERFUL	1	2	3	4	5	6	7
DISGUST (i.e. repulsion)	1	2	3	4	5	6	7

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

The following questions ask you to think about HOW OFTEN you do certain things IN RESPONSE to your emotions. You do not have to think about specific emotions but just how often you GENERALLY do the things listed below. Please tick the box corresponding to the answer that fits best.
We all respond to our emotions in different ways so there are no right or wrong answers.

In GENERAL how do you respond to your emotions?	**Never**	**Seldom**	**Often**	**Very often**	**Always**
1. I talk to someone about how I feel	☐	☐	☐	☐	☐
2. I take my feelings out on others verbally (e.g. shouting, arguing)	☐	☐	☐	☐	☐
3. I seek physical contact from friends or family (e.g. a hug, hold hands)	☐	☐	☐	☐	☐
4. I review (rethink) my thoughts or beliefs	☐	☐	☐	☐	☐
5. I harm or punish myself in some way	☐	☐	☐	☐	☐
6. I do something energetic (e.g. play sport, go for a walk)	☐	☐	☐	☐	☐
7. I dwell on my thoughts and feelings (e.g. It goes round and round in my head and I can't stop it)	☐	☐	☐	☐	☐
8. I ask others for advice	☐	☐	☐	☐	☐
9. I review (rethink) my goals or plans	☐	☐	☐	☐	☐
10. I take my feelings out on others physically (e.g. fighting, lashing out)	☐	☐	☐	☐	☐
11. I put the situation into perspective	☐	☐	☐	☐	☐
12. I concentrate on a pleasant activity	☐	☐	☐	☐	☐
13. I try to make others feel bad (e.g. being rude, ignoring them)	☐	☐	☐	☐	☐
14. I think about people better off and make myself feel worse	☐	☐	☐	☐	☐
15. I keep the feeling locked up inside	☐	☐	☐	☐	☐
16. I plan what I could do better next time	☐	☐	☐	☐	☐
17. I bully other people (e.g. saying nasty things to them, hitting them)	☐	☐	☐	☐	☐
18. I take my feelings out on objects around me (e.g. deliberately causing damage to my house or outdoor things)	☐	☐	☐	☐	☐
19. Things feel unreal (e.g. I feel strange, things around me feel strange, I daydream)	☐	☐	☐	☐	☐
20. I telephone friends or family	☐	☐	☐	☐	☐
21. I go out and do something nice (e.g. cinema, shopping, go for a meal, meet people)	☐	☐	☐	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

The following question will inquire about events that may or may not have happened to you during the last six months. You may give the answer by simply stating, "yes" or "no" to each item.
Have any of the following events or problems happened to you during the last 6 months?

LIFE EVENT	YES	NO
You yourself suffered a serious illness, injury or assault.	☐	☐
A serious illness, injury or assault happened to a close relative.	☐	☐
Your parent, child or spouse died.	☐	☐
A close family friend or another relative (aunt, cousin, grandparent) died.	☐	☐
You had a separation due to marital difficulties.	☐	☐
You broke off a steady relationship.	☐	☐
You had a serious problem with a close friend, neighbour or relative.	☐	☐
You became unemployed or were seeking work unsuccessfully for more than one month.	☐	☐
You were sacked from your job.	☐	☐
You had a major financial crisis.	☐	☐
You had problems with the police and a court appearance.	☐	☐
Something you valued was stolen or lost.	☐	☐
Other (please state) ……	☐	☐
……	☐	☐
……	☐	☐
You secured a new or better job.	☐	☐
Your financial status has improved.	☐	☐
You, a close friend or a close relative had a child.	☐	☐
You, a close friend or a close relative got married.	☐	☐
You made up with a close friend or a relative.	☐	☐
Other (please state) ……	☐	☐
……	☐	☐
……	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

This set of questions focuses on several aspects of your medical history, treatment and current health status.

Please tick the boxes corresponding to the answers that describe your experience or fill in the space provided.

How long have you had your pain? ____ years ____months____ weeks

How did your pain start?

☐ Gradually
☐ Suddenly
☐ Suddenly and gradually worse
☐ As a result of an accident
☐ As a result of an illness
☐ Following surgery
☐ Other (please state) …….

What previous treatments you have followed?

☐ pain relief medication
☐ antidepressants
☐ sleeping tablets
☐ surgery
☐ physiotherapy
☐ psychological therapies
☐ pain management programmes
☐ complementary therapies (acupuncture, homeopathy, herbal remedies etc.)
☐ Other (please state) …….

What is your current treatment?

- ☐ pain relief medication
- ☐ antidepressants
- ☐ sleeping tablets
- ☐ surgery (waiting for)
- ☐ physiotherapy
- ☐ psychological therapies
- ☐ pain management programmes
- ☐ complementary therapies (acupuncture, homeopathy, herbal remedies etc.)
- ☐ Other (please state) …….

For each of the next 3 sets of questions, please tick only one box corresponding to the answer that fits best.

During the last 3 months, how often did you take the following steps to ease your pain?

	I didn't	Once	2-5 times	More than 5 times
Contact your GP	☐	☐	☐	☐
Go to an emergency service	☐	☐	☐	☐
Stay in hospital for more than a few hours	☐	☐	☐	☐
Go to a physiotherapist	☐	☐	☐	☐
Go to an acupuncturist	☐	☐	☐	☐
Go to a chiropractor	☐	☐	☐	☐
Go to a homeopathist	☐	☐	☐	☐
Go to a practicer of naturopathy	☐	☐	☐	☐
Go to other therapist (please state)……	☐	☐	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

During the last 2 weeks, how often did you use the following medication to ease your pain?

	Several times a day	Daily	Several times a week	Weekly	More seldom	Never
Aspirin / paracetamol	☐	☐	☐	☐	☐	☐
Combined analgesics	☐	☐	☐	☐	☐	☐
Combined muscle relaxants	☐	☐	☐	☐	☐	☐
Tranquilisers/sedatives	☐	☐	☐	☐	☐	☐
Hypnotics	☐	☐	☐	☐	☐	☐
Ointments	☐	☐	☐	☐	☐	☐
Natural medicines	☐	☐	☐	☐	☐	☐
Other (please state)……..	☐	☐	☐	☐	☐	☐
…….	☐	☐	☐	☐	☐	☐
…….	☐	☐	☐	☐	☐	☐

During the last 2 weeks, how often did you use the following methods to ease your pain?

	Several times a day	Daily	Several times a week	Weekly	More seldom	Never
Use heat	☐	☐	☐	☐	☐	☐
Rest	☐	☐	☐	☐	☐	☐
Physical activity (walking, jogging, swimming etc.)	☐	☐	☐	☐	☐	☐
Other (please state)……..	☐	☐	☐	☐	☐	☐
…….	☐	☐	☐	☐	☐	☐
…….	☐	☐	☐	☐	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

The following is a list of common problems. Please indicate if you currently have the problem in the first column. If you do not have the problem, skip to the next problem.
If you do have the problem, please indicate in the second column if you receive medications or some other type of treatment for the problem.
In the third column indicate if the problem limits any of your activities.
Finally, indicate all medical conditions that are not listed under "other medical problems" at the end of the page.

	Do you have the problem?		Do you receive treatment for it?		Does it limit your activities?	
PROBLEM	No	Yes	No	Yes	No	Yes
Heart disease	N	Y	N	Y	N	Y
High blood pressure	N	Y	N	Y	N	Y
Lung disease	N	Y	N	Y	N	Y
Diabetes	N	Y	N	Y	N	Y
Ulcer or stomach disease	N	Y	N	Y	N	Y
Kidney disease	N	Y	N	Y	N	Y
Liver disease	N	Y	N	Y	N	Y
Anaemia or other blood disease	N	Y	N	Y	N	Y
Cancer	N	Y	N	Y	N	Y
Depression	N	Y	N	Y	N	Y
Osteoarthritis, degenerative arthritis	N	Y	N	Y	N	Y
Rheumatoid arthritis	N	Y	N	Y	N	Y
Other medical problems (please write in)						
……	N	Y	N	Y	N	Y
……	N	Y	N	Y	N	Y
……	N	Y	N	Y	N	Y
……	N	Y	N	Y	N	Y
……	N	Y	N	Y	N	Y

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

This set of questions has been designed to tell us more about your pain. It is important that you tell us how your pain feels now.

	None	Mild	Moderate	Severe
1. THROBBING	☐	☐	☐	☐
2. SHOOTING	☐	☐	☐	☐
3. STABBING	☐	☐	☐	☐
4. SHARP	☐	☐	☐	☐
5. CRAMPING	☐	☐	☐	☐
6. GNAWING	☐	☐	☐	☐
7. HOT-BURNING	☐	☐	☐	☐
8. ACHING	☐	☐	☐	☐
9. HEAVY	☐	☐	☐	☐
10. TENDER	☐	☐	☐	☐
11. SPLITTING	☐	☐	☐	☐
12. TIRING-EXHAUSTING	☐	☐	☐	☐
13. SICKENING	☐	☐	☐	☐
14. FEARFUL	☐	☐	☐	☐
15. CRUEL-PUNISHING	☐	☐	☐	☐

Indicate on this line how bad your pain is - at the left end of line means no pain at all, at right end means worst pain possible.

No Pain _____ Worst Possible Pain

Which word best describes your pain right now?

Mild	Discomforting	Distressing	Horrible	Excruciating
1	2	3	4	5

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Please tick the box corresponding to the answer that fits best.

	Never true	Very rarely true	Seldom true	Sometimes true	Often true	Almost always true	Always true
1. I am getting on with the business of living no matter what my level of pain is.	☐	☐	☐	☐	☐	☐	☐
2. My life is going well, even though I have chronic pain.	☐	☐	☐	☐	☐	☐	☐
3. It's ok to experience pain.	☐	☐	☐	☐	☐	☐	☐
4. I would gladly sacrifice important things in my life to control this pain better.	☐	☐	☐	☐	☐	☐	☐
5. It's not necessary for me to control my pain in order to handle my life well.	☐	☐	☐	☐	☐	☐	☐
6. Although things have changed, I am living a normal life despite my chronic pain.	☐	☐	☐	☐	☐	☐	☐
7. I need to concentrate on getting rid of my pain.	☐	☐	☐	☐	☐	☐	☐
8. There are many activities that I do when I feel pain.	☐	☐	☐	☐	☐	☐	☐
9. I lead a full life even though I have chronic pain.	☐	☐	☐	☐	☐	☐	☐
10. Controlling pain is less important that any other goals in my life.	☐	☐	☐	☐	☐	☐	☐
11. My thoughts and feelings about pain must change before I can take important steps in my life.	☐	☐	☐	☐	☐	☐	☐
12. Despite the pain, I am now sticking to a certain course in my life.	☐	☐	☐	☐	☐	☐	☐
13. Keeping my pain level under control takes first priority whenever I'm doing something.	☐	☐	☐	☐	☐	☐	☐
14. Before I can make any serious plans, I have to get some control over my pain.	☐	☐	☐	☐	☐	☐	☐
15. When my pain increases, I can still take care of my responsibilities.	☐	☐	☐	☐	☐	☐	☐
16. I will have better control over my life if I can control my negative thoughts about pain.	☐	☐	☐	☐	☐	☐	☐
17. I avoid putting myself in situations where my pain might increase.	☐	☐	☐	☐	☐	☐	☐
18. My worries and fears about what pain will do to me are true.	☐	☐	☐	☐	☐	☐	☐
19. It's a relief to realize that I don't have to change my pain to get on with my life.	☐	☐	☐	☐	☐	☐	☐
20. I have to struggle to do things when I have pain.	☐	☐	☐	☐	☐	☐	☐

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

When you are in pain you may or may not find it difficult to do some of the things you normally do. This list contains some sentences that people have used to describe themselves when they are in pain. When you read them you may find that some stand out because they describe you <u>over the past few days including today.</u>

As you read the list, think of yourself. When you read a sentence that describes you put a tick against it. If the sentence does not describe you then leave the space blank and move onto the next one. Remember only to tick the sentence if you are sure that it describes how you have been recently.

1. I stay at home most of the time because of my pain.	
2. I change position frequently to try and get comfortable.	
3. I walk more slowly than usual because of my pain.	
4. Because of my pain I am not doing any of the jobs that I usually do around the house.	
5. Because of my pain I use a handrail to get upstairs.	
6. Because of my pain I lie down to rest more often.	
7. Because of my pain I have to hold on to something to get out of an easy chair.	
8. Because of my pain I try to get other people to do things for me.	
9. I get dressed more slowly than usual because of my pain.	
10. I only stand up for short periods of time because of my pain.	
11. Because of my pain I try not to bend or kneel down.	
12. I find it difficult to get out of a chair because of my pain.	
13. I find it difficult to turn over in bed because of my pain.	
14. My appetite is not very good because of my pain.	
15. I have trouble putting on my socks (stockings / tights) because of my pain.	
16. I only walk short distances because of my pain.	
17. I sleep less well because of my pain.	
18. Because of my pain I get dressed with help from someone else.	
19. I sit down for most of the day because of my pain.	
20. I avoid heavy jobs around the house because of my pain.	
21. Because of my pain I am more irritable and bad tempered with people than usual.	
22. Because of my pain I go upstairs more slowly than usual	
23. I stay in bed most of the time because of my pain.	
24. I am in pain almost all of the time.	

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

For the following questions, please circle the number that best corresponds to your views:

How much does your chronic pain affect your life?

0 1 2 3 4 5 6 7 8 9 10

no affect at all — severely affects my life

How long do you think your chronic pain will continue?

0 1 2 3 4 5 6 7 8 9 10

a very short time — forever

How much control do you feel you have over your chronic pain?

0 1 2 3 4 5 6 7 8 9 10

absolutely no control — extreme amount of control

How much do you think your treatment can help your chronic pain?

0 1 2 3 4 5 6 7 8 9 10

not at all — extremely helpful

How much do you experience symptoms from your chronic pain?

0 1 2 3 4 5 6 7 8 9 10

no symptoms at all — many severe symptoms

How concerned are you about your chronic pain?

0 1 2 3 4 5 6 7 8 9 10

not at all concerned — extremely concerned

How well do you feel you understand your chronic pain?

0 1 2 3 4 5 6 7 8 9 10

don't understand at all — understand very clearly

How much does your chronic pain affect you emotionally? (e.g. does it make you angry, scared, upset or depressed?)

0 1 2 3 4 5 6 7 8 9 10

not at all affected emotionally — extremely affected emotionally

Please list in rank-order the three most important factors that you believe caused your chronic pain.

The most important causes for me:

1. _____
2. _____
3. _____

Do you consider that the causes of your chronic pain are rather:

- ☐ medical
- ☐ social
- ☐ psychological ?

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: March - June 2007

CONTACT INFORMATION

This survey is the first of a 3-stage study. The next two stages of the survey will consist in sending you one questionnaire each time with similar questions for you to answer. The goal of the next two stages is to identify how different aspects of your emotional life and ways of thinking and dealing with your health condition change in time. We are also interested to see if these changes influence your health status in addition to the treatment you receive.

We would like to contact you by phone and post in 3-4 months time for the second stage, and in 3-4 months after that for the third stage. If you agree in taking part in the next stages, please specify your preferred method of being contacted and provide your contact details.

☐ Yes, I would like to take part in the next 2 stages and I would like to be contacted by:
- ☐ Phone
- ☐ Post
- ☐ E-mail
- ☐ Other (please specify)

☐ No, I would not like to take part in the next 2 stages

My contact details are:
Name: ……
Address: ……
Telephone: ……
E-mail: ……..

If your contact details change during this period, please let us know. Please bear in mind that you have the right to withdraw from this study at any time. If you decide that you would like not to be contacted by us for the next stages, please inform us of your decision.

We would like to inform your GP of your participation in this study. Please provide the details of your GP:
Name: ……
Address: ……
Telephone: ……
Other: ……

We would like to present to you a brief description of the findings of this study at the end of the research project. Please specify if you agree to be sent a letter by post by July 2008 presenting the results and relevance of findings.

☐ Yes, I would like to receive a letter presenting the study findings.
☐ No, I would not like to receive a letter presenting the study findings

---

Did someone help you to fill out this form?.....
How long did it take to fill this form out? .....
Do you have any comments about the assessment? .....
.....
.....

THANK YOU FOR YOUR HELP!

University Hospitals Division

NHS Lothian

Emotions and living with chronic pain

Letter of invitation

Dear Mr/Mrs/Miss ……

You have an appointment at the Pain Clinic coming up shortly and we wanted to use this opportunity to ask you to consider becoming involved in a research project. The Lothian Chronic Pain Service and the University of Edinburgh, Section of Clinical and Health Psychology, would like to invite you to participate in a study of the relationship between emotions and health in chronic pain.

You may recently have completed questionnaires for another of our research projects. If so, thank you very much for participating. You can also be involved in this one as well, whether or not you have been involved in previous research or not.

We enclose a copy of the participant information sheet, two consent forms, a questionnaire and a prepaid return envelope. Please study these documents carefully and decide whether you want to take part in this research. If you decide to participate, you will find further information in the documents enclosed.

We would like to mention that this study is completely independent of your treatment within the Lothian Chronic Pain Service and that a decision not to take part or to withdraw from the study at any time in the future will absolutely not affect the standard of care you receive.

We regard this study as relevant for further improving treatment methods and thank you in advance for considering this invitation.

Yours sincerely

Alexandra Dima
Researcher

Dr. David Gillanders
Chartered Clinical Psychologist

Dr. Ivan Marples
Consultant in Anaesthesia and Pain Medicine

SECTION OF CLINICAL & HEALTH PSYCHOLOGY
School of Health in Social Science
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 0131-651 3972
Fax: 0131-651-3971

The University of Edinburgh

PATIENT INFORMATION SHEET

1. Study title

Emotions and living with chronic pain

2. Invitation

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

3. What is the purpose of the study?

Previous research has shown that each person's unique experience of chronic pain is related to the way one feels, thinks and deals with their health condition. This study aims to analyse how these relationships develop in time and how emotions, thoughts, attitudes and health status influence each other.

To be able to study this, it is necessary to gather information about your emotions, thoughts, attitudes and health status at three stages, separated by time intervals of around 3-4 months. Therefore, the total duration of the study is 6-8 months, starting from the first time you participate until you complete the last questionnaire of the three.

4. Why have I been chosen?

You are one of the service users of the chronic pain centres of the Lothian Health Board. As one of the aims of this research is to improve the services you receive, we are particularly interested in finding out more about your experience of chronic pain. We have asked permission from your pain team to contact you and invite you to take part. We are hoping that around 200 service users will agree to participate in all 3 stages of our study.

5. Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. You will also be given a copy of the signed consent form to keep. If you decide to

take part you are still completely free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will absolutely not affect the standard of care you receive.

6. What will happen to me if I take part?

If you agree to take part, please bring the consent forms with you to the next appointment at the clinic. There you will meet the researcher who will answer any additional questions you might have. If at this stage you agree to participate in this study, you will be asked to sign the consent forms. We will give you a questionnaire and ask you to fill it in as soon as you have some time available for this. The questionnaire takes around 60 minutes to fill in. You will be advised to answer it during the following week and post it back to us in the prepaid return envelope provided.
As part of the questionnaire you will be asked to provide your and your GP's contact details. We will inform your GP of your participation in this study.
You are advised to contact the researcher in case of changes in your contact details or interest in this study.
After around 3-4 months we will contact you by phone to update your details accordingly. If you are still interested in participating, we will invite you to take part in the second stage. You will be given a similar questionnaire to fill in and return by post within a week. If we will not receive your letter within 2 weeks, we will send you a reminder together with another copy of the questionnaire and a prepaid return envelope. If we will not receive a completed questionnaire after sending you the reminder, we will assume that you do not wish to take part and we will not contact you again.
After another interval of around 3-4 months, the same procedure as in stage 2 will be repeated.
After the data will be analyzed, we will send you a brief report of the general results of the study by post.

Stage	Time period	Our responsibilities	Your responsibilities
1	March - June 2007	- give you a patient information sheet and 2 consent forms - answer all your questions about the study - give you a questionnaire, and a prepaid return envelope	- answer the questionnaire as soon as you can - send us the completed questionnaire and the consent form by post in the prepaid return envelope
2	July - October 2007	- contact you and update your interest in the study and your contact details - send you a questionnaire and a prepaid return envelope - send you a reminder if we don't receive your letter within 2 weeks	- contact us in case your interest in the study or your contact details change - answer the questionnaire as soon as you can - send us the completed questionnaire by post in the prepaid return envelope
3	November 2007 - February 2008	- similar to stage 2	- similar to stage 2
End of study	June - July 2008	- send you a brief report of the results	

7. What else do I have to do?

Participating in this research will NOT involve any lifestyle and dietary restrictions, and you are encouraged to follow your treatment as prescribed by the service providers.

11. **What are the possible disadvantages and risks of taking part?**

As your participation in this study involves only answering a questionnaire at 3 times separated by 3-4 month intervals, the only risks of taking part could be related to any questions that you might consider too sensitive, intruding or upsetting.
We have taken all precautions to exclude any unnecessary sensitive questions. We use only questions that have been previously used in similar studies and received good feedback. However, we realize that people could feel different about emotional and health related topics. Therefore, if you will consider one of the questions as being inappropriate, please feel free not to give any answer.

12. **What are the possible benefits of taking part?**

The information we get from this study will help us improve the health care services and treat future patients with chronic pain better.

Taking part in this study will not involve direct benefits while the research is progressing. Still, we hope that the questions we ask will provide good food for thought and maybe help you a bit in your own search for understanding and coping with this condition. We also hope that the brief report you will receive at the end will provide useful information.

15. **What if something goes wrong?**

If you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for a legal action but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanisms should be available to you.

16. **Will my taking part in this study be kept confidential?**

All information collected about you will be kept strictly confidential. Your questionnaire results will be identified by a special code. The data relating the code with your personal details will be kept separate from the questionnaire responses. All data is stored in secure locations and only authorized personnel have access to it. Data will be kept for 5 years and then be destroyed.

17. **What will happen to the results of the research study?**

The results of the study will be presented to the pain team within the Lothian Health Board, will be published in peer reviewed journals in the area of pain research, health psychology and emotion research and will be presented to fellow researchers and practitioners via conference presentations and posters. Your participation will not be identified in any report/publication.

You may express you interest to obtain a copy of the published results by contacting the researcher.

18. **Who is organising and funding the research?**

The research is sponsored and funded by the University of Edinburgh, as part of supporting the studies of the researcher towards an academic qualification (PhD by research). It is organized in collaboration with the Lothian Chronic Pain Service.

19. **Who has reviewed the study?**

The study has been reviewed by the Lothian Research Ethics Committee.

20. **Contact for Further Information**

Alexandra Dima
Address: Clinical and Health Psychology
School of Health in Social Sciences
University of Edinburgh
Medical School
Teviot Place
Edinburgh
EH8 9AG

Tel : 07903385294

E-mail: a.dima@sms.ed.ac.uk

If following this presentation you have decided to take part in this study, we would like to thank you for taking part!

If you have decided that it is not possible for you to participate, we thank you for considering this invitation!

SECTION OF CLINICAL & HEALTH PSYCHOLOGY
School of Health in Social Science
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 0131-651 3972
Fax: 0131-651-3971

The University of Edinburgh

Patient Identification Number:

CONSENT FORM

Title of Project: Emotions and Living with Chronic Pain

Name of Researcher: Alexandra Dima

1. I confirm that I have read and understand the patient information sheet dated ..... (version .....) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am absolutely free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree to take part in the above study. ☐

_____	_____	_____
Name of Participant	Date	Signature
_____	_____	_____
Researcher	Date	Signature

1 for participant; 1 for researcher;

Code | | | | |

EMOTIONS AND LIVING WITH CHRONIC PAIN

Longitudinal Survey 2007 - 2008 - 1st Stage: July - October 2007

What is the **current location** of your pain?	Do you feel pain in this location? **0** - No **1** - Yes	I had this pain for the last **1** - Less than 3 months **2** - 3-5 months **3** - 6 months - 1 year **4** - 1-5 years **5** - More than 5 years	I feel it **1** - Continuously **2** - Every day **3** - More than once a week **4** - Once a week **5** - More than once a month **6** - Once a month **7** - Less than once a month	and it usually lasts **1** - a few minutes **2** - a few hours **3** - one day **4** - a few days **5** - a few weeks **6** - more than a few weeks **7** - continuously
Head	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Neck	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Shoulder	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Elbow	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Wrist / hand	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Chest	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Abdominal	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Upper back	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Lower back	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Hip/thigh	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Knee	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Ankle/foot	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Other pain 1 (please specify) …….	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Other pain 2 …….	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Other pain 3 …….	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Other pain 4 …….	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7
Other pain 5 …….	0 1	1 2 3 4 5	1 2 3 4 5 6 7	1 2 3 4 5 6 7

SECTION OF CLINICAL & HEALTH PSYCHOLOGY
School of Health in Social Science
Medical School
Teviot Place
Edinburgh
EH8 9AG

The University of Edinburgh

Re: LIVING WITH CHRONIC PAIN Survey 2007-2008 – research report for participants

Dear ……..,

I am contacting you because you kindly participated in the above survey. After almost two years of data collection and one year of data analysis, I am now able to present to you a brief summary of our findings. You have been one of the 265 people who responded to our questionnaires, and your participation has allowed us to examine many specific questions regarding the complex and difficult issue of the psychological aspects of pain. We thank you for your time and openness!

I would like to share with you some of the findings of the study. They are based on the analysis of the questionnaire responses, on many other previous studies and theories I have studied, and also on the useful and interesting comments, thoughts and suggestions that many of you have given me during data collection. Our hope and intention is that the information and understanding we have gained from this contact with all of you will lead to more focused research efforts. Our findings may not have an immediate influence on the way chronic pain is managed, but all of you have given us valuable information that will enable us to build more reliable assessments, focus our approach and further test new methods of coping with pain.

Previous research has shown that each person's experience of chronic pain is unique, and is related to the sufferer's emotions, thoughts and ways of coping with their health condition. In general, these three aspects (emotions, thoughts and behaviour) have been studied separately, and one aim of our study was to find out what they have in common. The results indicate that the common element is the *emotional response to pain*.

Emotion is indeed part and parcel of the pain experience. The fundamental purpose of the pain response is to give us warning of physical threat, and it prompts us to search for a way to draw back quickly, in order to avoid danger. As one leading researcher has stated, pain interrupts any ongoing activities, and "chronic pain means chronic interruption". This interruption is a normal response to pain, and is closely related to how a person experiences and understands pain, and what that person does because of the pain. The link between pain and the interruption of ongoing activity is so strong that often it is hard to distinguish the different aspects of the pain – the sensations, emotions, thoughts and actions. It can often feel as if the pain dominates everything, and that there is no choice about how to experience and manage that challenge.

However, we need to search for ways to diminish the impact that chronic pain has on our lives. The data we have collected from you all, together with the data we have looked at in previous studies, strongly suggest that trying to influence the emotions that pain provokes could well be useful. I am not suggesting that one should strive to get rid of the anxiety, frustration or feeling of loss that can be a prominent aspect of the emotional response to chronic pain. I am rather referring to a range of possibilities of influencing the emotions we experience, depending on the situation.

For example, appreciating that some emotions are a natural result of pain or realising that some might be short lived may already lead to feeling less dominated by their presence. Seeking to understand their different messages might lead to finding specific solutions to the situations that generated them, irrespective of whether pain contributed or not. Putting things into perspective or changing plans might also help avoiding some distressing situations altogether. Other emotions might diminish if they are discussed with family or friends, while others might be worth experiencing if by doing this one achieves something one considers important and valuable. For some, being able to focus strongly on enjoying the good things in life is very helpful, however brief or mundane such experiences might be. Choosing the most suitable way of dealing with emotions ultimately depends on the specific details of each situation.

During the three stages of this study, only slight improvements in health have taken place. This means that we cannot answer with certainty any questions about causes and effects of such changes. It also means that we need to continue the search for effective methods of intervention. However, our findings support previous research in indicating that there are several specific areas for further study, for example in training the ability to perceive separately the pain sensations and the associated emotions. Although it is premature to communicate these results, we hope to follow them up in future studies in a more applied and intervention-related setting.

Another important part of the analysis of your responses looked closely at the usefulness and precision of the questions that we used. Most of the questions were part of established questionnaires that are frequently used in chronic pain assessment. The analysis of your responses, as well as your additional comments, helped us to identify ways to improve the clarity and focus of these questionnaires. This means that your contributions will help in the task of improving chronic pain assessment in the future.

What will happen next? Using publications and oral presentations, we plan to communicate the results obtained to the research and medical community, in order to raise awareness and stimulate further research on the emotional side of chronic pain. We will continue to perform additional analyses on the data collected, and also to set up other studies to address more specifically the issues we have identified.

On a final note, I would like to reassure you that your contact details have been kept safe until now, and will be deleted immediately after sending this letter to you. The data linking your name with the questionnaire responses, which have been kept separately, will also be destroyed. The questionnaire responses will be kept in safe storage for another 5 years within the University of Edinburgh, according to the agreed research data policy.

I would like to thank you again for your participation. I assure you that the information you have given will be used to further our understanding and improve our response to those who are suffering chronic pain now, and those who will be suffering in the future.

Wishing you all the best,

Alexandra Dima
PhD student, School of Health in Social Science, University of Edinburgh
a.dima@sms.ed.ac.uk

Lothian NHS Board

Mrs Alexandra Lelia Dima
PhD student
University of Edinburgh
School of Health in Social Sciences
Medical School, Teviot Place
Edinburgh
EH8 9AG

Deaconess House
148 Pleasance
Edinburgh
EH8 9RS
Telephone 0131 536 9000
Fax 0131 536 9009
www.nhslothian.scot.nhs.uk

NHS
Lothian

Lothian Local Research Ethics Committee 01

Telephone: 0131 536 9050
Facsimile: 0131 536 9346
Email: elaine.racionzer@lhb.scot.nhs.uk

22 January 2007

Dear Mrs Dima

Full title of study:	**Longitudinal study on the role of emotions, acceptance and illness perceptions in influencing the health status of adults living with benign chronic pain**
REC reference number:	**06/S1101/57**

Thank you for your letter of 19 December 2006, responding to the Committee's request for further information on the above research and submitting your revised documentation.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

There are only two small points which the members of the sub-committee have suggested you amend.

a) On page 4 of the patient information leaflet the word 'Place' has been omitted in 'Teviot Place'
b) On the Contact Information page of the Living with Chronic Pain, Lognitudinal Survey 2007 – 1st Stage: January – March, rather than use the word 'cognitive' could you use a phrase that would be understandable.

You do not need to send these documents for further review.

Ethical review of research sites

The Committee has not yet been notified of the outcome of any site-specific assessment (SSA) for the research site(s) taking part in this study. The favourable opinion does not therefore apply to any site at present. I will write to you again as soon as one Local Research Ethics Committee has notified the outcome of a SSA. In the meantime no study procedures should be initiated at sites requiring SSA.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Application		07 November 2006
Investigator CV		07 November 2006
Protocol	0.1	07 November 2006
Covering Letter		
Letter from Sponsor		07 November 2006
Questionnaire: Stage 1 - Jan-March	2	19 December 2006
Letter of invitation to participant	2	19 December 2006
GP/Consultant Information Sheets	2	19 December 2006
Participant Information Sheet: Patient Information Sheet	2	19 December 2006
Participant Consent Form: Patient Consent Form	2	19 December 2006
Response to Request for Further Information		19 December 2006
Longitudinal Survey 2007 Stage 2& 3	0.1	07 November 2006
Supervisor CV		
Insurance Details		28 July 2006

Research governance approval

The study should not commence at any NHS site until the local Principal Investigator has obtained final research governance approval from the R&D Department for the relevant NHS care organisation.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

06/S1101/57	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely
pp

Mr Nicholas Grier
Chair

Enclosures: *Standard approval conditions SL-AC2*
Site approval form

Copy to: Prof Mick Power
University of Edinburgh
School of Health in Social Science, The University of Edinburgh
Medical School, Teviot Place, Edinburg, EH8 9AG

NHS Lothian Research & Development Group

Lothian NHS Board

Lothian Research Ethics Committees
Deaconess House
148 Pleasance
Edinburgh
EH8 9RS
Telephone 0131 536 9000
Fax 0131 536 9346
www.nhslothian.scot.nhs.uk

NHS
Lothian

Mrs Alexandra Lelia Dima
PhD student
School of Health in Social Sciences
Medical School, Teviot Place
Edinburgh
EH8 9AG

Date 17 April 2007
Our Ref 07/S1101/17
Enquiries to Chris Graham
Extension 89027
Direct Line 0131 536 9027
Email chris.graham@lhb.scot.nhs.uk

Dear Mrs Dima,

Study title: **Longitudinal study on the role of emotions, acceptance and illness perceptions in influencing the health status of adults living with benign chronic pain**
REC reference: **06/S1101/57**
Amendment number: **1**
Amendment date: **26 March 2007**

The above amendment was reviewed at the meeting of the Sub-Committee of the REC held on 28 March 2007.

Ethical opinion

The members of the Committee present gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

The Committee did comment that the PIS does include a lot "you are advised" text which might be thought of as bossy. The letter of invitation could also be less blunt and more friendly.

Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Participant Information Sheet	4	26 March 2007
Letter of Invitation	4	26 March 2007
Notice of Substantial Amendment (non-CTIMPs)		26 March 2007

NHS
Lothian

Covering Letter		28 March 2007

Membership of the Committee

The members of the Committee who were present at the meeting are listed on the attached sheet.

R&D approval

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

06/S1101/57 Please quote this number on all correspondence

Yours sincerely

C. Graham

Chris Graham
Committee Co-ordinator

Enclosures List of names and professions of members who were present at the meeting and those who submitted written comments

University Hospitals Division

Queen's Medical Research Institute
47 Little France Crescent, Edinburgh, EH16 4TJ

HAC/SM/approval/Dima/2e

24th January 2007

Mrs Alexandra Leila Dima
University of Edinburgh
School of Health in Social Sciences
Medical School,
Teviot Place
Edinburgh
EH8 9AG

Dear Mrs Dima

MREC No:	**N/A**
CRF No:	**N/A**
LREC No:	**06/S1101/57**
R&D ID No:	**2006/P/PSY/23**
Title of Research	Longitudinal study on the role of emotions, acceptance and illness perceptions in influencing the health status of adults living with benign chronic pain.
Protocol No/Acronym:	**N/A**

The above project has undergone an assessment of risk to NHS Lothian and review of resource and financial implications. I am satisfied that all the necessary arrangements have been set in place and that all Departments contributing to the project have been informed.

I note that this is a single centre study sponsored by **University of Edinburgh.**

On behalf of the Chief Executive and Medical Director, I am happy to grant management approval from NHS Lothian to allow the project to commence, subject to the approval of the appropriate Research Ethics Committee(s) having also been obtained. You should note that any substantial amendments must be notified to the relevant Research Ethics Committee and to R&D Management with approval being granted from both before the amendments are made.

Please note that under Section A, Q35, NHS Lothian provides indemnity for negligence for NHS and Honorary clinical staff for research associated with their clinical duties. It is not empowered to provide non-negligent indemnity cover for patients. NHS Lothian does not provide indemnity against negligence for healthy volunteer studies. This is the personal responsibility of both NHS and honorary employees and is usually arranged with a medical defence organisation or through the University of Edinburgh.

This letter of approval is your assurance that NHS Lothian is satisfied with your study. As Chief Investigator or local Principal Investigator, you should be fully committed to your

NHS

Lothian

RESEARCH & DEVELOPMENT OFFICE
Room E1.12

Tel: 0131 242 3330
Fax: 0131 242 3343
Email:
R&DOffice@luht.scot.nhs.uk

Director:
Professor Heather A Cubie

R&D Governance Manager
DrTina McLelland

PA to Professor Cubie & Dr McLelland:
Miss Jill Dobbie

Commercial Research Manager:
Dr Douglas Young

Research Manager Capacity & Capability:
Dr Janet Hanley

Research Governance Co-ordinator:
Mrs Susan Shepherd

Information & Knowledge Manager
Miss Heather Coupar

AHP Research & Development Facilitator:
Dr Colette Fulton

Accountant:
Ms Sheevaun McIntyre

Assistant Accountant:
Mr Neil McLean

Trial Support Officer:
Ms Dorothy Aitken

Office Manager:
Mrs Glynis Omond

Administrative Assistant:
Ms Sandra Muir

St John's – Administrator:
Mrs Anne Addison

responsibilities within the Research Governance Framework for Health and Community Care, an extract of which is attached to this letter.

Yours sincerely

Heather A Cubie

Professor Heather A Cubie
R&D Director

Enc	Research Governance Certificate	☑ (to be signed and returned)
	NRR authorisation	☑ (to be signed and returned)
	Tissue Policy (if applicable)	☐
	MTA (if applicable)	☐ (to be signed and returned)

Copies *Administrators, Research Ethics Committee*
Professor M Power, University of Edinburgh

University Hospitals Division

Queen's Medical Research Institute
47 Little France Crescent, Edinburgh, EH16 4TJ

HAC/SM/app-lrecamend

3rd May 2007

Mrs Alexandra Lelia Dima
Clinical and Health Psychology
University of Edinburgh
Medical School
Teviot Place
Edinburgh
EH8 9AG

Dear Mrs Dima

LREC No:	**06/S1101/57**
R&D Project ID No:	**2006/P/PSY/23**
Title of Research	Longitudinal study on the role of emotions , acceptance and illness preceptions in influencing the health status of adults living with benign chronic pain

I am writing in reply to recent correspondence in relation to the following amendment(s) to the above project.

Amendment:	***Amendment No1 Dated 26th March 2007.*** ***As per*** ***Letter listing the changes and explaining both the previous and new protocol V1 Dated 26th March 2007.*** ***Modified PIS version4 Dated 26th March 2007.*** ***Modified letter of invitation V4 Dated 26th March 2007.***

We have now received a copy of the amendment(s) and assessed any consequential changes in Division resource use. I confirm that Division management approval is extended to cover the specific changes intimated. You should be aware that approval for this amendment must also be received from Lothian Research Ethics Committee before it is implemented

Yours sincerely

Heather A Cubie

Professor Heather A Cubie
R&D Director

Copies:

NHS
Lothian
RESEARCH & DEVELOPMENT OFFICE
Room E1.12

Tel: 0131 242 3330
Fax: 0131 242 3343
Email:
R&DOffice@luht.scot.nhs.uk

Appendix B

SEM Data analysis strategy

B.1 Introduction

The increasing use of SEM methodology in psychological research has led to both considerable progress in substantive areas and problematic issues in its use (MacCallum and Austin, 2000). Due to the controversial issues associated with SEM model testing and reporting, it is necessary to detail the methodological position taken in this study regarding data analysis within this domain, from data preparation and model specification to model interpretation and modification. To illustrate the current state of the art regarding practical applications of SEM in chronic pain, several examples are presented in the final section.

B.2 Data preparation

As with all analyses based on linear equations, it is necessary to examine data for linearity, normality and presence of outliers, as well as for multicollinearity and singularity (Ullman, 2001). These checks were performed for all analyses and were reported where relevant. Regarding the requirement of multivariate normality, one exception is the robust correction available in EQS for several estimation methods. As many variables in the present dataset show significant departures from normality, the robust ML method was used in all analyses, as recommended by Bentler (2004) and Ullman (2001). No data transformation was performed. Missing values treatment was reported in Appendix C, while issues of model identification were mentioned only if problematic (for brevity).

Given that the use of correlation matrices leads to biased parameter estimates (MacCallum and Austin, 2000), covariance matrices based on raw data were used in all analyses. This issue is particularly relevant when using ordinal data. In such cases,

there is the option of considering polychoric correlation matrices, but according to Bentler (2004) this means ignoring variance differences between variables (p. 145) plus the assumptions of normality and large enough sample size (p. 150). On the other hand, if the item distributions are relatively normal (especially symmetrical) and have more than 3 categories, treating ordinal variables as continuous will lead to only minor distortions (p. 148–9). For binary data however, both types of matrices were used where possible, since considering this data as continuous may lead to more substantial distortions and also impact on fit indices.

I have also addressed in relevant sections of the thesis further recommendations by Boomsma (2000) regarding reporting information about the theoretical framework of the models and the theoretical implications of model testing results, the necessity of applying SEM, the population and sample of study, the characteristics of the dataset.

B.3 Power in SEM

Model testing is essentially dependent on the sensitivity of the test to detect model misspecifications. However in SEM power does not have a simple relation with sample size, number of parameters, significance levels and effect sizes (as for multiple regression, for example). Unfortunately power is rarely discussed in SEM applications, and most authors only discuss it (often erroneously) as a justification for significant χ^2 results (Tomarken and Waller, 2003).

Power (for both likelihood ratio χ^2 tests and χ^2 difference tests between nested models) varies with many factors: sample size, the difference between the true value and the specified value of a parameter, the location of the parameter in the overall model, the reliability and error of the manifest variables, the number of manifest variables per latent, overall magnitude of covariances between variables, parsimony, normality of distributions of manifest variables, estimation method, missing data treatment and in longitudinal studies number of time points assessed (Tomarken and Waller, 2003). Although power cannot be calculated for many fit indices due to unknown distributional properties, simulation studies show they are sensitive to many of the above mentioned factors (Tomarken and Waller, 2003). The authors recommend the use of additional diagnoses apart from model fit statistics and computing power and conducting simulations on the effects of different factors on the sensitivity of measures of fit. However these computation methods add an

unnecessary level of complexity to the analyses. They represent a area under development in SEM and are not yet applicable to routine implementation (Bentler, 2000; Barrett, 2007).

Bentler (2004) recommends testing of multiple models and examination of standard errors for assessing variability of estimates in small samples (fewer than 100 cases); if some models are rejected and standard errors are relatively small adequate sample size can be inferred. This approach was used in the present study. It is worth mentioning that the sample size of the present study is not considered small in the literature. According to a review of the recent CFA applications by Jackson et al. (2009), 20% of studies have a sample size of less than 200. As model size impacts significantly on power, a special concern was to limit the number of measured variables included in the model to the minimum necessary. Jackson et al. (2009) report a median number of 17 (12 as the 25^{th} percentile, and 24 as the 75^{th} percentile) for variables used in recent CFA studies.

B.4 Model specification and analysis strategy

Using specifications of directional relations in cross-sectional designs is justified only if some theoretical assumptions are met. Either that the causal influence is virtually instantaneous, or that the causal variable does not change from the moment of causal influence to the moment of concurrent measurement. If none of these is met, estimates in a cross-sectional design are highly biased (MacCallum and Austin, 2000). Therefore, theoretical justifications for causality were given if directional relations were used cross-sectional models, and equivalent models (with directional relations replaced with covariances between latents) were discussed where appropriate.

In both cross-sectional and longitudinal designs, the validity of the model depends on the assumption that all relevant variables are included, to rule out the possibility of a spurious relationship between the stated causes and effects (MacCallum and Austin, 2000). The great majority of SEM applications do not include known relevant variables, which results in biased parameter estimates and standard errors, and fit indices are sensitive only partially to this type of misspecification; on the other hand, increasing the number of measured variables included in the model impacts negatively on the sensitivity of the tests to detect misspecifications (Tomarken and Waller, 2003). Thus, additional relevant factors measured were included in the models tested where possible, acknowledging their limitation to estimate a higher degree

of complexity. Also, the limits of generalizability regarding population sampling, choice of measured variables and measurement tools, and occasions of measurement were addressed directly, as recommended by MacCallum and Austin (2000).

MacCallum and Austin (2000) recommend testing alternative models as the best strategy in data analysis, since testing a single model is too restrictive and model generation based on an initial model tends to capitalise on chance. This strategy is particularly relevant in terms of taking into account one of the main limitations of SEM: the existence of alternative models that might explain the relations between the measured variables better or equally well (Tomarken and Waller, 2003). Without a proper treatment of this possibility (for example by identifying equivalent models or testing qualitatively different nonnested models), models are subject to confirmation bias, especially if accompanied by a lax application of criteria for adjudging model fit (MacCallum and Austin, 2000). The use of experimental or longitudinal design is highly recommended in order to limit the number of plausible alternative models (Tomarken and Waller, 2003). Based on these recommendations, several models were developed where relevant, and equivalent models discussed. Also, models tested in the cross-sectional design were reconsidered based on the longitudinal data where possible.

An important and controversial issue regarding the available analysis strategies in SEM is the distinction between measurement and structural components of a model. Some authors (Mulaik and Millsap, 2000) recommend testing the measurement model (specifying only correlated disturbances between all latent variables) before testing the composite model (which includes the hypothesised unidirectional paths between latents). Other authors (Hayduk and Glaser, 2000b,a) argue against this distinction and advise considering the composite model globally[1]. This controversy is relevant for interpreting model fit and diagnosing model misfit. Tomarken and Waller (2003) note that the distinction between measurement and structural components is only one of the multiple possible ways of parsing composite models. They describe a case in which good fit at the measurement level can mask ill fit at the structural level (which is usually the main theoretical focus of the analysis) and therefore recommend reporting model fit at both levels.

This distinction is particularly important for this study as the constructs and measures are already developed. There are three strategies available: using the total

[1]This dispute would require much more space to expose in fairness. Suffice it to say that arguments exist for both strategies in certain situations, and both have limitations. Also, other strategies are available for modeling, as described by Bollen (2000).

scores in path models (irrespective of the internal structure of the questionnaires), using all questionnaire items or selecting a set of best indicators from the questionnaire items.

The first strategy has several drawbacks, as path models hold additional assumptions: error-free measurement, uncorrelated error terms, unidirectionality of relationships (Schreiber et al., 2006). These assumptions are rarely met in practice, especially in longitudinal models (Maruyama, 1998), therefore using only manifest variables is not recommended in SEM. Consequently, using total questionnaire scores might bias estimates by not accounting for measure validity, possible lack of construct unidimensionality and even content overlap of selected items.

Using all questionnaire items as manifest variables in composite models makes testing these assumptions possible. However, this would increase the number of estimated parameters and lower the power of the analysis. Moreover, CFA tests show suboptimal fit for the majority of the measures used (see Chapter 8).

The remaining option is using only a set of indicators. This strategy relates to another important SEM controversy: the selection and optimum number of indicators of a latent variable. Some authors are supportive of single indicators (Hayduk and Glaser, 2000b), and some of multiple-indicator latents (Mulaik and Millsap, 2000), while others advise the use of item parcels - aggregates (sum or average) of items from the same scale (MacCallum and Austin, 2000). Parceling has multiple drawbacks, such leading to biased estimates under certain conditions and hiding various model misspecifications (Hall et al., 1999; Little et al., 2002), and will not be considered further.

Selection of the best indicators from questionnaire items is a delicate enterprise. The highest loading in a CFA model is not considered a good criterion, as the model is not complete (and parameters can be misleading) unless the relevant structural relations are included (cause or effect latents; Beckie and Hayduk, 1997). The criteria proposed are "based on the methodology that provided the data (i.e., clear and appropriate wordings of questions, appropriately scaled answer categories, sufficient variance, etc.)" (Hayduk, 1996, p. 25–30). On the other hand, criticisms of item selection include the subjectivity of the item selection process, the limitations imposed to the construct's definition by insufficient sampling of its content area, the limits to generalisation resulting from the use of a different construct definition when compared to other studies.

Given all these considerations, the following strategy was applied in the present study: use of questionnaire scores in regression analyses and path models, followed by use of selected items in SEM analyses where appropriate. The selection of four items and equal numbers of positive and negative items was preferred based on arguments regarding the possibility to test latent unidimensionality detailed by Mulaik and Millsap (2000) (although see Hayduk and Glaser, 2000a) and regarding modeling method bias as explained by Billiet and McClendon (2000). If measures of fit indicate potential problems with the resulting models, a complete diagnosis includes a test of the measurement model (as a preferred model parsing method given the state of the substantive area) and further tests of the model starting with single indicator latents, as recommended by Hayduk (1996, p. 29).

B.5 Model reporting and interpretation

Recommendations on model reporting converge on several aspects: complete model specification including indicators for each latent variable, the type of data used, the software and method of estimation, complete results including measures of fit with confidence intervals and parameter estimates with confidence intervals or standard errors (MacCallum and Austin, 2000; Boomsma, 2000; McDonald and Ho, 2002; Schumacker and Lomax, 2004; Bentler, 2007; McIntosh, 2007; Jackson et al., 2009). Model specification in a path diagram format is preferred (Boomsma, 2000). Parameter estimates are best reported in table format for the relations between manifest and latent variables and in path diagram format for latent variable relations (McDonald and Ho, 2002).

In terms of measures of fit, reports should include results of the likelihood ratio χ^2 test (including degrees of freedom and p-values); however, p-values should not necessarily be considered as the final indicator of model fit (McIntosh, 2007). Although χ^2 has several limitations including its tendency to detect even small discrepancies given a large enough sample size, a significant χ^2 should not be dismissed as invariably an artifact of a big sample size if sample size is above 200 (a common error in the literature, according to Barrett, 2007).

Reporting all fit indices (minimum two) is recommended given their controversial status (MacCallum and Austin, 2000; McDonald and Ho, 2002; Tomarken and Waller, 2003), but accept/reject decisions based on some thresholds are not (Markland, 2007). Although several cutoff criteria were proposed (for example, Hu and Bentler (1999) recommend a cutoff of .95 for incremental fit indices such as NNFI

and CFI and of .05 for RMSEA), the power of fit measures to detect model misspecifications varies considerably depending on the characteristics of the model therefore other authors advise against using these stringent criteria indiscriminately (Marsh et al., 2004) or even banning the use of fit indices (Barrett, 2007).

Other diagnoses are also recommended: standardized root mean square residual (SRMR) or average absolute standardized residual (and the largest several residuals in a correlation metric), results of Lagrange Multiplier and Wald tests, Fisher's C test, Heywood cases, latent multicollinearity (Bentler, 2007; McIntosh, 2007). For reasons of brevity, these were reported only where relevant. In addition, reporting covariance or correlation matrices is advised; these were included in Appendix D.

Model interpretation should consider both measures of global fit and parameter estimates. Good overall fit does not imply automatically strong effects, which are assessed by examining parameter estimates and residual variances in dependent variables (MacCallum and Austin, 2000). There are several recommendations regarding model interpretation if measures of fit indicate considerable discrepancies between the model implied and the data based covariance matrices. First, if the χ^2 test is significant, the parameters cannot be trusted as they may be based on biased estimates. Also, the χ^2 difference test cannot be used to justify the selection of one of the nested models (Yuan and Bentler, 2004). In these cases, a proper diagnosis is recommended in relation to the substantive theory and many authors strongly advise against considering close fit as satisfactory, as this hinders the advancement of a field (e.g. McIntosh, 2007, p. 861). On the other hand, model testing needs to set theoretically relevant and achievable goals: "the best one can hope for is to identify a parsimonious, substantively meaningful model that fits observed data adequately well" (MacCallum and Austin, 2000). The balance between rigorous model testing and unrealistic goals lies in the careful examination of diagnostic criteria in light of substantive theory.

I have attempted to follow all these recommendations in reporting and interpreting SEM results throughout the data analysis chapters[2].

[2]To allow interpretation of the effect sizes in a common metric, such as "small" for $< .10$, medium for around .30 and large for $> / = .50$ (Kline, 2004), standardized parameter estimates were reported. Standard errors of unstandardized estimates were examined in each case, but were only reported where relevant.

B.6 Model modification

Warnings regarding model modification are given across the methodological literature (MacCallum and Austin, 2000; Boomsma, 2000; Tomarken and Waller, 2003). Model modification should be based on substantive theory, not only data driven, and replicated on a new sample to check generalizability of the new model (MacCallum and Austin, 2000; Tomarken and Waller, 2003). Also, introducing modifications based on modification indices and χ^2 difference tests starting from an ill-fitting model may be misleading (Yuan and Bentler, 2004). As mentioned before, a different strategy for modifying composite models is to run further tests starting with single indicators (Hayduk, 1996).

If model modification is attempted, it is recommended to report all model modifications and also the similarity of the estimates of a priori parameters before and after modification; close values would indicate "that the model was incomplete, but not fundamentally biased" (Bentler, 2007, p. 826). In this case, replication on a different sample is necessary for cross-validation of the new model.

B.7 SEM and chronic pain research

Despite its limitations and caveats and the multiple methodological requirements presented above, SEM has often been regarded as a panacea in many substantive domains including chronic pain, leading to misinterpretations. For example, Skevington (1995, p. 55) presented SEM as a solution to many problems encountered in pain assessment: the difficulty to measure pain could be solved by modeling pain as a latent trait, while the difficulty of inducing pain experimentally could be solved by using non-experimental data in SEM. She cited the study by Holroyd et al. (1992) as an example of SEM application who helped test and refine theory and thus clarify controversial results in prior literature, in this case the Pain Rating Index structure of the McGill Pain Questionnaire. As discussed previously, SEM does not hold any a priori answers for either measurement error or the disadvantages of using nonexperimental data in pain research.

In fact, Holroyd et al.'s (1992) study is a good example of the complexity of using SEM in pain research. SEM was actually only used here in a limited application as part of a wider analysis of MPQ. It compared three confirmatory factor models identified in earlier studies (with 1, 3 and 4 factors) as reflecting the structure of MPQ. Even if they interpreted the 4-factor model as being the best fitting factor

structure for their data sets, their results did not provide a decisive solution, only a different perspective on the still controversial issue of the psychometric properties of MPQ. Moreover, their use of SEM methodology could be critiqued. For example their interpretation of the significant χ^2 difference test for comparing goodness-of-fit between two models as indicating improvements in model specification (in the absence of a non-significant χ^2 goodness-of-fit test) is still a matter of dispute nowadays in SEM literature, as even the 4-factor model can be considered inadequate. In addition, the authors themselves warned against overconfidence in the results of this method. For example, they mentioned that the values for the correlations between latent factors are upper-bound estimates rather that the real values, due to the correction for attenuation; such words of caution often get lost in the praise of latent trait models as a cure for measurement error.

SEM has been infrequently applied in chronic pain research. However, to illustrate the complexities of using this methodology, several studies are briefly described and critiqued next. The focus on the limitations of the presented studies does not intend to diminish their contribution to the field of chronic pain, but to highlight the difficulties of implementing this methodology and to fully use its potential.

An early application of SEM in chronic pain is Rudy et al.'s (1988) study testing the cognitive-behavioural mediation model of pain and depression, which supported a total mediation of the relation between pain and depression by perceptions of increased life interference and reduced personal control. Although the study was highly innovative at the time, there are several limitations worth discussing. First, the authors employed total scores as manifest indicators for latent variables, and thus did not address the possible content contamination acknowledged in the depression-pain measurement literature (Pincus and Williams, 1999). Second, only one model was tested against a null model (assuming no relationships), and no alternative models were specified. Given the use of cross-sectional data, an equally valid model (actually equivalent statistically) would specify directional relationships from depression to pain. Moreover, as there is no time sequence, the data can also be interpreted as the structure of conceptual similarities in the measures employed which address various facets of the overall pain experience. The authors did acknowledge the limitations inherent in the use of concurrent correlational data.

Asmundson and Taylor (1996) used SEM on data from a musculoskeletal pain sample to test the role of anxiety sensitivity in exacerbating fear of pain, and its influence via the mediating role of fear of pain on pain-related avoidance, over and above the role of pain severity. In this study, the latents were constructed based on subscale

scores and other single-item variables, for example pain severity had as manifest variables the sensory subscale of the MPQ and a question about pain duration; this allowed only a partial test of the measurement component of the model. Only one goodness-of-fit index was reported (NNFI= .92), and no additional diagnostics were reported. The authors acknowledged the data were cross-sectional and that other factors might have intervened (such as fear of other consequences, expectations, beliefs, coping). The study was replicated for headache pain by Norton and Asmundson (2004), employing a similar strategy, with similar drawbacks (except reporting a broader range of fit indices). As an example of the possible consequences of the limited testing of the measurement component, the parameter estimate reported for the relation between fear of pain and avoidance was .90 (.83 in the first study), indicating latent multicollinearity and thus suggesting that an alternative model with a single latent combining the indicators of these two concepts might fit the data equally well, or better. The possible problems at the measurement level in this second study were reflected in the unexpectedly small (e.g. .03) or large (e.g. 1.21) standardized loadings of some indicators on their latents. Although the possibility of alternative models was mentioned, only one model was tested.

The sequential processing model (Price and Harkins, 1992; Price et al., 2001) was also tested using SEM. Wade et al. (1996) modeled the serial relationships between pain stages using pain intensity, pain unpleasantness, cognitive evaluation (appraisals of interruption, difficulty and concern for future), secondary affect (combined ratings of pain-related emotions, i.e. depression, anxiety, frustration, anger, fear), and behaviour as latent variables, each measured by single-item manifest variables (except pain behaviour indicators, which were subscale scores). They started with an exploratory analysis on half of the sample, which led to the elimination of several indicators and the cognitive evaluation latent; the resulting model, although it did show an optimal fit on the exploratory data[3], showed a better fit to the confirmatory data ($\chi^2(40) = 62$, $p = .02$, NFI= .97, NNFI= .95). A reanalysis of an extended sample (Price et al., 2001, p. 68–70) compared three alternative models with all concepts included and found support for the partial mediation by cognitive evaluation of the relation between unpleasantness and secondary affect[4]. The use of SEM in this research programme has many methodological strengths, such as the comparative testing of alternative models, the use of single items for

[3]The authors repeated here the controversial argument regarding the dependency of the χ^2 test on sample size.

[4]Although none of the models optimally fit the data in this sample (N=1647), the authors used the χ^2 test to compare nested models, which can be considered controversial according to newer methodological research.

manifest variables, the detailed consideration of the measurement and structural levels, splitting the sample for exploratory and confirmatory analysis, the analysis of parameter indicators in addition to global model fit, and the acknowledgement of other possible intervening factors[5].

Davis et al. (2000) combined EFA with SEM in an attempt to identify the underlying factors behind three frequently used psychological instruments (MMPI-2, MPI, BDI) and described the relations between them in a sample of patients with headache and orofacial (myofascial, neuropathic and neurovascular) pain. They first performed an EFA on half of the sample using selected subscale scores from MMPI-2 and MPI, the total BDI score and the scores on a VAS scale for functional limitation and identified a 3-factor solution: depression, pain impact and somatic focus. The authors reported this structure as confirmed by CFA on the other half of the sample, although the χ^2 test was reported as significant. The section of the article reporting the SEM analysis showed multiple errors, showing a very limited understanding of this methodology: reporting standardized parameters between latents as 'R^2', stating that CFA results "revealed that Depression determined 61% of the variance in Illness Conviction" (when CFA only specifies bidirectional relationships between latents), not reporting GOF for the final model selected. It is not clear why a model replacing bidirectional relationships with "a relationship other than correlational" led to an invalid model, since the models are practically equivalent (if no additional parameters are fixed), but this would not be a test of directional (causal) relationships, given that the data were cross-sectional, as the authors stated several times.

Covic et al. (2003) used path analysis on a sample of rheumatoid arthritis sufferers with a longitudinal design (three time points, 12-month interval). They assessed the role of physical disability, mediated by helplessness and passive coping in predicting depression and pain using both cross-sectional and longitudinal data (of all three stages, in all combinations). Although most models had an adequate fit to the data, the limitations of this application were similar to those of the studies mentioned above: the use of total scores, no alternative or equivalent models presented (plausible especially in the cross-sectional analysis). Also, longitudinal models did not control for values of the same variable at a prior stage (the data showed stability,

[5]Another minor limitation was not considering equivalent models in the context of cross-sectional data. Although results of experimental studies (Price, 2000) supported the direction of causality proposed by the authors, in this particular design it was equally plausible for example that reports of pain unpleasantness were influenced by current experience of discrete emotions and current assessments of pain implications.

except for an improvement in depression scores), and therefore can be considered similar to a cross-sectional analysis, except the lack of common sources of variation due to simultaneous measurement, which led to lower parameter estimates between variables in different stages (some becoming non-significant). Thus, the study was actually a limited test of the mediation role of helplessness and passive coping.

Goubert et al. (2004a) applied SEM to investigating the relationships between pain severity, neuroticism, pain catastrophizing and pain-related fear in vigilance to pain in a sample of low back pain sufferers. They tested a serial model from neuroticism via pain catastrophizing, fear of movement, vigilance to pain to pain severity. Most latents had subscale scores as indicators, except pain severity which was measured by 3 selected MPQ items. The parameter estimates of the paths between pain catastrophizing, fear of movement and vigilance to pain were .85 and .95, indicating latent multicollinearity and suggesting that an alternative model in which these three latents would be merged into a single latent should have been considered. These limitations of Goubert et al.'s (2004a), together with the cross-sectional design acknowledged by the authors, suggest that the associations between these measures might also be explained by the measurement overlap between the instruments selected: since the three concepts tap into pain-related distress, it is unsurprising that they are in a sense midway between a trait-measure of distress and indicators of pain perception.

Cook et al. (2006) tested Vlaeyen et al.'s (1995) fear-avoidance model via a SE model of the relationship between catastrophizing, pain-related fear, depression, disability and pain severity. Catastrophizing was specified to influence fear of injury, which in turn determined both depression and disability (also determined directly by catastrophizing), both impacting on perception of pain severity; the model showed a good fit: $\chi^2(29) = 42$, $p = .06$, GFI= .98, AGFI= .97, CFI= .99, RMSEA= $.03(.00 - .05)$. Multigroup analyses indicated age differences between middle-aged and older patients in the strength of relationships between the constructs. As in previous studies, the use of total subscale scores as manifest variables limited theory testing on the measurement level, especially given issues of content overlap between scales. The authors did consider alternative models (partial versus total mediation role of fear of injury), but not equivalent models (relevant especially given the cross-sectional design), although they did specify the cyclical nature of the process and the selection of pain severity as outcome as one of the possible solutions. The authors encouraged the replication of this study on longitudinal data.

Gheldof et al. (2006) studied the role of work-related, psychosocial and psychological factors in relation to functional and social disability based on cross-sectional data in a sample of employees reporting back pain. They first used regression analyses to explore the relationships between the study variables, performed mediation analyses of the role of pain-related fear in the relation between pain severity and negative affectivity as predictors and functional and social disability as outcomes. Based on these analyses, they further explored the mediation relationships together by comparing two alternative path models. One methodological limitation of the study (in addition to the lack of measurement level and a liberal interpretation of fit indices) is that the models were not stipulated a priori, but built based on the previous analyses and in sequence, the second model benefiting from the results of the first model. Nevertheless, splitting the sample ensured that the final model was validated on a new sample. The final model specified negative affect influencing pain-related fear and pain severity which in turn determined both functional and social disability; unidirectional paths were also specified from pain-related fear to pain severity, and from functional to social disability ($\chi^2(2) = 6.2$, $p < .05$, GFI= .99, AGFI= .96, CFI= .99, RMSEA= .07). Despite its limitations, this study highlights the importance of testing multiple relationships simultaneously in comparison to 3-variable mediation analyses.

The studies presented here only partially adhere to the methodological requirements summarised in the previous sections. While it can be argued that a method should be adapted to the practical goals and constraints of a specific application, I consider that these examples illustrate the fact that taking full advantage of the methodological possibilities of SEM remains a future direction in chronic pain research.

Appendix C

Data preparation and missing data analysis

C.1 Data preparation

The data from online questionnaires were first combined with paper questionnaire data from PAS and NHS, with particular attention paid to identical coding of variables. Five participants in the online survey could not be matched to their first stage responses due to lack of correspondence between their date of birth, height and colour of eyes data (the responses in stage 2 and 3 were matched in these 5 cases). From the 269 participants, only 208 cases had responses for all three stages. Selection based on inclusion and exclusion criteria was performed at the data preparation stage for participants from chronic pain support organisations. Four participants were excluded for analyses on the basis of reporting cancer as comorbid condition. The age requirement (above 18 years) was met by all participants. Therefore 265 cases remained available for the first stage analysis, 224 for the second stage, 209 for the third stage and 204 cases for a longitudinal analysis (Figure C.1).

Questions about comorbidity, current treatment, and medication were apparently difficult for some respondents, as the categories provided were not sufficiently comprehensive and clear and contained technical terms. These questions were checked for consistency based on the responses to subsequent open-ended questions (such as "other medical problems") and recoded where necessary. The difficulty the participants had in choosing categories indicates that such questions could be improved in further studies to tackle both general linguistic issues of categorisation and specific issues of adapting medical language to lay use.

The responses to the open-ended questions of the Self-Administered Comorbidity Questionnaire ("other medical problems") included diagnoses related to chronic

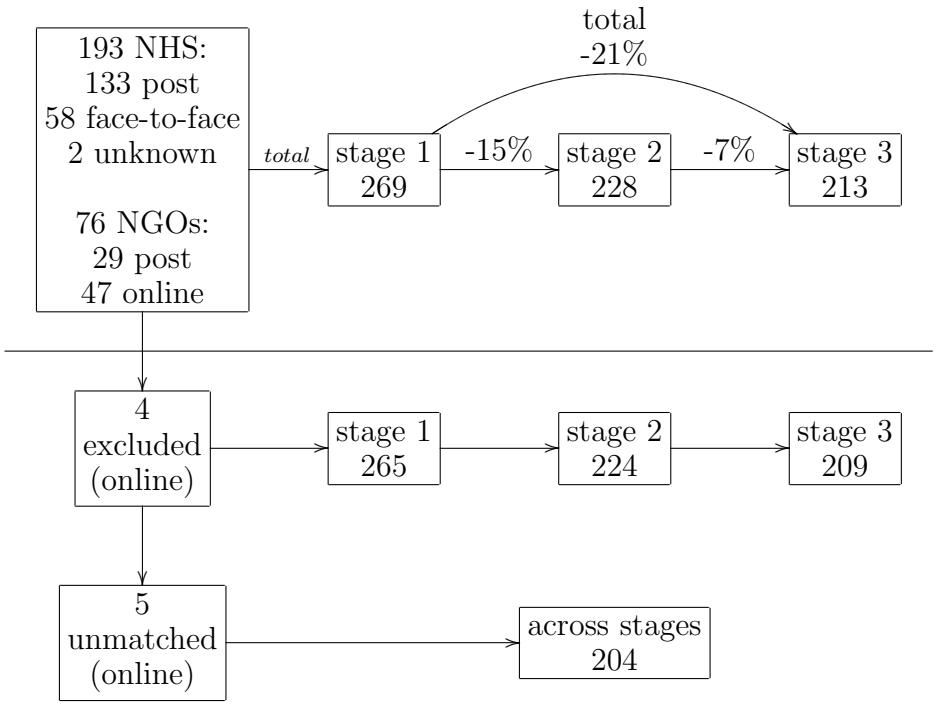


Figure C.1: Number of cases for analysis considering attrition and data preparation

pain (CRPS, fibromyalgia, sciatica), problems related to other body locations (bladder, bowel), less severe conditions (hypothyroidism, eczema), less frequent conditions (Hirschsprung's disease, Crohn's disease), or lay diagnoses (cholesterol, poor circulation, palpitations). The total score was computed as the sum of all conditions reported and used as a control variable to account for decreases in health status due not to the pain itself, but to the number and severity of accompanying conditions. To increase the accuracy of the total scores, these types of responses were treated differently. As not all participants reported their chronic pain as separate condition, all chronic pain diagnoses reported (e.g. CRPS) were deleted. Less severe and less common conditions and reports of problems in other locations were considered valid and included in the total score. Lay terms that focused on limited symptoms with unclear relationship to a medical diagnosis were deleted. After these changes, the number of patients who reported one or more other conditions decreased from 89% to 34%.

Some of the responses to open-ended categories of the List of Threatening Events included descriptions of 'other' events that could be included in one of the already provided categories. These responses were recoded for consistency.

Pain location was recoded based on the open-ended questions in the NHS questionnaires according to the categories provided for the rest of the participants. Complaints of jaw pain, dental pain and other facial pain were considered head pain, throat pain - neck pain, upper arm - shoulder, lower arm - wrist/hand, breast - chest, pelvic, groin and testicle - abdominal, midback - lower back, lower leg - ankle/foot, leg pain - both hip and ankle/foot, arm - both shoulder and wrist/hand, back pain - both upper and lower. Broader categories were computed based on the existing locations: head and neck, limb, back and visceral pain. Generalised pain was considered a separate category to include all comments about pain allover body both muscular and joint-related[1]. Also, responses for the participants that were given the categories format were recoded for consistency where necessary, based on the rules above and their responses to the open-ended questions ('other pain' locations). The number of individual pain locations (up to 12) and the number of broader categories of pain reported (up to 4) were computed as indicators of the spread of pain by adding the responses to the above questions (reports of generalized pain were given the maximum score).

Questionnaire scores were computed from the raw data according to their authors' instructions. Further data preparation for questionnaires and a detailed analysis of their psychometric properties is presented in Chapter 8.

Several other scores were computed based on existing variables: age at survey (difference between the date when the study started and birth date), number of weeks with pain (sum of years, months and weeks with pain considered as 52, 4 and 1 week, respectively), age at pain onset (difference between age at survey and years with pain - weeks of pain divided by 52), time to respond to questionnaire (difference between date of posting/handing and date of posting the response letter), and time between stages (difference between date of posting the response letter in stage 1 or 2 and stage 2 or 3 respectively).

Most of the statistical analyses necessary (correlation, regression, SEM) require absence of univariate outliers and assume (or are enhanced by) normal distribution for continuous variables and good proportion of cases in all categories for dichotomous and categorical variables (Tabachnick and Fidell, 2001). Therefore these criteria were checked in the data preparation stage for all variables other than questionnaire items: gender, age at survey, education, marital status, vocational status,

[1]Left-right distinctions are lost in such a categorisation, but arguably they are less important for this type of study and more relevant for treatment.

annual income, ethnicity, nationality, number of weeks with pain, age at pain onset, pain location, previous and current type of pain treatment, time to respond to questionnaire, time between stages. As most data analyses are based on ungrouped data, outliers were sought in the entire data set (Tabachnick and Fidell, 2001, p. 67).

From the categorical demographic variables, ethnicity, nationality and several previous (pain relief medication) and current types of pain treatment (pain relief medication, surgery, psychological therapies) had an uneven split (more than or closely approaching 90% in one category; Tabachnick and Fidell, 2001, p. 58, 67) and were excluded from further analyses. Education, vocational status and marital status had several categories with very few cases which were therefore merged into more general categories: none/secondary school versus college/university (education); unable to work because of pain versus working/training versus unable to work for other reasons (vocational status); married/living as married versus single/separated/divorced/widowed (marital status).

Several individual pain locations (e.g. elbow, chest) had an uneven split and therefore only the broader categories were used in these cases. From the two indicators of spread of pain, the number of individual pain locations had a non-normal distribution; thus, only the number of broader categories (from 1 to 4) was kept for further analyses.

For the continuous variables, the data was screened for normality of distribution based on the Kolmogorov-Smirnov and Shapiro-Wilk tests, inspection of the Q-Q plots, histograms and boxplots. The Kolmogorov-Smirnov and Shapiro-Wilk tests were significant for all variables except age at pain onset. However, Kolmogorov-Smirnov and Shapiro-Wilk are known to be too sensitive if dealing with big samples (Field, 2005, p. 93). Based on inspection of the graphs, age at pain onset and age at survey were considered as having relatively normal distributions.

Pain duration, time to respond to questionnaire and time between stages had extremely skewed distributions and numerous outliers. As this was not due to data entry or sampling errors, several options were available: variable transformation, deleting or replacing the outlier scores with less extreme values, variable dichotomisation via median split.

Transforming variables was not considered, as it would make the interpretation very difficult and applying non-parametric tests would not answer the research questions.

In addition, normality of the data is only a condition that enhances prediction in linear statistics, not an assumption. Only residuals need to be characterised by normality, linearity and homoscedasticity, according to Tabachnick and Fidell (2001), and regression analysis is robust to moderate violations of the normality assumption especially in large samples (Berry and Feldman, 1985, p. 11). Also, in SEM analysis the use of the ML robust method gives reliable estimates even with moderate violation of non-normality (Bentler, 2004).

Reducing the influence of outliers, although does not significantly improve very skewed distributions, prevents loss of information in comparison to the median split method. This was considered useful for pain duration; therefore all scores bigger than two standard deviations from the mean (12) were replaced by this value (mean+2SD; Field, 2005, p. 79). It can be argued that in terms of pain duration, the behaviour of chronic pain sufferers which have a history of more than 28 years of pain is similar to those whose pain has started about 28 years ago. The analyses that include pain duration can be run comparatively with replaced outliers and with the dichotomised variable via median split.

Time to respond to questionnaire and time between stages are relevant only to the extent that the late respondents were different from those that responded in a short time. Therefore these variables were dichotomised using the median for the purpose of these analyses.

Examining missing data revealed few missing values (below 2%) or none for most of the above variables, except annual income (5%), pain duration (and consequently age at pain onset, 5.6%), and the missing data due to attrition. For the variables with less than 2% missing values, any procedure would give similar results Tabachnick and Fidell (2001, p. 59) and therefore listwise or pairwise deletion were considered suitable (selected depending on analysis). For annual income, missing values were replaced with the mean value (of the three ordered categories). For pain duration, missing values were replaced using the expectation maximisation method (EM), a procedure usually used for randomly missing data, which was the case in this dataset (see Peng et al., 2006, for discussion of random patterns of missing data). It is a two-step iterative procedure that uses maximum likelihood estimation to improve the initial expected values of the missing data based on the observed values and the assumed distribution. It is a better method of computing missing data in comparison to others (e.g. using prior knowledge, mean substitution

and regression) for several reasons such as realistic variance estimates and avoiding overfitting (Tabachnick and Fidell, 2001, p. 63). Age at pain onset was then recomputed based on age and pain duration.

Listwise deletion was used for missing data due to attrition both for cross-sectional analyses of the second and third stage and for the longitudinal analysis. The use of this method in comparison to data imputation (for example by EM) is controversial in the literature. Deletion is considered adequate only in cases where data is missing completely at random (the mechanism behind non-response is independent of the variables studied; Peng et al., 2006), otherwise results are biased to a certain extent and generalizability is limited; if missing values are spread across cases, it also leads to substantial sample size decrease. On the other hand, methods such as EM assume normal distributions (Newman, 2009) and are rather suited to continuous variables (while many variables in the present data set are categorical or ordinal). According to Bentler (2004), using an imputed data matrix in SEM does not adequately account for the fact that the data is not complete, as does the ML method available in EQS. Its implementation in both SPSS and EQS does not handle well a high cases-to-variables ratio, which would be the case in the longitudinal data set, while computing missing values separately for sets of variables potentially introduces other sources of bias. If missing data characterises only selected cases (therefore not impacting dramatically on sample size), and the bias is minor (leading to only minor distortions of parameter estimates, Newman, 2009), listwise deletion can be considered a valid approach.

To assess the suitability of listwise deletion, mean differences between participants that have responded to all three stages and those that only answered to the first stages were explored. Several variables were considered: demographics (gender, age, education, vocational and marital status), pain location, age at pain onset, pain duration, healthcare utilisation, source of participants (NHS or other) and questionnaire scores (AAQ, BES, REQ, LTE, SACQ, MPQ, CPAQ, SIP, BIPQ)[2]. Participants recruited from the NHS by face-to-face contact or by post were more likely to participate to all stages than participants from other sources (stage 2: $\chi^2(1) = 7.3$, $p = .01$; stage 3: $\chi^2(1) = 7.1$, $p = .01$). This is more likely to be due to the use of the online questionnaire and email remainders and not to some characteristics intrinsic to the respondents. Participants to all stages tended to report that their life is less affected by their chronic pain (stage 2: means 7.9 vs 7.5,

[2]Only differences that were consistent between the two last stages are reported. If differences appear only in one of the stages, they were considered more likely to be due to random variations.

$t(261) = 4.76$, $p = .03$; stage 3: means 8.2 vs 7.3, $t(261) = 6.03$, $p = .015$) and that they are also less affected emotionally (stage 2: means 7.8 vs 7.1, $t(260) = 6.74$, $p = .01$; stage 3: means 7.8 vs 7.0, $t(260) = 8.15$, $p = .005$).

These differences could indicate a tendency for those participants that felt overwhelmed by their condition to give up responding to the questionnaire. It is important to note that these participants did not report higher pain intensity, more pain related disability, more frequent emotions, more life events (positive or negative) or other pain related beliefs. Therefore these differences are not indicating consistently higher illness severity and pain-related distress in general for non-respondents compared to respondents and thus for the purposes of this analysis the missing data mechanism was considered independent of the variables of study[3]. In SEM models, listwise deletion and ML imputation were used comparatively[4].

As participants were recruited from two sources (NHS and support organisations), it is important to check if there are any differences between groups which would indicate that the sample analysed is not homogeneous. Several variables were considered: demographics (gender, age, education, vocational and marital status), pain location, age at pain onset, pain duration, healthcare utilisation, and questionnaire scores (AAQ, BES, REQ, LTE, SACQ, MPQ, CPAQ, SIP, BIPQ). At a conservative $\alpha = .01$ (selected due to the high number of comparisons), the two groups differed only in the level of education ($\chi^2(1) = 6.4$, $p = .01$), frequency of anger-related emotions reported ($t(219) = 2.95$, $p = .003$) and presence of pain reported in most body areas.

The difference in the level of education was unsurprising, as searching for and joining support groups, especially on the internet, require a higher level of literacy and abilities to proactively search for information, which are usually the result of education. The differences in reports on the anger subscale of BES indicated that participants of support groups are less angry ($t(218) = 2.79$, $p = .006$) and aggressive ($t(45) = 3.95$, $p < .001$), which was also unsurprising in people that have been participating in group activities, but not less frustrated or irritated, which are less socially destructive emotions. Given that no other differences were identified

[3]Nevertheless, it is acknowledged that the sample might not be representative for all the chronic pain population, given the low initial response rate, which might also be due to the higher emotional-motivational impact of the chronic pain condition.

[4]Also, EM computation was used for a selected sample of variables and estimates of associations between variables were compared to those based on listwise deletion, resulting in very similar values; therefore it was considered that the two methods would likely lead to similar results across analyses.

in emotional reports, it was concluded that the two groups came from the same population.

The differences in reports of pain location have an obvious explanation: pain location was measured via open-ended questions in the NHS sample, while the questionnaires given to support organisations had a more detailed format in which a list of pain locations was given. The presence of the list made it easier for respondents to report the presence of pain, which led to more pain reporting. Since there are no differences in other indicators of pain severity (such as pain related disability or pain intensity), it can be concluded that this differences are solely due to the questionnaire format. Therefore data related to pain location was analysed separately for the two groups, while the whole dataset was used for all other analyses.

Another potential source of sample heterogeneity is represented by differences due to time of response (the time the participants took to respond to the stage 1 questionnaire, the time interval between first two stages and between the last two). If participants that took longer to respond are consistently and significantly different from the quick respondents, change statistics might be influenced by these variables rather than represent intrinsic characteristics of the individuals. Demographics (gender, age, education, vocational and marital status), pain location, age at pain onset, pain duration, healthcare utilisation, source of participants (NHS or other) and questionnaire scores (AAQ, BES, REQ, LTE, SACQ, MPQ, CPAQ, SIP, BIPQ) were considered as potential sources of differences, and a conservative α level of .01 was selected due to the high number of comparisons. There were no differences between quick and slow respondents except the fact that participants from the NHS had longer response intervals between the last two stages (probably due to the use of the postal services which were on strike several times during that period). Therefore it was concluded that the sample was homogeneous from this point of view.

Several variables had a special status due to a small subsample of participants from the chronic pain support organisations responding to additional questions: the presence of a medical diagnosis, the degree to which they felt they understood the diagnosis, whether they received benefits, whether they were involved in litigation and to what extent they felt they received social support from family, friends, colleagues, etc. As none of the participants reported being involved in litigation, this variable was excluded from further analyses. Within this subsample, missing values due to non-response had negligible levels. Ordinal variables had non-normal distribution and were therefore dichotomised. As medians for these variables ranged

Variable name	Problem	Decision
Gender	-	-
Age at survey	-	-
Education	Underrepresented categories	Recoded into broader categories
Marital status	Underrepresented categories	Recoded into broader categories
Vocational status	Underrepresented categories	Recoded into broader categories
Annual income	Missing values 5%	Replaced with mean
Ethnicity	Uneven split	Excluded
Nationality	Uneven split	Excluded
Time to respond, time between stages	Skewed distribution	Dichotomised (median split)
Pain duration	Outliers present Missing values 5.6%	Replaced with mean+2SD Median split (comparative analyses) Computed via expectation maximisation
Age at pain onset	-	-
Pain location	Uneven split for some variables Group differences depending on the type of questions used	Recoded into new variables with broader categories Separate analysis for subsamples from NHS and from support organisations
Previous treatment	Uneven split: pain relief medication	Pain relief medication excluded
Current treatment	Uneven split: pain relief med, surgery, psychological therapies	Problem variables excluded
Diagnosis	-	-
Clear diagnosis	-	-
Benefits	-	-
Litigation	Uneven split	Excluded
Social support	Skewed distributions	Dichotomized (cut-off - middle value of scale)
Questionnaire scores	See Section C.2 and Chapter 8	-

Table C.1: Summary of data preparation decisions

from 4 to 9 on a scale from 0 to 10, it was considered more theoretically meaningful to use the middle point of the scale as cut-off point. All resulting variables had both categories well represented. The data preparation decisions are summarised in Table C.1.

C.2 Missing data analysis

As a preliminary step, missing data analysis was performed for all questionnaires at each stage. Except BES and SF-MPQ, all questionnaires had less than 10% missing values. Inspection of missing data patterns for each of the stages revealed several cases with a higher number of missing values (excluding BES and SF-MPQ data:

6 cases with more than 10%, 2 cases with more than 25% for stage 1; 6 cases with more than 10%, 3 cases with more than 25% for stage 2; 5 cases with more than 10%, 3 cases with more than 25% for stage 3). These respondents skipped one or more questionnaires in part or in full, however their response to other instruments did not seem to be affected; as this may be due to turning pages (or double clicking on the 'next' button in the online version) and it is not necessarily an indicator of low overall reliability of responses, the cases were kept in the dataset. The rest of missing values were scattered across cases. The decisions or replacing missing values for each questionnaire are presented in Tables C.2 and C.3

Expectation maximisation (EM) was performed for ordinal questionnaire scores in EQS (the method is not available for categorical data). The computation based on EM was based on all ordinal questionnaire scores for each stage (except MPQ: AAQ, BES, REQ, CPAQ and BIPQ) in order to take into consideration as much variation as possible from the whole survey. MPQ was excluded due to the special missing data pattern that needed separate consideration (detailed next). EM could not be computed for all stages together due to algorithm convergence problems both in SPSS and EQS. As the missing values were generated in a continuous format, the rounding (RND) function was used to transform the values back into ordinal format. Additional checks were made to ensure that the estimated values were within the correct response range for each questionnaire.

The missing data analysis of the SF-MPQ identified two main patterns (the percentages of missing data for each item are presented in Table C.4). Both patterns were characterised by the omission of responses to some descriptors, while differing in the type of responses selected. One category of respondents never selected 'none' from the response options, and only reported 'mild', 'moderate' and 'severe' levels. The second category selected from all options, including 'none'. These patterns were spread across cases (first pattern is present in 28.1%, 15.2% and 10.0% in the three stages, while pattern 2 in 18.9%, 27.4%, and 33.3%, respectively) and therefore computing missing values was essential. Due to this potentially meaningful difference, the choice of the imputation method needed to carefully consider the possible mechanisms of non-response[5].

[5]Other minor patterns were skipping the questionnaire entirely (very few respondents per stage: 2, 1 and 1, respectively) and responding only to the VAS and/or PPI and skipping all the descriptors (3 cases in stage 1). Their impact on the dataset was less dramatic, and any data imputation method would lead to similar results.

Scale	Missing data patterns	Decision	Rationale
AAQ	stage 1: 1 case - 75-100%, 1 case - 50-75%, 18 cases <25%; stage 2: 8 cases - 75-100%, 8 cases <25%; stage 3: 7 cases - 75-100%, 11 cases <25%	Use EM (see details in text)	Missing values are most probably due skipping random questions while following the text. As questions are related, the most probable answer can be computed from the remaining data. 100% missing values are probably due to skipping the full questionnaire when turning the pages - the most probable answers are estimated based on responses to the other questionnaires.
BES	stage 1: all data missing in the online sample, in the rest of the sample: 1 case - 75-100%, 1 case - 50-75%, 28 cases <25%; stage 2: except the online sample, 22 cases <25%; stage 3: except the online sample, 16 cases <25%	Use only PAS & NHS for analysis. Use EM (see details in text) for PAS & NHS cases.	For online respondents, the data is missing due to due to a programming error in the online survey tool. For PAS & NHS cases - as for AAQ
REQ	stage 1: 18 cases <25%; stage 2: 1 case - 25-50%, 14 cases <25%; stage 3: 2 cases - 75-100%, 29 cases <25%	Use EM (see details in text)	As for AAQ
LEN	stage 1: 1 case - 75-100%, 4 cases <25%; stage 2: 3 cases <25%; stage 3: 1 case - 75-100%, 5 cases <25%	Replace with 0	If participants did not respond, the most likely situation is that they did not experience that event, and so the item was not relevant for them and therefore not worth responding
SACQ	2 cases - 75-100%, 30 cases <25%	Replace with 0	If participants did not respond, the most likely situation is that they did not have that condition and so the item was not relevant for them and therefore not worth responding

Table C.2: Missing data analysis - summary

Scale	Missing data patterns	Decision	Rationale
Health-care Utilisa-tion	stage 1: 2 cases - 25-50%, 7 cases <25%;stage 2: 1 case - 25-50%, 4 cases <25%; stage 3: 1 case - 75-100%, 2 cases - 25-50%, 29 cases <25%	Replace with 0	If participants did not respond, the most likely situation is that they did not use the service, or medicine, or self-help method, and so the item was not relevant for them and therefore not worth responding
MPQ	stage 1: 19 cases - 75-100%, 33 cases - 50-75%, 21 cases - 25-50%, 70 cases <25%; item percentages and patterns described in subsection 8.8; stage 2: similar to stage 1; stage 3: similar to stage 1	Decisions are dis-cussed in text	Rationale is given in text
CPAQ	stage 1: 3 cases - 75-100%, 1 case - 25-50%, 36 cases <25%; stage 2: 4 cases - 75-100%, 2 cases - 50-75%, 29 cases <25%; stage 3: 1 case - 75-100%, 1 case - 50-75%, 2 cases - 25-50%, 27 cases <25%	Use EM (see details in text)	As for AAQ
SIP	stage 1: no missing values; stage 2: 3 cases - 100%, Case 95 - no data CPAQ & BIPQ, but pain intensity high and SIP has data in other stages; Case 179 - no data CPAQ & BIPQ, but pain intensity medium, SIP has data in other stages; Case 200 - no data MPQ, CPAQ & BIPQ, but pain intensity high and CPAQ, BIPQ & SIP have data in other stages; 1 case <25% (168); stage 3: 1 case - 100% (113 - mv at CPAQ & BIPQ, low values MPQ - no pain in stage 2); 2 cases <25%	Replace with 0 sin-gle missing values and case 113, leave the others missing	The instructions were to leave the space blank if the sentence does not describe the respondent's experience. If the pain intensity is low and other answers indicate lack of pain (or if only a few answers are missing), all spaces left blank are more likely to mean no disability, missing values in the online study are more likely to mean the item is not relevant for the respondent. If more questionnaires have missing data, and in different stages SIP responses indicate higher levels of disability, the data is genuinely missing.
BIPQ	stage 1: 1 case - 75-100%, 1 case - 50-75%, 1 case - 25-50%, 11 cases <25% ;stage 2: 6 cases - 75-100%, 1 case - 50-75%, 13 cases <25%; stage 3: 1 case - 75-100%, 9 cases <25%	Use EM (see details in text)	As for AAQ

Table C.3: Missing data analysis - summary, continued

Item	Stage 1	Stage 2	Stage 3
throbbing	15.5	9.7	8.5
shooting	21.9	12.8	9.5
stabbing	21.9	16.8	12.3
sharp	23.4	11.1	12.3
cramping	23.4	12.8	11.4
gnawing	23.0	12.8	11.4
hot-burning	20.8	13.3	10.9
aching	10.6	6.2	3.3
heavy	22.6	11.1	9.0
tender	17.4	11.1	10.4
splitting	31.7	16.8	16.6
tiring-exhausting	10.2	5.3	6.2
sickening	23.0	12.4	10.4
fearful	24.9	15.0	13.3
cruel-punishing	21.9	15.0	11.4
VAS	22.3	22.6	22.3
PPI	3.0	6.6	9.5

Table C.4: Mising values SF-MPQ (percentages)

Regarding the two pain patterns, several mechanisms were hypothesised and tested. One possible explanation considered was that all respondents skipped the missing items by mistake, but their answers can be reconstituted from their remaining answers, given the inter-item correlations. If this were the case, non-response would not be related to any other variable, as it would be a random phenomenon. This hypothesis was disconfirmed in an analysis at the level of the whole data set: non-response in most descriptors was significantly associated overall with higher scores at other descriptors in the same stage (the majority of mean differences belonged to this trend, a fair percentage of t-tests were significant at $\alpha = .05$), but not with VAS and PPI scores (non-response in VAS and PPI were unrelated to any of the SF-MPQ items).

Two explanations were considered for this association. First, respondents did not skip items by mistake, but by choice, and compensated the lack of response by giving higher ratings to the remaining items. This suggested that computing the missing values via EM would bias the overall scores by inflating the non-respondents' ratings. Second, respondents that skipped items mistakenly had genuinely higher scores (and this possibly contributed also to their lack of attention to the task). In this case, EM would be a suitable procedure. No test of the probability/adequacy of these mechanisms could be performed on the whole data set. Therefore, separate missing value analyses were performed for the two patterns. For the first pattern (no 'none' response selected), non-response was indeed consistently associated with

higher scores at the other items. Therefore the compensation mechanism was considered more likely for this pattern. As in this case 'none' and non-response were not differentiated, it was considered probable that missing values corresponded to 'none' even if they did not correlate to lower values at the same item in other stages, since the items did not show stability. For the second pattern (only some 'none' responses selected), no consistent trend of association was found, therefore skipping items mistakenly was considered a more probable mechanism in this case, as 'none' answers were differentiated from non-response, and implied computing missing values via expectation maximisation. Thus, missing values were computed by replacing them with 'none' for pattern one and using EM for the second pattern, the VAS and PPI scales and the rest of the minor patterns. Several other missing data mechanisms were considered but deemed less likely (such as unfamiliarity with specific words, questionnaire length, not experiencing the particular quality of pain, random response due to task difficulty). The full analysis is not reported here for reasons of brevity.

As missing data analysis is not usually reported in the literature for SF-MPQ, it was considered that the default computation is considering 'none' as equivalent of all missing data. Therefore this second method for computing missing data was also used for comparative purposes. The correlations between the scores obtained on descriptors based on the two methods had values between $r = .93 - 1.00$, while the correlations between pain quality indexes exceeded .98, and reliability and stability estimates were very similar, indicating that the two methods resulted in largely equivalent scores. The first missing data computation result was kept for further analyses.

From all other variables in the data set, missing data was consistently associated only with age. In this sample, older people are consistently more prone to non-response at descriptors and VAS in all stages (the majority of t-tests are significant at an α level of .05 in pattern one responses). This result contradicts the preliminary evidence for the suitability of SF-MPQ in older populations (Gagliese and Melzack, 1997) and adds to the concerns regarding MPQ expressed by Herr and Mobily (1993, as cited in Gagliese and Melzack, 1997): SF-MPQ might not be suitable for older adults.

Appendix D

Covariance matrices for SE models

As detailed in Appendix B, reporting covariance matrices is recommended in SEM analyses. The following tables present this data for the analyses in Chapter 7.

	AAQ1	AAQ2	AAQ3	CPAQ1	CPAQ2	CPAQ3
AAQ1	.872					
AAQ2	.683	.935				
AAQ3	.637	.658	.855			
CPAQ1	-.483	-.537	-.478	.888		
CPAQ2	-.489	-.590	-.533	.780	.970	
CPAQ3	-.523	-.561	-.574	.791	.854	.975

Table D.1: Covariance matrix for CPA-PF longitudinal path model

	I2	I3	I7	I8	I9	I1R	I4R	I5R	I6R
I2	1.652								
I3	0.634	2.874							
I7	0.802	1.746	2.770						
I8	0.543	0.772	1.054	2.366					
I9	0.945	0.825	1.257	1.266	3.878				
I1R	-0.017	0.416	0.403	0.025	0.320	1.708			
I4R	0.191	0.342	0.543	0.152	0.487	0.329	2.772		
I5R	0.633	0.783	1.013	0.416	0.833	0.466	1.070	2.781	
I6R	0.145	0.513	0.800	0.309	0.740	0.494	0.921	1.108	1.872

Table D.2: Covariance matrix for AAQ CFA model, stage 1

	PI1	AAQ1	AAQ2	AAQ5	AAQ7	CPAQ1	CPAQ3	CPAQ4	CPAQ18	PS	CMB	NLE1	PLE1
PI1	1109.253												
AAQ1	-10.114	1.708											
AAQ2	2.635	0.017	1.652										
AAQ5	-6.125	0.466	-0.633	2.781									
AAQ7	12.169	-0.403	0.802	-1.013	2.77								
CPAQ1	-14.817	0.428	-0.13	0.545	-1.032	2.627							
CPAQ3	-12.307	0.491	-0.321	0.593	-0.549	0.79	2.325						
CPAQ4	14.815	-0.398	0.203	-0.365	0.631	-0.729	-0.331	2.657					
CPAQ18	8.793	-0.291	0.593	-0.58	1.019	-0.86	-0.755	0.997	3.214				
PS	13.229	-0.161	-0.41	0.496	0.018	0.199	0.614	0.103	-0.231	11.415			
CMB	22.204	-0.446	0.023	0.167	0.608	-1.029	-0.243	0.715	0.794	3.985	15.647		
PD	1088.266	39.74	-43.746	74.107	-63.247	7.652	71.961	-9.217	-68.388	280.166	331.367 1	55107.698	
NLE1	2.022	-0.111	0.093	-0.24	0.286	-0.142	0.261	0.03	0.024	0.439	0.859	21.622	1.782
PLE1	-1.664	0.034	0.066	-0.053	-0.178	0.235	0.055	-0.095	-0.086	-0.068	-0.269	-36.604	0.283

Table D.3: Covariance matrix for CPA-PF cross-sectional SEM, stage 1

	BES1	BES4	BES5	BES7	BES8	BES12	BES16	BES18	BES19	BES20	BIPQ1	BIPQ2	BIPQ5	BIPQ6	BIPQ8
BES1	2.369														
BES4	1.139	2.534													
BES5	-0.895	-1.014	1.790												
BES7	1.463	1.438	-1.288	3.366											
BES8	0.860	1.006	-0.912	1.526	3.320										
BES12	1.166	1.390	-1.056	2.259	1.384	2.899									
BES16	2.068	1.019	-0.960	1.686	1.095	1.383	3.489								
BES18	1.179	1.202	-0.823	1.713	1.783	1.586	1.463	3.096							
BES19	1.002	1.452	-0.795	1.490	0.981	1.192	0.984	1.184	2.153						
BES20	-0.874	-0.914	1.433	-1.372	-0.871	-1.163	-0.806	-0.795	-0.631	1.793					
BIPQ1	0.725	0.555	-0.517	1.478	0.214	0.796	0.622	0.442	0.515	-0.576	4.214				
BIPQ2	0.248	0.317	-0.084	0.438	0.090	0.404	0.316	0.509	0.148	-0.112	1.405	2.268			
BIPQ5	0.352	0.449	-0.026	0.641	0.039	0.521	0.311	0.258	0.480	0.024	2.326	0.757	3.899		
BIPQ6	1.235	1.213	-0.502	1.855	0.701	1.257	1.235	1.197	1.179	-0.585	2.885	1.191	2.131	5.903	
BIPQ8	2.152	1.816	-1.445	2.802	1.728	2.052	2.069	1.695	1.818	-1.384	2.523	0.991	0.941	3.676	6.132
CPAQ1	-0.721	-0.975	0.894	-1.344	-1.040	-1.025	-0.815	-0.540	-0.900	0.778	-1.207	-0.279	-0.620	-1.202	-1.564
CPAQ3	-0.432	-0.646	0.371	-0.780	-0.267	-0.535	-0.670	-0.195	-0.512	0.512	-0.814	-0.415	-0.646	-1.452	-0.891
CPAQ4	0.645	0.681	-0.475	0.881	0.541	0.731	0.646	0.516	0.698	-0.442	1.250	0.401	0.917	1.410	1.686
CPAQ18	0.837	1.126	-0.623	1.246	0.965	1.082	1.116	0.995	1.148	-0.696	1.162	0.583	0.709	2.318	2.242
MPQ1	0.304	0.426	-0.018	0.496	0.172	0.413	0.326	0.467	0.404	-0.044	0.502	0.149	0.600	0.806	0.502
MPQ4	0.255	0.216	-0.166	0.543	0.271	0.204	0.507	0.367	0.421	-0.037	0.813	0.236	0.619	0.815	0.709
MPQ12	0.170	0.440	-0.129	0.324	0.011	0.056	0.235	0.137	0.317	-0.023	0.944	0.253	0.554	0.685	0.677
MPQ13	0.355	0.366	-0.104	0.551	0.241	0.325	0.498	0.236	0.399	-0.035	0.725	0.093	0.528	0.738	0.859
SIP1	0.215	0.224	-0.262	0.271	0.194	0.198	0.239	0.152	0.150	-0.200	0.554	0.155	0.289	0.453	0.508
SIP6	0.099	0.120	-0.088	0.137	0.108	0.107	0.118	0.182	0.070	-0.090	0.299	0.130	0.161	0.173	0.224
SIP9	0.065	0.060	-0.082	0.177	0.073	0.059	0.142	0.095	0.071	-0.055	0.463	0.148	0.306	0.242	0.274
SIP20	0.034	0.037	-0.047	0.097	0.065	0.098	0.014	0.047	0.071	-0.029	0.300	0.169	0.178	0.246	0.245

Table D.4: Covariance matrix - emotions, IPs, CPA, pain intensity and disability

	CPAQ1	CPAQ3	CPAQ4	CPAQ18	MPQ1	MPQ4	MPQ12	MPQ13	SIP1	SIP6	SIP9	SIP20
CPAQ1	2.707											
CPAQ3	0.833	2.344										
CPAQ4	-0.680	-0.232	2.657									
CPAQ18	-0.915	-0.792	1.078	3.421								
MPQ1	-0.246	-0.359	0.445	0.462	1.243							
MPQ4	-0.493	-0.448	0.224	0.405	0.255	1.652						
MPQ12	-0.408	-0.232	0.334	0.438	0.193	0.440	1.096					
MPQ13	-0.497	-0.177	0.415	0.546	0.346	0.670	0.451	1.213				
SIP1	-0.321	-0.133	0.211	0.345	0.095	0.181	0.166	0.130	0.249			
SIP6	-0.128	-0.073	0.011	0.165	0.083	0.098	0.082	0.078	0.067	0.239		
SIP9	-0.178	-0.122	0.113	0.139	0.084	0.108	0.122	0.120	0.086	0.033	0.237	
SIP20	-0.100	-0.051	0.120	0.163	0.046	0.063	0.042	0.022	0.055	0.021	0.055	0.169

Table D.5: Covariance matrix - emotions, IPs, CPA, pain intensity and disability (2)

	I1	I 2	I 3	I 4	I 5	I 6	I 7	I 8	I 9	I 10	I 11	I 12	I 13	I 14	I 15	I 16	I 17
I 1	2.408																
I 2	1.374	3.117															
I 3	1.114	1.520	2.841														
I 4	1.126	1.640	1.185	2.513													
I 5	-0.900	-1.168	-0.919	-0.995	1.817												
I 6	1.011	1.282	0.677	0.821	-0.657	1.915											
I 7	1.516	2.300	1.478	1.427	-1.270	1.377	3.418										
I 8	0.887	1.326	1.800	1.004	-0.871	0.829	1.572	3.350									
I 9	0.963	1.415	1.129	1.477	-0.685	0.921	1.275	1.017	2.805								
I 10	-0.729	-1.012	-0.834	-0.963	1.478	-0.611	-1.011	-0.693	-0.517	1.845							
I 11	1.158	1.122	0.720	0.984	-0.692	1.128	1.412	0.731	0.815	-0.687	2.006						
I 12	1.214	1.984	1.376	1.376	-1.050	1.092	2.306	1.414	1.310	-0.951	1.309	2.930					
I 13	1.102	1.440	2.007	1.068	-0.896	0.805	1.694	1.759	1.255	-0.706	0.687	1.426	2.778				
I 14	1.005	1.415	0.861	1.420	-0.816	0.978	1.351	1.021	1.398	-0.827	0.961	1.478	1.013	2.274			
I 15	-0.755	-0.647	-0.674	-0.472	1.023	-0.367	-0.878	-0.491	-0.458	1.158	-0.508	-0.866	-0.606	-0.649	2.024		
I 16	2.097	1.383	1.174	1.008	-0.957	0.922	1.724	1.116	0.976	-0.583	1.322	1.416	1.338	1.089	-0.784	3.495	
I 17	1.237	1.459	1.362	1.087	-0.986	0.896	1.804	1.436	1.074	-0.703	0.750	1.660	1.377	0.904	-0.628	1.582	2.803
I 18	1.206	1.555	1.956	1.189	-0.827	0.798	1.736	1.786	1.128	-0.603	0.634	1.606	1.897	0.991	-0.720	1.479	1.736
I 19	1.000	1.412	0.941	1.442	-0.775	0.812	1.492	0.991	1.335	-0.755	0.852	1.191	1.123	1.289	-0.395	0.982	0.963
I 20	-0.880	-1.201	-0.890	-0.896	1.464	-0.758	-1.353	-0.830	-0.632	1.398	-0.712	-1.156	-0.879	-0.775	1.187	-0.805	-0.904
I 21	1.208	1.329	1.717	0.946	-0.773	0.718	1.508	1.456	0.904	-0.414	0.813	1.322	1.759	0.688	-0.583	1.581	1.442

	I 18	I 19	I 20	I 21
I 18	3.091			
I 19	1.177	2.139		
I 20	-0.799	-0.612	1.823	
I 21	1.728	0.735	-0.724	2.999

Table D.6: Covariance matrix for BES CFA model

	I1	I2	I3	I4	I5	I6	I7	I8	I9	I10	I11	I12	I13	I14	I15	I16	I17
I1	1.055																
I2	-0.104	1.005															
I3	0.591	-0.121	1.372														
I4	0.220	-0.071	0.210	0.898													
I5	-0.048	0.101	-0.032	0.110	0.542												
I6	0.174	-0.144	0.015	0.046	-0.079	1.195											
I7	-0.087	0.290	-0.139	0.134	0.297	-0.173	1.326										
I8	0.584	-0.079	0.458	0.275	-0.038	0.094	-0.054	0.932									
I9	0.181	-0.148	0.066	0.333	-0.076	0.213	-0.006	0.229	0.777								
I10	-0.064	0.293	-0.024	0.012	0.095	-0.032	0.181	-0.029	-0.071	0.518							
I11	0.179	-0.323	0.237	0.244	-0.184	0.179	-0.384	0.106	0.240	-0.175	0.841						
I12	0.235	-0.234	0.266	0.206	-0.126	0.340	-0.300	0.169	0.194	-0.136	0.492	0.913					
I13	-0.074	0.234	-0.100	0.005	0.086	-0.054	0.183	-0.009	-0.068	0.167	-0.173	-0.156	0.491				
I14	-0.147	0.306	-0.114	0.090	0.191	-0.241	0.549	-0.107	-0.082	0.099	-0.332	-0.233	0.167	1.086			
I15	-0.556	0.282	-0.384	-0.074	0.219	-0.171	0.565	-0.340	-0.144	0.160	-0.275	-0.222	0.160	0.463	1.450		
I16	0.068	-0.095	0.070	0.274	-0.034	0.084	0.012	0.010	0.306	-0.104	0.282	0.214	-0.111	0.031	0.128	0.863	
I17	-0.051	0.170	-0.030	-0.028	0.036	-0.083	0.073	-0.032	-0.075	0.199	-0.111	-0.092	0.213	0.099	0.096	-0.081	0.274
I18	-0.085	0.226	-0.048	0.024	0.149	-0.107	0.211	-0.018	-0.055	0.205	-0.161	-0.121	0.096	0.232	0.232	-0.108	0.126
I19	-0.118	0.122	-0.136	0.146	0.279	-0.070	0.609	-0.088	0.033	0.156	-0.247	-0.179	0.119	0.448	0.509	0.044	0.073
I20	0.507	-0.130	0.614	0.182	-0.188	0.119	-0.237	0.455	0.238	-0.116	0.309	0.343	-0.122	-0.141	-0.389	0.115	-0.145
I21	0.452	-0.090	0.330	0.117	-0.145	0.362	-0.355	0.305	0.237	-0.123	0.367	0.385	-0.116	-0.268	-0.437	0.160	-0.086

	I18	I19	I20	I21
I18	0.460			
I19	0.227	1.097		
I20	-0.164	-0.141	1.325	
I21	-0.143	-0.248	0.605	1.015

Table D.7: Covariance matrix for REQ CFA model

	I1	I2	I3	I4	I5	I6	I7	I8	I9	I10	I11	I12	I13	I14	I15
I1	1.273														
I2	0.448	1.558													
I3	0.467	0.883	1.516												
I4	0.329	0.763	0.934	1.631											
I5	0.367	0.466	0.513	0.624	1.345										
I6	0.212	0.261	0.184	0.225	0.255	1.333									
I7	0.257	0.363	0.343	0.358	0.256	0.282	1.464								
I8	0.322	0.299	0.200	0.336	0.211	0.309	0.126	1.046							
I9	0.360	0.459	0.450	0.532	0.331	0.220	0.485	0.468	1.472						
I10	0.266	0.368	0.419	0.389	0.271	0.266	0.384	0.402	0.398	1.362					
I11	0.361	0.452	0.500	0.498	0.319	0.251	0.298	0.246	0.456	0.353	0.982				
I12	0.285	0.407	0.443	0.447	0.348	0.268	0.318	0.428	0.397	0.365	0.271	1.165			
I13	0.385	0.455	0.466	0.635	0.444	0.303	0.299	0.407	0.479	0.402	0.494	0.487	1.230		
I14	0.425	0.456	0.502	0.583	0.370	0.115	0.257	0.363	0.471	0.384	0.500	0.398	0.597	1.065	
I15	0.525	0.548	0.554	0.650	0.405	0.402	0.252	0.478	0.568	0.455	0.591	0.493	0.714	0.804	1.494

Table D.8: Covariance matrix for SF-MPQ descriptors, CFA model, stage 1

	I1	I2	I3	I4	I5	I6	I7	I8	I9	I10	I11	I12	I13	I14	I15	I16
I1	2.627															
I2	1.860	2.427														
I3	0.790	0.850	2.325													
I4	-0.729	-0.715	-0.331	2.657												
I5	0.557	0.719	0.724	-0.296	3.234											
I6	1.529	1.858	0.797	-0.476	1.124	2.937										
I7	-0.322	-0.450	-0.576	0.633	-0.370	-0.745	2.477									
I8	0.955	1.073	0.623	-0.703	0.592	1.098	0.070	3.511								
I9	1.756	2.005	0.856	-0.854	1.124	2.344	-0.586	1.554	3.369							
I10	0.739	0.832	0.560	-0.590	1.236	0.828	-0.338	0.847	1.151	2.893						
I11	-0.703	-0.695	-0.578	0.534	0.134	-0.511	0.627	-0.565	-0.531	0.257	2.798					
I12	1.204	1.394	0.757	-0.404	0.489	1.614	-0.261	1.243	1.730	0.736	-0.507	2.541				
I13	-0.264	-0.462	-0.488	0.978	-0.648	-0.501	1.083	-0.436	-0.520	-0.639	0.543	-0.265	2.594			
I14	-0.748	-0.885	-0.670	1.297	-0.719	-1.056	1.393	-0.547	-1.037	-0.616	1.060	-0.455	1.849	2.773		
I15	1.269	1.316	0.745	-0.643	0.772	1.524	-0.439	1.119	1.705	0.668	-0.357	1.301	-0.574	-0.816	2.669	
I16	-0.064	-0.136	-0.353	0.779	-0.036	-0.195	0.502	-0.512	-0.141	0.120	1.346	-0.145	0.706	1.045	0.114	2.998
I17	-0.493	-0.700	-0.317	0.783	-0.602	-0.701	0.825	-0.795	-0.785	-0.495	0.745	-0.627	1.356	1.465	-0.734	0.721
I18	-0.860	-1.108	-0.755	0.997	-0.569	-1.135	1.171	-0.756	-1.140	-0.147	1.166	-0.813	1.313	1.668	-0.941	1.072
I19	1.075	1.232	0.867	-0.470	0.936	1.335	-0.618	0.852	1.639	0.880	-0.292	1.210	-0.659	-1.089	1.484	0.029
I20	-0.418	-0.524	-0.387	0.592	-0.507	-0.825	0.818	-0.565	-0.900	-0.489	0.499	-0.434	0.999	1.220	-0.817	0.329

	I17	I18	I19	I20
I17	2.640			
I18	1.425	3.214		
I19	-0.611	-0.883	2.647	
I20	0.825	0.978	-0.758	1.956

Table D.9: Covariance matrix for CPAQ CFA model, stage 1

	I 1	I 2	I 3	I 4	I 5	I 7	I 8	I 9	I 10	I 11	I 12	I 13	I 14	I 15	I 16	I 17	I 18	I 19	I 20
I 1	1.000																		
I 2	0.446	1.000																	
I 3	0.583	0.437	1.000																
I 4	0.520	0.364	0.243	1.000															
I 5	0.410	0.589	0.598	0.247	1.000														
I 7	0.508	0.710	0.575	0.283	0.733	1.000													
I 8	0.310	0.407	0.248	0.488	0.129	0.095	1.000												
I 9	0.509	0.601	0.511	0.487	0.552	0.679	0.202	1.000											
I 10	0.530	0.436	0.704	0.366	0.502	0.443	0.201	0.481	1.000										
I 11	0.412	0.592	0.627	0.527	0.669	0.619	0.299	0.478	0.485	1.000									
I 12	0.524	0.548	0.514	0.440	0.625	0.841	0.293	0.664	0.513	0.697	1.000								
I 13	0.342	0.637	0.356	0.201	0.420	0.495	0.141	0.456	0.300	0.503	0.557	1.000							
I 14	0.452	0.299	0.302	0.356	0.318	0.233	0.174	0.319	0.210	0.308	0.249	0.089	1.000						
I 15	0.400	0.569	0.448	0.390	0.601	0.682	0.233	0.790	0.388	0.552	0.754	0.532	0.252	1.000					
I 16	0.576	0.458	0.729	0.444	0.632	0.572	0.259	0.512	0.722	0.585	0.636	0.225	0.222	0.509	1.000				
I 17	0.424	0.657	0.385	0.259	0.565	0.460	0.127	0.559	0.373	0.548	0.474	0.645	0.400	0.566	0.200	1.000			
I 18	0.631	0.842	0.475	0.570	0.532	0.526	0.513	0.661	0.598	0.669	0.683	0.620	0.282	0.645	0.598	0.564	1.000		
I 19	0.638	0.238	0.524	0.457	0.304	0.455	0.293	0.513	0.524	0.350	0.403	0.145	0.282	0.326	0.623	0.087	0.461	1.000	
I 20	0.483	0.271	0.306	0.485	0.318	0.446	0.510	0.429	0.238	0.611	0.412	0.325	0.150	0.459	0.478	0.302	0.317	0.356	1.000
I 21	0.433	0.294	0.291	0.365	0.184	0.219	0.521	0.385	0.258	0.266	0.322	0.359	0.224	0.347	0.220	0.339	0.331	0.196	0.303
I 22	0.472	0.596	0.705	0.288	0.768	0.630	0.078	0.652	0.600	0.635	0.611	0.512	0.354	0.542	0.655	0.452	0.569	0.402	0.392
I 23	0.693	0.751	0.480	0.483	0.420	0.355	0.352	0.367	0.282	0.241	0.407	0.307	0.345	0.200	0.374	0.383	0.494	0.507	0.218
I 24	0.432	0.514	0.347	0.420	0.493	0.498	0.217	0.553	0.465	0.290	0.655	0.399	0.267	0.494	0.503	0.508	0.545	0.145	0.497

	I 21	I 22	I 23	I 24
I 21	1.000			
I 22	0.230	1.000		
I 23	0.452	0.372	1.000	
I 24	0.455	0.477	0.364	1.000

Table D.10: Polychoric correlation matrix for SIP, stage 1

References

Affleck, G., Tennen, H., Keefe, F. J., Lefebvre, J. C., Kashikar-Zuck, S., Wright, K., Starr, K., and Caldwell, D. S. (1999). Everyday life with osteoarthritis or rheumatoid arthritis: independent effects of disease and gender on daily pain, mood, and coping. *Pain*, 83(3):601–609.

Affleck, G., Tennen, H., Urrows, S., and Higgins, P. (1992). Neuroticism and the pain-mood relation in rheumatoid arthritis: insights from a prospective daily study. *J Consult Clin Psychol*, 60(1):119–126.

Aldrich, S., Eccleston, C., and Crombez, G. (2000). Worrying about chronic pain: vigilance to threat and misdirected problem solving. *Behav Res Ther*, 38(5):457–470.

Andersson, H. I., Ejlertsson, G., Leden, I., and Scherstn, B. (1999). Impact of chronic pain on health care seeking, self care, and medication. results from a population-based swedish study. *J Epidemiol Community Health*, 53(8):503–509.

Arendt-Nielsen, L. and Lautenbacher, S. (2004). Assessment of pain perception. In Lautenbacher, S. and Fillingim, R. B., editors, *Pathophysiology of Pain Perception*, chapter 2, pages 25–42. New York: Springer.

Arnow, B. A., Hunkeler, E. M., Blasey, C. M., Lee, J., Constantino, M. J., Fireman, B., Kraemer, H. C., Dea, R., Robinson, R., and Hayward, C. (2006). Comorbid depression, chronic pain, and disability in primary care. *Psychosom Med*, 68(2):262–268.

Arnstein, P., Caudill, M., Mandle, C. L., Norris, A., and Beasley, R. (1999). Self efficacy as a mediator of the relationship between pain intensity, disability and depression in chronic pain patients. *Pain*, 80(3):483–491.

Asmundson, G. J., Kuperos, J. L., and Norton, G. R. (1997a). Do patients with chronic pain selectively attend to pain-related information?: preliminary evidence for the mediating role of fear. *Pain*, 72(1-2):27–32.

Asmundson, G. J., Norton, G. R., and Allerdings, M. D. (1997b). Fear and avoidance in dysfunctional chronic back pain patients. *Pain*, 69(3):231–236.

Asmundson, G. J., Norton, P. J., and Norton, G. R. (1999). Beyond pain: the role of fear and avoidance in chronicity. *Clin Psychol Rev*, 19(1):97–119.

Asmundson, G. J. and Taylor, S. (1996). Role of anxiety sensitivity in pain-related fear and avoidance. *J Behav Med*, 19(6):577–586.

Asmundson, G. J. G., Collimore, K. C., Bernstein, A., Zvolensky, M. J., and Hadjistavropoulos, H. D. (2007). Is the latent structure of fear of pain continuous or discontinuous among pain patients? taxometric analysis of the pain anxiety symptoms scale. *J Pain*, 8(5):387–395.

Asmundson, G. J. G. and Wright, K. D. (2004). Biopsychosocial appproaches to pain. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 2, pages 35–57. Psychology Press.

Astin, J. A., Anton-Culver, H., Schwartz, C. E., Shapiro, D. H., McQuade, J., Breuer, A. M., Taylor, T. H., Lee, H., and Kurosaki, T. (1999). Sense of control and adjustment to breast cancer: the importance of balancing control coping styles. *Behav Med*, 25(3):101–109.

Averill, P. M., Novy, D. M., Nelson, D. V., and Berry, L. A. (1996). Correlates of depression in chronic pain patients: a comprehensive examination. *Pain*, 65(1):93–100.

Bair, M. J., Robinson, R. L., Katon, W., and Kroenke, K. (2003). Depression and pain comorbidity: a literature review. *Arch Intern Med*, 163(20):2433–2445.

Banks, S. M. and Kerns, R. D. (1996). Explaining high rates of depression in chronic pain: A diathesis-stress framework. *Psychological bulletin*, 119(1):95–110.

Barrett, L. (2006a). Are emotions natural kinds? *Perspectives on Psychological Science*, 1:28–58.

Barrett, L. (2006b). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review*, 10:20–46.

Barrett, L. and Wager, T. (2006). The structure of emotion: Evidence from neuroimaging studies. *Current Directions in Psychological Science*, 15:79–83.

Barrett, P. (2007). Structural equation modelling: Adjudging model fit. *Personality and Individual Differences*, 42(5):815–824.

Batten, S V; Follette, V. M. and Hayes, S. C. (1997). Experiential avoidance as a model for understanding psychological correlates of hiv and aids: Review, theory, and treatment implications. *The Behavior Therapist*, 20:99–103.

Beattie, P. F., Dowda, M., and Feuerstein, M. (2004). Differentiating sensory and affective-sensory pain descriptions in patients undergoing magnetic resonance imaging for persistent low back pain. *Pain*, 110(1-2):189–196.

Beckie, T. and Hayduk, L. (1997). Measuring quality of life. *Social Indicators Research*, 42:21–39.

Bedeian, A. G., Day, D. V., and Kelloway, E. K. (1997). Correcting for measurement error attenuation in structural equation models: Some important reminders. *Educational and Psychological Measurement*, 57:785–799.

Bennett, M. I., editor (2006). *Neuropathic pain*. Oxford University Press.

Bentler, P. M. (2000). Rites, wrongs, and gold in model testing. *Structural Equation Modeling: A Multidisciplinary Journal*, 7(1):82–91.

Bentler, P. M. (2004). *EQS 6 Structural Equations Program Manual*. Encino, CA: Multivariate Software, Inc.

Bentler, P. M. (2007). On tests and indices for evaluating structural models. *Personality and Individual Differences*, 42(5):825–829.

Berkley, K. J. (1997). Sex differences in pain. *Behav Brain Sci*, 20(3):371–80; discussion 435–513.

Berry, W. D. and Feldman, S. (1985). *Multiple Regression in Practice*. Sage University Paper Series on Quantitative Applications in the Social Sciences, series no. 07-050. Newbury Park, CA: Sage.

Billiet, J. B. and McClendon, M. J. (2000). Modeling acquiescence in measurement models for two balanced sets of items. *Structural Equation Modeling*, 7(4):608–628.

Blackburn-Munro, G. and Blackburn-Munro, R. E. (2001). Chronic pain, chronic stress and depression: coincidence or consequence? *J Neuroendocrinol*, 13(12):1009–1023.

Blackledge, J. T. and Barnes-Holmes, D. (2009). Core processes in acceptance and commitment therapy. In Blackledge, J., Ciarrochi, J., and Deane, F., editors, *Acceptance and Commitment Therapy: Contemporary theory, research, and practice*, pages 41–58. Bowen Hills, Australia: Australian Academic Press.

Blackledge, J. T. and Hayes, S. C. (2001). Emotion regulation in acceptance and commitment therapy. *J Clin Psychol*, 57(2):243–255.

Blyth, F. M., March, L. M., Brnabic, A. J. M., and Cousins, M. J. (2004). Chronic pain and frequent use of health care. *Pain*, 111(1-2):51–58.

Bollen, K. (1989). *Structural equations with latent variables*. NY: Wiley.

Bollen, K. A. (2000). Modeling strategies: In search of the holy grail. *Structural Equation Modeling: A Multidisciplinary Journal*, 7(1):74–81.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., and Coifman, K. (2004). The importance of being flexible: the ability to both enhance and suppress emotional expression predicts long-term adjustment. *Psychol Sci*, 15(7):482–487.

Bond, F. W., Hayes, S. C., Baer, R. A., Carpenter, K. M., Orcutt, H Kand Waltz, T., and Zettle, R. D. (submitted). Preliminary psychometric properties of the Acceptance and Action Questionnaire II: A revised measure of psychological flexibility and acceptance.

Bonica, J. J. (2001). *Bonica's management of pain*. Philadelphia : Lippincott Williams & Wilkins, 3rd edition.

Boomsma, A. (2000). Reporting analyses of covariance structures. *Structural Equation Modeling*, 7(3):461483.

Borsboom, D. (2005). *Measuring the Mind. Conceptual Issues in Contemporary Psychometrics*. Cambridge: Cambridge University Pres.

Botha-Scheepers, S., Riyazi, N., Kroon, H. M., Scharloo, M., Houwing-Duistermaat, J. J., Slagboom, E., Rosendaal, F. R., Breedveld, F. C., and Kloppenburg, M. (2006). Activity limitations in the lower extremities in patients with osteoarthritis: the modifying effects of illness perceptions and mental health. *Osteoarthritis Cartilage*, 14(11):1104–1110.

Bradley, L. A. and McKendree-Smith, N. L. (2001). Assessment of psychological status using interviews and self-report instruments. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 16, pages 292–319. New York: Guilford Press, 2nd edition.

Breivik, H., Collett, B., Ventafridda, V., Cohen, R., and Gallacher, D. (2006). Survey of chronic pain in europe: prevalence, impact on daily life, and treatment. *Eur J Pain*, 10(4):287–333.

Breugelmans, S. M., Poortinga, Y. H., Ambadar, Z., Setiadi, B., Vaca, J. B., Widiyanto, P., and Philippot, P. (2005). Body sensations associated with emotions in rarmuri indians, rural javanese, and three student samples. *Emotion*, 5(2):166–174.

Broadbent, E., Ellis, C. J., Gamble, G., and Petrie, K. J. (2006a). Changes in patient drawings of the heart identify slow recovery after myocardial infarction. *Psychosom Med*, 68(6):910–913.

Broadbent, E., Ellis, C. J., Thomas, J., Gamble, G., and Petrie, K. J. (2009a). Can an illness perception intervention reduce illness anxiety in spouses of myocardial infarction patients? a randomized controlled trial. *J Psychosom Res*, 67(1):11–15.

Broadbent, E., Ellis, C. J., Thomas, J., Gamble, G., and Petrie, K. J. (2009b). Further development of an illness perception intervention for myocardial infarction patients: a randomized controlled trial. *J Psychosom Res*, 67(1):17–23.

Broadbent, E., Niederhoffer, K., Hague, T., Corter, A., and Reynolds, L. (2009c). Headache sufferers' drawings reflect distress, disability and illness perceptions. *J Psychosom Res*, 66(5):465–470.

Broadbent, E., Petrie, K. J., Ellis, C. J., Ying, J., and Gamble, G. (2004). A picture of health–myocardial infarction patients' drawings of their hearts and subsequent disability: a longitudinal study. *J Psychosom Res*, 57(6):583–587.

Broadbent, E., Petrie, K. J., Main, J., and Weinman, J. (2006b). The brief illness perception questionnaire. *J Psychosom Res*, 60(6):631–637.

Brown, G. W. and Harris, T. (1978). *Social origins of depression: a study of psychiatric disorder in women*. London: Taylor & Francis.

Bruehl, S., al'Absi, M., France, C. R., France, J., Harju, A., Burns, J. W., and Chung, O. Y. (2007a). Anger management style and endogenous opioid function: is gender a moderator? *J Behav Med*, 30(3):209–219.

Bruehl, S., Burns, J. W., Chung, O. Y., and Chont, M. (2009). Pain-related effects of trait anger expression: neural substrates and the role of endogenous opioid mechanisms. *Neurosci Biobehav Rev*, 33(3):475–491.

Bruehl, S., Burns, J. W., Chung, O. Y., and Quartana, P. (2008a). Anger management style and emotional reactivity to noxious stimuli among chronic pain patients and healthy controls: the role of endogenous opioids. *Health Psychol*, 27(2):204–214.

Bruehl, S., Burns, J. W., Chung, O. Y., Ward, P., and Johnson, B. (2002). Anger and pain sensitivity in chronic low back pain patients and pain-free controls: the role of endogenous opioids. *Pain*, 99(1-2):223–233.

Bruehl, S., Carlson, C. R., and McCubbin, J. A. (1993). Two brief interventions for acute pain. *Pain*, 54(1):29–36.

Bruehl, S., Chung, O. Y., and Burns, J. W. (2003a). Differential effects of expressive anger regulation on chronic pain intensity in CRPS and non-CRPS limb pain patients. *Pain*, 104(3):647–654.

Bruehl, S., Chung, O. Y., and Burns, J. W. (2006a). Anger expression and pain: an overview of findings and possible mechanisms. *J Behav Med*, 29(6):593–606.

Bruehl, S., Chung, O. Y., and Burns, J. W. (2008b). The mu opioid receptor A118G gene polymorphism moderates effects of trait anger-out on acute pain sensitivity. *Pain*, 139(2):406–415.

Bruehl, S., Chung, O. Y., Burns, J. W., and Biridepalli, S. (2003b). The association between anger expression and chronic pain intensity: evidence for partial mediation by endogenous opioid dysfunction. *Pain*, 106(3):317–324.

Bruehl, S., Chung, O. Y., Burns, J. W., and Diedrich, L. (2007b). Trait anger expressiveness and pain-induced beta-endorphin release: support for the opioid dysfunction hypothesis. *Pain*, 130(3):208–215.

Bruehl, S., Chung, O. Y., Donahue, B. S., and Burns, J. W. (2006b). Anger regulation style, postoperative pain, and relationship to the a118g mu opioid receptor gene polymorphism: a preliminary study. *J Behav Med*, 29(2):161–169.

Brugha, T., Bebbington, P., Tennant, C., and Hurry, J. (1985). The List of Threatening Experiences: a subset of 12 life event categories with considerable long-term contextual threat. *Psychol Med*, 15(1):189–194.

Brugha, T. S. and Cragg, D. (1990). The List of Threatening Experiences: the reliability and validity of a brief life events questionnaire. *Acta Psychiatr Scand*, 82(1):77–81.

Buck, R. (1999). The biological affects: a typology. *Psychol Rev*, 106(2):301–336.

Burns, J. W. (1997). Anger management style and hostility: predicting symptom-specific physiological reactivity among chronic low back pain patients. *J Behav Med*, 20(6):505–522.

Burns, J. W. and Bruehl, S. (2005). Anger management style, opioid analgesic use, and chronic pain severity: a test of the opioid-deficit hypothesis. *J Behav Med*, 28(6):555–563.

Burns, J. W., Bruehl, S., and Caceres, C. (2004). Anger management style, blood pressure reactivity, and acute pain sensitivity: evidence for "Trait x Situation" models. *Ann Behav Med*, 27(3):195–204.

Burns, J. W., Bruehl, S., and Quartana, P. J. (2006). Anger management style and hostility among patients with chronic pain: effects on symptom-specific physiological reactivity during anger- and sadness-recall interviews. *Psychosom Med*, 68(5):786–793.

Burns, J. W., Holly, A., Quartana, P., Wolff, B., Gray, E., and Bruehl, S. (2008a). Trait anger management style moderates effects of actual ("state") anger regulation on symptom-specific reactivity and recovery among chronic low back pain patients. *Psychosom Med*, 70(8):898–905.

Burns, J. W., Johnson, B. J., Mahoney, N., Devine, J., and Pawl, R. (1996). Anger management style, hostility and spouse responses: gender differences in predictors of adjustment among chronic pain patients. *Pain*, 64(3):445–453.

Burns, J. W., Kubilus, A., and Bruehl, S. (2003). Emotion induction moderates effects of anger management style on acute pain sensitivity. *Pain*, 106(1-2):109–118.

Burns, J. W., Quartana, P., Gilliam, W., Gray, E., Matsuura, J., Nappi, C., Wolfe, B., and Lofland, K. (2008b). Effects of anger suppression on pain severity and pain behaviors among chronic pain patients: evaluation of an ironic process model. *Health Psychol*, 27(5):645–652.

Burns, J. W., Quartana, P. J., and Bruehl, S. (2008c). Anger inhibition and pain: conceptualizations, evidence and new directions. *J Behav Med*, 31(3):259–279.

Burns, J. W., Quartana, P. J., and Bruehl, S. (2009). Anger management style moderates effects of attention strategy during acute pain induction on physiological responses to subsequent mental stress and recovery: a comparison of chronic pain patients and healthy nonpatients. *Psychosom Med*, 71(4):454–462.

Byrne, M., Walsh, J., and Murphy, A. W. (2005). Secondary prevention of coronary heart disease: patient beliefs and health-related behaviour. *J Psychosom Res*, 58(5):403–415.

Cacioppo, J., Tassinary, L., and Berntson, G. (2000). Psychophysiological science. In Cacioppo, J., Tassinary, L., and Berntson, G., editors, *Handbook of psychophysiology*, pages 3–26. New York: Cambridge University Press.

Cacioppo, J. T. (2003). Introduction: Emotion and health. In Davidson, R. J., Sherer, K. R., and Goldsmith, H. H., editors, *Handbook of affective sciences*, chapter 55, pages 1047–1052. New York: Oxford University Press.

Cameron, L., Leventhal, E. A., and Leventhal, H. (1993). Symptom representations and affect as determinants of care seeking in a community-dwelling, adult sample population. *Health Psychol*, 12(3):171–179.

Campbell, L. C., Clauw, D. J., and Keefe, F. J. (2003). Persistent pain and depression: a biopsychosocial perspective. *Biol Psychiatry*, 54(3):399–409.

Chamberlin, T. C. (1890). The method of multiple working hypotheses. *Science*, 15:92–96. reprinted in Science 148:754-759 [1965].

Chapman, R. C. (2004). Pain perception, affective mechanisms, and conscious experience. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 3, pages 59–85. Psychology Press.

Clark, W. C., Kuhl, J. P., Keohan, M. L., Knotkova, H., Winer, R. T., and Griswold, G. A. (2003). Factor analysis validates the cluster structure of the dendrogram underlying the Multidimensional Affect and Pain Survey (MAPS) and challenges the a priori classification of the descriptors in the McGill Pain Questionnaire (MPQ). *Pain*, 106(3):357–363.

Clark, W. C., Yang, J. C., Tsui, S. L., Ng, K.-F., and Clark, S. B. (2002). Unidimensional pain rating scales: a multidimensional affect and pain survey (MAPS) analysis of what they really measure. *Pain*, 98(3):241–247.

Clatworthy, J., Buick, D., Hankins, M., Weinman, J., and Horne, R. (2005). The use and reporting of cluster analysis in health psychology: a review. *Br J Health Psychol*, 10(Pt 3):329–358.

Cleland, J. A., Fritz, J. M., and Brennan, G. P. (2008). Predictive validity of initial fear avoidance beliefs in patients with low back pain receiving physical therapy: is the fabq a useful screening tool for identifying patients at risk for a poor recovery? *Eur Spine J*, 17(1):70–79.

Cohen, J., Cohen, P., West, S. G., and Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences*. Hillsdale: Erlbaum, 3 edition.

Connelly, M., Keefe, F. J., Affleck, G., Lumley, M. A., Anderson, T., and Waters, S. (2007). Effects of day-to-day affect regulation on the pain experience of patients with rheumatoid arthritis. *Pain*, 131(1-2):162–170.

Conner, T. S., Tennen, H., Zautra, A. J., Affleck, G., Armeli, S., and Fifield, J. (2006). Coping with rheumatoid arthritis pain in daily life: within-person analyses reveal hidden vulnerability for the formerly depressed. *Pain*, 126(1-3):198–209.

Consedine, N. S. (2008). Health-promoting and health-damaging effects of emotions: The view from developmental functionalism. In Lewis, M., Haviland-Jones, J. M., and Barrett, L. F., editors, *Handbook of Emotions*, chapter 42. New York: Guilford Press, 3 edition.

Consedine, N. S. and Moskowitz, J. T. (2007). The role of discrete emotions in health outcomes: A critical review. *Applied and Preventive Psychology*, 12:59–75.

Cook, A. J., Brawer, P. A., and Vowles, K. E. (2006). The fear-avoidance model of chronic pain: validation and age analysis using structural equation modeling. *Pain*, 121(3):195–206.

Cordova, M. J., Walser, R., Neff, J., and Ruzek, J. I. (2005). Predictors of emotional adjustment following traumatic injury: personal, social, and material resources. *Prehosp Disaster Med*, 20(1):7–13.

Covic, T., Adamson, B., Spencer, D., and Howe, G. (2003). A biopsychosocial model of pain and depression in rheumatoid arthritis: a 12-month longitudinal study. *Rheumatology (Oxford)*, 42(11):1287–1294.

Cox, B. J., Kuch, K., Parker, J. D., Shulman, I. D., and Evans, R. J. (1994). Alexithymia in somatoform disorder patients with chronic pain. *J Psychosom Res*, 38(6):523–527.

Craig, K., Prkachin, K., and Grunau, R. (2001). The facial expression of pain. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 9, pages 153–169. New York: Guilford Press, 2nd edition.

Craig, K. D. and Hadjistavropoulos, T. (2004). Psychological perspectives on pain: Controversies. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 11, pages 303–326. Psychology Press.

Crawford, C. S. (2009). From pleasure to pain: The role of the MPQ in the language of phantom limb pain. *Soc Sci Med*.

Crocker, L. M. and Algina, J. (1986). *Introduction to classical and modern test theory*. New York: Holt, Rinehart and Winston.

Crombez, G., Vlaeyen, J. W., Heuts, P. H., and Lysens, R. (1999). Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. *Pain*, 80(1-2):329–339.

Currie, S. R. and Wang, J. (2005). More data on major depression as an antecedent risk factor for first onset of chronic back pain. *Psychol Med*, 35(9):1275–1282.

Dahl, J., Wilson, K. G., Luciano, C., and Hayes, S. C. (2005). *Acceptance and Commitment Therapy for Chronic Pain*. Reno, NV: Context Press.

Dahl, J., Wilson, K. G., and Nilsson, A. (2004). Acceptance and commitment therapy and the treatment of persons at risk for long-term disability resulting from stress and pain symptoms: A preliminary randomized trial. *Behavior Therapy*, 35(4):785–801.

Damme, S. V., Crombez, G., and Eccleston, C. (2008). Coping with pain: a motivational perspective. *Pain*, 139(1):1–4.

Davis, P. J., Reeves, J. L., Hastie, B. A., Graff-Radford, S. B., and Naliboff, B. D. (2000). Depression determines illness conviction and pain impact: a structural equation modeling analysis. *Pain Med*, 1(3):238–246.

de Wied, M. and Verbaten, M. N. (2001). Affective pictures processing, attention, and pain tolerance. *Pain*, 90(1-2):163–172.

DeGood, D. and Tait, R. (2001). Assessment of pain beliefs and pain coping. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 17, pages 320–345. New York: Guilford Press, 2nd edition.

Deyo, R. A. (1986). Comparative validity of the sickness impact profile and shorter scales for functional assessment in low-back pain. *Spine*, 11(9):951–954.

Dickens, C., McGowan, L., and Dale, S. (2003). Impact of depression on experimental pain perception: a systematic review of the literature with meta-analysis. *Psychosom Med*, 65(3):369–375.

Diefenbach, M. A. (2008). Cognitive and emotional processing of health information from a self-regulation perspective. *Emotion Researcher*, 23(1):12–15.

Diefenbach, M. A., Miller, S. M., Porter, M., Peters, E., Stefanek, M., and Leventhal, H. (2008). Emotions and health behavior: A self-regulation perspective. In Lewis, M., Haviland-Jones, J. M., and Barrett, L. F., editors, *Handbook of Emotions*, chapter 40, pages 645–660. New York (NY): Guildford, 3 edition.

Dima, A. (2009). The interpretation of facial behaviour in communication: From universal codes to intersubjective situated meaning. *Studies in Communication Science*, 9(1):51–72.

Dooremalen, H., de Regt, H., and M., S. (2007). *Exploring Humans, An Introduction to the Philosophy of the Social Sciences*. Amsterdam: Boom.

Dunn, K. M., Croft, P. R., Main, C. J., and Korff, M. V. (2008). A prognostic approach to defining chronic pain: replication in a UK primary care low back pain population. *Pain*, 135(1-2):48–54.

Dworkin, S. and Sherman, J. (2001). Relying on objective and subjective measures of chronic pain: Guidelines for use and interpretation. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 32, pages 619–638. New York: Guilford Press, 2nd edition.

Eccleston, C. and Crombez, G. (1999). Pain demands attention: a cognitive-affective model of the interruptive function of pain. *Psychol Bull*, 125(3):356–366.

Eccleston, C. and Crombez, G. (2007). Worry and chronic pain: a misdirected problem solving model. *Pain*, 132(3):233–236.

Eccleston, C., Crombez, G., Aldrich, S., and Stannard, C. (2001). Worry and chronic pain patients: a description and analysis of individual differences. *Eur J Pain*, 5(3):309–318.

Edwards, R. R., Moric, M., Husfeldt, B., Buvanendran, A., and Ivankovich, O. (2005a). Ethnic similarities and differences in the chronic pain experience: a comparison of african american, hispanic, and white patients. *Pain Med*, 6(1):88–98.

Edwards, R. R., Sarlani, E., Wesselmann, U., and Fillingim, R. B. (2005b). Quantitative assessment of experimental pain perception: multiple domains of clinical relevance. *Pain*, 114(3):315–319.

Ekman, P. (1994). Strong evidence for universals in facial expressions: a reply to Russell's mistaken critique. *Psychol Bull*, 115(2):268–287.

Ekman, P. (1999). Basic emotions. In Dalgleish, T. and Power, M., editors, *Handbook of Cognition and Emotion*. Sussex, U.K.: John Wiley & Sons, Ltd.

Ekman, P. and Friesen, W. V. (1971). Constants across cultures in the face and emotion. *J Pers Soc Psychol*, 17(2):124–129.

Elliott, A. M., Smith, B. H., Penny, K. I., Smith, W. C., and Chambers, W. A. (1999). The epidemiology of chronic pain in the community. *Lancet*, 354(9186):1248–1252.

Elliott, L. P. and Brook, B. W. (2007). Revisiting Chamberlin: Multiple working hypotheses for the 21st century. *Bioscience*, 57(7):608–614.

Ellis, A. (2005). Can Rational-Emotive Behavior Therapy (REBT) and Acceptance and Commitment Therapy (ACT) resolve their differences and be integrated? *Journal of Rational-Emotive & Cognitive-Behavior Therapy*, 23(2):153–168.

Ellsworth, P. C. and Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, K. R. S. and Goldsmith, H. H., editors, *Handbook of affective sciences*, chapter 29, pages 572–595. New York: Oxford University Press.

Engel, G. L. (1959). Psychogenic pain and pain-prone patient. *Am J Med*, 26(6):899–918.

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science*, 196(4286):129–136.

Everitt, B. S., Landau, S., and Leese, M. (2001). *Cluster Analysis*. London: Arnold, 4th edition.

Evers, A. W., Kraaimaat, F. W., van Lankveld, W., Jongen, P. J., Jacobs, J. W., and Bijlsma, J. W. (2001). Beyond unfavorable thinking: the illness cognition questionnaire for chronic diseases. *J Consult Clin Psychol*, 69(6):1026–1036.

Faul, F., Erdfelder, E., Lang, A. G., and Buchner, A. (2007). G*power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39:175–191.

Feldner, M. T., Hekmat, H., Zvolensky, M. J., Vowles, K. E., Secrist, Z., and Leen-Feldner, E. W. (2006). The role of experiential avoidance in acute pain tolerance: a laboratory test. *J Behav Ther Exp Psychiatry*, 37(2):146–158.

Fernandez, E. and Boyle, G. J. (2001). Affective and evaluative descriptors of pain in the mcgill pain questionnaire: reduction and reorganization. *J Pain*, 2(6):318–325.

Fernandez, E. and Milburn, T. W. (1994). Sensory and affective predictors of overall pain and emotions associated with affective pain. *Clin J Pain*, 10(1):3–9.

Fernandez, E. and Towery, S. (1996). A parsimonious set of verbal descriptors of pain sensation derived from the mcgill pain questionnaire. *Pain*, 66(1):31–37.

Fernandez, E. and Turk, D. C. (1992). Sensory and affective components of pain: separation and synthesis. *Psychol Bull*, 112(2):205–217.

Fernandez, E. and Turk, D. C. (1995). The scope and significance of anger in the experience of chronic pain. *Pain*, 61(2):165–175.

Fernandez-Dols, J. M. and Ruiz-Belda, M. A. (1997). Spontaneous facial behavior during intense emotional episodes: Artistic truth and optical truth. In Russell, J. A. and Fernandez-Dols,

J. M., editors, *The psychology of facial expression*, chapter 11, pages 254–273. Cambridge, UK: Cambridge University Press.

Field, A. (2005). *Discovering Statistics Using SPSS*. London, Thousand Oaks, New Delhi: Sage Publications.

Figueiras, M. J. and Weinman, J. (2003). Do similar patient and spouse perceptions of myocardial infarction predict recovery? *Psychology and Health*, 18(2):201–216.

Fillingim, R. B. and Lautenbacher, S. (2004). The importance of quantitative sensory testing in the clinical setting. In Lautenbacher, S. and Fillingim, R. B., editors, *Pathophysiology of Pain Perception*, chapter 15, pages 215–27. New York: Springer.

Fishbain, D. A., Cutler, R., Rosomoff, H. L., and Rosomoff, R. S. (1997). Chronic pain-associated depression: antecedent or consequence of chronic pain? a review. *Clin J Pain*, 13(2):116–137.

Fontaine, J. R. J., Scherer, K. R., Roesch, E. B., and Ellsworth, P. C. (2007). The world of emotions is not two-dimensional. *Psychol Sci*, 18(12):1050–1057.

Fordyce, W. E. (1976). *Behavioral Methods for Chronic Pain and Illness*. St. Louis: C.V. Mosby.

Fortune, D. G., Richards, H. L., Main, C. J., and Griffiths, C. E. M. (2000). Pathological worrying, illness perceptions and disease severity in patients with psoriasis. *British Journal of Health Psychology*, 5(1):71–82.

Foster, N. E., Bishop, A., Thomas, E., Main, C., Horne, R., Weinman, J., and Hay, E. (2008). Illness perceptions of low back pain patients in primary care: what are they, do they change and are they associated with outcome? *Pain*, 136(1-2):177–187.

French, D. P., Cooper, A., and Weinman, J. (2006). Illness perceptions predict attendance at cardiac rehabilitation following acute myocardial infarction: a systematic review with meta-analysis. *J Psychosom Res*, 61(6):757–767.

French, D. P., Lewin, R. J. P., Watson, N., and Thompson, D. R. (2005). Do illness perceptions predict attendance at cardiac rehabilitation and quality of life following myocardial infarction? *J Psychosom Res*, 59(5):315–322.

Frijda, N. (2008). The psychologists' point of view. In Lewis, M., Haviland-Jones, J. M., and Barrett, L. F., editors, *Handbook of Emotions*, chapter 5, pages 68–87. New York: Guilford Press, 3 edition.

Frijda, N. H. and Tcherkassof, A. (1997). Facial expressions as modes of action readiness. In Russell, J. A. and Fernandez-Dols, J. M., editors, *The psychology of facial expression*, chapter 4, pages 78–102. Cambridge, UK: Cambridge University Press.

Friman, P. C., Hayes, S. C., and Wilson, K. G. (1998). Why behavior analysts should study emotion: the example of anxiety. *J Appl Behav Anal*, 31(1):137–156.

Fritz, J. M., George, S. Z., and Delitto, A. (2001). The role of fear-avoidance beliefs in acute low back pain: relationships with current and future disability and work status. *Pain*, 94(1):7–15.

Gagliese, L. and Melzack, R. (1997). Chronic pain in elderly people. *Pain*, 70(1):3–14.

Gagliese, L. and Melzack, R. (2003). Age-related differences in the qualities but not the intensity of chronic pain. *Pain*, 104(3):597–608.

Gaskin, M. E., Greene, A. F., Robinson, M. E., and Geisser, M. E. (1992). Negative affect and the experience of chronic pain. *J Psychosom Res*, 36(8):707–713.

Gatchel, R. J. and Okifuji, A. (2006). Evidence-based scientific data documenting the treatment and cost-effectiveness of comprehensive pain programs for chronic nonmalignant pain. *J Pain*, 7(11):779–793.

Geerlings, S. W., Twisk, J. W. R., Beekman, A. T. F., Deeg, D. J. H., and van Tilburg, W. (2002). Longitudinal relationship between pain and depression in older adults: sex, age and physical disability. *Soc Psychiatry Psychiatr Epidemiol*, 37(1):23–30.

Geisser, M. E., Robinson, M. E., Keefe, F. J., and Weiner, M. L. (1994). Catastrophizing, depression and the sensory, affective and evaluative aspects of chronic pain. *Pain*, 59(1):79–83.

Gelman, A. and Hill, J. (2007). *Data analysis using regression and multilevel/ hierarchical models*. Cambridge, UK: Cambridge University Press.

Gheldof, E. L. M., Vinck, J., den Bussche, E. V., Vlaeyen, J. W. S., Hidding, A., and Crombez, G. (2006). Pain and pain-related fear are associated with functional and social disability in an occupational setting: evidence of mediation by pain-related fear. *Eur J Pain*, 10(6):513–525.

Gibson, S. J. and Chambers, C. T. (2004). Pain over the life span: A developmental pespective. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 5, pages 113–153. Psychology Press.

Gillanders, D. (in press). Acceptance and commitment therapy and motivational interviewing for health behaviour change. In McCracken, L., editor, *Acceptance and Commitment Therapy and Mindfulness-Based Approaches in Behavioral Medicine: An Integrative Clinical and Research Guide*. Oakland: New Harbinger.

Glaros, A. G. and Lumley, M. A. (2005). Alexithymia and pain in temporomandibular disorder. *J Psychosom Res*, 59(2):85–88.

Goubert, L., Crombez, G., and Damme, S. V. (2004a). The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. *Pain*, 107(3):234–241.

Goubert, L., Crombez, G., Eccleston, C., and Devulder, J. (2004b). Distraction from chronic pain during a pain-inducing activity is associated with greater post-activity pain. *Pain*, 110(1-2):220–227.

Gracely, R. H. (1992). Evaluation of multi-dimensional pain scales. *Pain*, 48(3):297–300.

Graham, J. E., Lobel, M., Glass, P., and Lokshina, I. (2008). Effects of written anger expression in chronic pain patients: making meaning from pain. *J Behav Med*, 31(3):201–212.

Gray, J. A. and McNaughton, N. (2003). *The Neuropsychology of Anxiety: An Enquiry into the Functions of the Septo-Hippocampal System*. NY: Oxford University Press, 2nd edition.

Greenberg, J. and Burns, J. W. (2003). Pain anxiety among chronic pain patients: specific phobia or manifestation of anxiety sensitivity? *Behav Res Ther*, 41(2):223–240.

Gregg, J. A., Callaghan, G. M., Hayes, S. C., and Glenn-Lawson, J. L. (2007). Improving diabetes self-management through acceptance, mindfulness, and values: a randomized controlled trial. *J Consult Clin Psychol*, 75(2):336–343.

Groarke, A. M., Curtis, R., Coughlan, R., and Gsel, A. (2005). The impact of illness representations and disease activity on adjustment in women with rheumatoid arthritis: A longitudinal study. *Psychology and Health*, 20(5):597–613.

Groenblad, M., Lukinmaa, A., and Konttinen, Y. T. (1990). Chronic low-back pain: intercorrelation of repeated measures for pain and disability. *Scand J Rehabil Med*, 22(2):73–77.

Gross, J. J. (1999). Emotion regulation: Past, present, future. *Cognition and Emotion*, 13(5):551–573.

Gross, J. J. (2008). Emotion regulation. In Lewis, M., Haviland-Jones, J. M., and Barrett, L. F., editors, *Handbook of Emotions*, chapter 31, pages 497–512. New York: Guilford Press, 3rd edition.

Guest, G. H. and Drummond, P. D. (1992). Effect of compensation on emotional state and disability in chronic back pain. *Pain*, 48(2):125–130.

Gutierrez-Martinez, O., Luciano-Soriano, C., Rodriguez-Valverde, M., and Fink, B. C. (2004). Comparison between an acceptance-based and a cognitive-control-based protocol for coping with pain. *Behavior Therapy*, 35:767–783.

Hadjistavropoulos, H. D. and Williams, A. C. d. C. (2004). Psychological interventions and chronic pain. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 10, pages 271–301. Psychology Press.

Hadjistavropoulos, T. and Craig, K. D. (2004). An introduction to pain: Psychological perspectives. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, pages 1–12. Psychology Press.

Hadjistavropoulos, T., Craig, K. D., and Fuchs-Lacelle, S. (2004). Social influences and the communication of pain. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 4, pages 87–112. Psychology Press.

Hall, R. J., Snell, A. F., and Foust, M. S. (1999). Item parceling strategies in SEM: investigating the subtle effects of unmodeled secondary constructs. *Organizational Research Methods*, 2(3):233–256.

Hallberg and Carlsson (1998). Anxiety and coping in patients with chronic work-related muscular pain and patients with fibromyalgia. *Eur J Pain*, 2(4):309–319.

Hamilton, N. A., Karoly, P., and Kitzman, H. (2004). Self-regulation and chronic pain: The role of emotion. *Cognitive Therapy and Research*, 28(5):559–576.

Harstall, C; Ospina, M. (2003). How prevalent is chronic pain? *Pain: Clinical Updates*, 9(2):1–4.

Hasenbring, M. I., Hallner, D., and Rusu, A. C. (2009). Comment on: Chronic pain: avoidance or endurance? by Petra Karsdorp and Johan Vlaeyen. *Eur J Pain*, 13(6):662–663.

Haslam, N. and Kim, H. C. (2002). Categories and continua: a review of taxometric research. *Genet Soc Gen Psychol Monogr*, 128(3):271–320.

Hayduk, L. A. (1996). *LISREL issues, debates, and strategies*. Baltimore: Johns Hopkins University Press.

Hayduk, L. A. and Glaser, D. N. (2000a). Doing the four-step, right-2-3, wrong-2-3: A brief reply to Mulaik and Millsap; Bollen; Bentler; and Herting and Costner. *Structural Equation Modeling: A Multidisciplinary Journal*, 7(1):111–123.

Hayduk, L. A. and Glaser, D. N. (2000b). Jiving the four-step, waltzing around factor analysis, and other serious fun. *Structural Equation Modeling: A Multidisciplinary Journal*, 7(1):1–35.

Hayes, S. C. (1993). Analytic goals and the varieties of scientific contextualism. In Hayes, S. C., Hayes, L. J., Reese, H. W., and Sarbin, T. R., editors, *Varieties of scientific contextualism*, chapter 1, pages 11–27. Reno, NV: Context Press.

Hayes, S. C. (2009). The Importance of RFT to the Development of Contextual Behavioral Science. In *Presidential address at the Association for Contextual Behavioral Science 3rd World Congress, University of Twente, Netherlands*.

Hayes, S. C., Barnes-Holmes, D., and Roche, B., editors (2001). *Relational Frame Theory: A Post-Skinnerian account of human language and cognition*. New York: Plenum Press.

Hayes, S. C., Bissett, R., Korn, Z., Zettle, R. D., Rosenfarb, I., Cooper, L., and Grundt, A. (1999a). The impact of acceptance versus control rationales on pain tolerance. *The Psychological Record*, 49(1):33–47.

Hayes, S. C., Hayes, L. J., and Reese, H. W. (1988). Finding the philosophical core: A review of Stephen C. Pepper's World Hypotheses. *Journal of the Experimental Analysis of Behavior*, 50:97–111.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., and Lillis, J. (2006). Acceptance and commitment therapy: model, processes and outcomes. *Behav Res Ther*, 44(1):1–25.

Hayes, S. C. and Shenk, C. (2004). Operationalizing mindfulness without unnecessary attachments. *Clinical psychology: Science and practice*, 11:249–254.

Hayes, S. C. and Smith, S. (2005). *Get out of your mind and into your life: The new Acceptance and Commitment Therapy*. Oakland, CA: New Harbinger.

Hayes, S. C., Stosahl, K., Wilson, K. G., Bissett, R. T., Pistorello, J., Toarmino, D., Polusny, M. A., Dykstra, T. A., V, B. S., Bergan, J., Stewart, S. H., Zvolensky, M. J., Eifert, G. H., and Bond, F. (2004a). Measuring experiential avoidance- a preliminary test of a working model. *The Psychological Record*, 54:553–578.

Hayes, S. C., Strosahl, K., and Wilson, K. G. (1999b). *Acceptance and commitment therapy: an experiential approach to behavior change*. New York (NY): Guildford.

Hayes, S. C., Strosahl, K. D., Bunting, K., Twohig, M., and Wilson, K. G. (2004b). What is Acceptance and Commitment Therapy? In Hayes, S. C. and Strosahl, K. D., editors, *A practical guide to Acceptance and Commitment Therapy*, chapter 1, pages 1–30. New York: Springer-Verlag.

Hayes, S. C., Strosahl, K. D., Bunting, K., Twohig, M., and Wilson, K. G. (2004c). What is acceptance and commitment therapy? In Hayes, S. C. and Strosahl, K. D., editors, *A practical guide to Acceptance and Commitment Therapy*, chapter 1, pages 3–29. New York: Springer-Verlag.

Heath, R. L., Saliba, M., Mahmassani, O., Major, S. C., and Khoury, B. A. (2008). Locus of control moderates the relationship between headache pain and depression. *J Headache Pain*, 9(5):301–308.

Hedeker, D. and Gibbons, R. D. (2006). *Longitudinal Data Analysis*. Hoboken, NJ: Wiley-Interscience.

Heffner, M; Eifert, G. H. and Parker, B. T. (2003). Valued directions: Acceptance and commitment therapy in the treatment of alcohol dependence. *Cognitive and Behavioral Practice*, 1(4):378–383.

Heffner, M; Sperry, J. and Eifert, G. H. (2002). Acceptance and commitment therapy in the treatment of an adolescent female with anorexia nervosa: A case example. *Cognitive and Behavioral Practice*, 9(3):232–236.

Heijmans, M. and de Ridder, D. (1998). Assessing illness representations of chronic illness: explorations of their disease-specific nature. *J Behav Med*, 21(5):485–503.

Heijmans, M. J. (1998). Coping and adaptive outcome in chronic fatigue syndrome: importance of illness cognitions. *J Psychosom Res*, 45(1 Spec No):39–51.

Henderson, C. J., Hagger, M. S., and Orbell, S. (2007). Does priming a specific illness schema result in an attentional information-processing bias for specific illnesses? *Health Psychol*, 26(2):165–173.

Hirani, S. P., Pugsley, W. B., and Newman, S. P. (2006). Illness representations of coronary artery disease: an empirical examination of the illness perceptions questionnaire (ipq) in patients undergoing surgery, angioplasty and medication. *Br J Health Psychol*, 11(Pt 2):199–220.

Hobro, N., Weinman, J., and Hankins, M. (2004). Using the self-regulatory model to cluster chronic pain patients: the first step towards identifying relevant treatments? *Pain*, 108(3):276–283.

Hofmann, S. G. and Asmundson, G. J. G. (2008). Acceptance and mindfulness-based therapy: new wave or old hat? *Clin Psychol Rev*, 28(1):1–16.

Holroyd, K. A., Holm, J. E., Keefe, F. J., Turner, J. A., Bradley, L. A., Murphy, W. D., Johnson, P., Anderson, K., Hinkle, A. L., and O'Malley, W. B. (1992). A multi-center evaluation of the McGill Pain Questionnaire: results from more than 1700 chronic pain patients. *Pain*, 48(3):301–311.

Holroyd, K. A., Talbot, F., Holm, J. E., Pingel, J. D., Lake, A. E., and Saper, J. R. (1996). Assessing the dimensions of pain: a multitrait-multimethod evaluation of seven measures. *Pain*, 67(2-3):259–265.

Horne, R., Buick, D., Fisher, M., Leake, H., Cooper, V., and Weinman, J. (2004). Doubts about necessity and concerns about adverse effects: identifying the types of beliefs that are associated with non-adherence to haart. *Int J STD AIDS*, 15(1):38–44.

Horne, R., James, D., Petrie, K., Weinman, J., and Vincent, R. (2000). Patients' interpretation of symptoms as a cause of delay in reaching hospital during acute myocardial infarction. *Heart*, 83(4):388–393.

Horne, R. and Weinman, J. (1999). Patients' beliefs about prescribed medicines and their role in adherence to treatment in chronic physical illness. *J Psychosom Res*, 47(6):555–567.

Hu, L. and Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, 6(1):1–55.

Huber, A., Suman, A. L., Biasi, G., and Carli, G. (2009). Alexithymia in fibromyalgia syndrome: associations with ongoing pain, experimental pain sensitivity and illness behavior. *J Psychosom Res*, 66(5):425–433.

Izard, C. E. (1993). Four systems for emotion activation: cognitive and noncognitive processes. *Psychol Rev*, 100(1):68–90.

Izard, C. E. (1994). Innate and universal facial expressions: evidence from developmental and cross-cultural research. *Psychol Bull*, 115(2):288–299.

Jackson, D. L., Gillaspy, J. A., and Purc-Stephenson, R. (2009). Reporting practices in confirmatory factor analysis: an overview and some recommendations. *Psychol Methods*, 14(1):6–23.

Jackson, J. E. (2005). Stigma, liminality, and chronic pain: Mind-body borderlands. *American Ethnologist*, 32(3):332–353.

Jacob, M. C. and Kerns, R. D. (2001). Assessment of the psychosocial context of the experience of chronic pain. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 18, pages 362–384. New York: Guilford Press, 2nd edition.

James, J. E. and Hardardottir, D. (2002). Influence of attention focus and trait anxiety on tolerance of acute pain. *Br J Health Psychol*, 7(Pt 2):149–162.

Jamison, R. N., Raymond, S. A., Levine, J. G., Slawsby, E. A., Nedeljkovic, S. S., and Katz, N. P. (2001). Electronic diaries for monitoring chronic pain: 1-year validation study. *Pain*, 91(3):277–285.

Jamison, R. N., Raymond, S. A., Slawsby, E. A., McHugo, G. J., and Baird, J. C. (2006). Pain assessment in patients with low back pain: comparison of weekly recall and momentary electronic data. *J Pain*, 7(3):192–199.

Janz, N. K., Champion, V. L., and Strecher, V. J. (2002). The health belief model. In Glanz, K., Rimer, B. K., and Lewis, F. M., editors, *Health Behavior and Health Education: Theory, Research and Practice*, chapter 3, pages 45–66. California: Jossey-Bass, 3 edition.

Jay, G. W. (2007). *Chronic Pain*. CRC Press.

Jensen, M. and Karoly, P. (2001). Self-report scales and procedures for assessing pain in adults. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 2, pages 15–34. New York: Guilford Press, 2nd edition.

Jensen, M. P., Strom, S. E., Turner, J. A., and Romano, J. M. (1992). Validity of the Sickness Impact Profile Roland scale as a measure of dysfunction in chronic pain patients. *Pain*, 50(2):157–162.

Jensen, M. P., Turner, J. A., Romano, J. M., and Karoly, P. (1991). Coping with chronic pain: a critical review of the literature. *Pain*, 47(3):249–283.

Jensen, M. P., Turner, J. A., Romano, J. M., and Lawler, B. K. (1994). Relationship of pain-specific beliefs to chronic pain adjustment. *Pain*, 57(3):301–309.

Johnson-Laird, P. N. and Oatley, K. J. (1992). Basic emotions, rationality, and folk theory. *Cognition and Emotion*, 6:201223.

Jones, A., Spindler, H., Jrgensen, M. M., and Zachariae, R. (2002). The effect of situation-evoked anxiety and gender on pain report using the cold pressor test. *Scand J Psychol*, 43(4):307–313.

Jones, A. and Zachariae, R. (2002). Gender, anxiety, and experimental pain sensitivity: an overview. *J Am Med Womens Assoc*, 57(2):91–94.

Karoly, P., Okun, M. A., Ruehlman, L. S., and Pugliese, J. A. (2008). The impact of goal cognition and pain severity on disability and depression in adults with chronic pain: An examination of direct effects and mediated effects via pain-induced fear. *Cognitive Therapy and Research*, 32:418–433.

Kashdan, T. B., Barrios, V., Forsyth, J. P., and Steger, M. F. (2006). Experiential avoidance as a generalized psychological vulnerability: comparisons with coping and emotion regulation strategies. *Behav Res Ther*, 44(9):1301–1320.

Keefe, FJ; Williams, D. and Smith, S. (2001). Assessment of pain behaviors. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 10, pages 170–187. New York: Guilford Press, 2nd edition.

Keefe, F. J. (1982). Behavioral assessment and treatment of chronic pain: current status and future directions. *J Consult Clin Psychol*, 50(6):896–911.

Keefe, F. J., Dunsmore, J., and Burnett, R. (1992). Behavioral and cognitive-behavioral approaches to chronic pain: recent advances and future directions. *J Consult Clin Psychol*, 60(4):528–536.

Keefe, F. J., Lumley, M., Anderson, T., Lynch, T., Studts, J. L., and Carson, K. L. (2001). Pain and emotion: new research directions. *J Clin Psychol*, 57(4):587–607.

Keefe, F. J., Rumble, M. E., Scipio, C. D., Giordano, L. A., and Perri, L. M. (2004). Psychological aspects of persistent pain: current state of the science. *J Pain*, 5(4):195–211.

Keltner, D., Ekman, P., Gonzaga, G., and Beer, J. (2003). Facial expression of emotion. In Davidson, R. J., Scherer, K. R., and Goldsmith, H. H., editors, *Handbook of Affective Sciences*, pages 415–431. Oxford: Oxford University Press.

Kenntner-Mabiala, R., Weyers, P., and Pauli, P. (2007). Independent effects of emotion and attention on sensory and affective pain perception. *Cognition and Emotion*, 21:1615–1629.

Keogh, E. and Birkby, J. (1999). The effect of anxiety sensitivity and gender on the experience of pain. *Cognition and Emotion*, 13(6):813–829.

Keogh, E., Bond, F. W., Hanmer, R., and Tilston, J. (2005). Comparing acceptance- and control-based coping instructions on the cold-pressor pain experiences of healthy men and women. *Eur J Pain*, 9(5):591–598.

Keogh, E. and Cochrane, M. (2002). Anxiety sensitivity, cognitive biases, and the experience of pain. *J Pain*, 3(4):320–329.

Keogh, E., McCracken, L. M., and Eccleston, C. (2006). Gender moderates the association between depression and disability in chronic pain patients. *Eur J Pain*, 10(5):413–422.

Kerns, R. D., Turk, D. C., and Rudy, T. E. (1985). The West Haven-Yale Multidimensional Pain Inventory (WHYMPI). *Pain*, 23(4):345–356.

Kerns, R. D., Wagner, J., Rosenberg, R., Haythornthwaite, J., and Caudill-Slosberg, M. (2005). Identification of subgroups of persons with chronic pain based on profiles on the pain stages of change questionnaire. *Pain*, 116(3):302–310.

Kerr, S., Fairbrother, G., Crawford, M., Hogg, M., Fairbrother, D., and Khor, K. E. (2004). Patient characteristics and quality of life among a sample of Australian chronic pain clinic attendees. *Intern Med J*, 34(7):403–409.

Kingston, J., Chadwick, P., Meron, D., and Skinner, T. C. (2007). A pilot randomized control trial investigating the effect of mindfulness practice on pain tolerance, psychological well-being, and physiological activity. *J Psychosom Res*, 62(3):297–300.

Kirwilliam, S. S. and Derbyshire, S. W. G. (2008). Increased bias to report heat or pain following emotional priming of pain-related fear. *Pain*, 137(1):60–65.

Kline, R. B. (2004). *Principles and Practice of Structural Equation Modeling*. New York (NY): Guildford, 2nd edition.

Kollman, D. M., Brown, T. A., and Barlow, D. H. (2009). The construct validity of acceptance: A multitrait-multimethod investigation. *Behavior Therapy*, 40(3):205–218.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion*, 23(1):4–41.

Kratz, A. L., Davis, M. C., and Zautra, A. J. (2007). Pain acceptance moderates the relation between pain and negative affect in female osteoarthritis and fibromyalgia patients. *Ann Behav Med*, 33(3):291–301.

Kruger, L., editor (2001). *Methods in Pain Research*. Boca Raton, FL: CRC Press.

Kuch, K., Cox, B. J., Woszczyna, C. B., Swinson, R. P., and Shulman, I. (1991). Chronic pain in panic disorder. *J Behav Ther Exp Psychiatry*, 22(4):255–259.

Lang, P. J. (1995). The emotion probe. studies of motivation and attention. *Am Psychol*, 50(5):372–385.

Larsen, D. K., Taylor, S., and Asmundson, G. J. (1997). Exploratory factor analysis of the pain anxiety symptoms scale in patients with chronic pain complaints. *Pain*, 69(1-2):27–34.

Lau-Walker, M. (2006). Predicting self-efficacy using illness perception components: A patient survey. *British Journal of Health Psychology*, 11(4):643–661.

Lautenbacher, S. and Spernal, J. (2004). Pain perception in psychiatric disorders. In Lautenbacher, S. and Fillingim, R. B., editors, *Pathophysiology of Pain Perception*, chapter 12, pages 163–183. New York: Springer.

Law, G. U. (2002). Dissimilarity in adolescent and maternal representations of type 1 diabetes: exploration of relations to adolescent well-being. *Child Care Health Dev*, 28(5):369–378.

Lawson, K., Reesor, K. A., Keefe, F. J., and Turner, J. A. (1990). Dimensions of pain-related cognitive coping: cross-validation of the factor structure of the coping strategy questionnaire. *Pain*, 43(2):195–204.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annu Rev Neurosci*, 23:155–184.

Leeuw, M., Goossens, M. E. J. B., Linton, S. J., Crombez, G., Boersma, K., and Vlaeyen, J. W. S. (2007). The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *J Behav Med*, 30(1):77–94.

LeResche, L. (2001). Gender, cultural, and environmental aspects of pain. In Loeser, J. D., Turk, D., Chapman, C. R., and Butler, S., editors, *Bonica's management of pain*, chapter 7, pages 191–195. Philadelphia : Lippincott Williams & Wilkins, 3rd edition.

Lethem, J., Slade, P. D., Troup, J. D., and Bentley, G. (1983). Outline of a fear-avoidance model of exaggerated pain perception–I. *Behav Res Ther*, 21(4):401–408.

Levenson, R. W. (1994). Human emotions: A functional view. In Ekman, P. and Davidson, R., editors, *The nature of emotion: Fundamental questions*, pages 123–126. New York: Oxford University Press.

Levenson, R. W. (2003). Autonomic specificity and emotion. In R. J. Davidson, K. R. S. and Goldsmith, H. H., editors, *Handbook of affective sciences*, chapter 11, pages 212–224. New York: Oxford University Press.

Leventhal, H. (1993). The pain system: A multilevel model for the study of motivation and emotion. *Motivation and Emotion*, 17(3):139–146.

Leventhal, H. (2008). Integration of emotion with illness cognitions. *Emotion Researcher*, 23(1):9–12.

Leventhal, H., Benyamini, Y., Brownlee, S., Diefenbach, M., Leventhal, E. A., Patrick-Miller, L., and Robitaille, C. (1997). Illness representations: Theoretical foundations. In Petrie, K. J. and Weinman, J. A., editors, *Perceptions of health and illness*, chapter 1, pages 19–45. Amsterdam: Harwood Academic Publishers.

Leventhal, H., Diefenbach, M., and Leventhal, E. (1992). Illness cognition: Using common sense to understand treatment adherence and affect cognition interactions. *Cognitive Therapy and Research*, 16(2):143–163.

Leventhal, H. and Everhart, D. (1979). Emotion, pain and physical illness. In Izard, C., editor, *Emotions in personality and psychopathology*, chapter 9, pages 263–299. New York: Plenum.

Leventhal, H. and Mosbach, P. A. (1983). The perceptual-motor theory of emotion. In Cacioppo, J. T. and Petty, R. E., editors, *Social psychophysiology*, chapter 12, pages 353–388. New York (NY): Guildford.

Leventhal, H. and Scherer, K. R. (1987). The relationship of emotion to cognition: A functional approach to a semantic controversy. *Cognition and Emotion*, 1(1):3–28.

Leventhal, H., Weinman, J., Leventhal, E. A., and Phillips, L. A. (2008). Health psychology: the search for pathways between behavior and health. *Annu Rev Psychol*, 59:477–505.

Leyland, A. H. (2006). A review of multilevel modelling in SPSS. Technical report, Centre for Multilevel Modelling Software Reviews of Multilevel Analysis Packages.

Lillis, J. and Hayes, S. C. (2007). Measuring avoidance and inflexibility in weight related problems. *International Journal of Behavior Consultation and Therapy*, 4:30–40.

Litcher-Kelly, L., Stone, A. A., Broderick, J. E., and Schwartz, J. E. (2004). Associations among pain intensity, sensory characteristics, affective qualities, and activity limitations in patients with chronic pain: a momentary, within-person perspective. *J Pain*, 5(8):433–439.

Little, T. D., Cunningham, W. A., and Shahar, G. (2002). To parcel or not to parcel: exploring the question, weighing the merits. *Structural Equation Modeling*, 9(2):151173.

Lumley, M. A., Asselin, L. A., and Norman, S. (1997). Alexithymia in chronic pain patients. *Compr Psychiatry*, 38(3):160–165.

Lumley, M. A., Gustavson, B. J., Partridge, R. T., and Labouvie-Vief, G. (2005). Assessing alexithymia and related emotional ability constructs using multiple methods: interrelationships among measures. *Emotion*, 5(3):329–342.

Lumley, M. A., Smith, J. A., and Longo, D. J. (2002). The relationship of alexithymia to pain severity and impairment among patients with chronic myofascial pain: comparisons with self-efficacy, catastrophizing, and depression. *J Psychosom Res*, 53(3):823–830.

Lundgren, T., Dahl, J., and Hayes, S. C. (2008). Evaluation of mediators of change in the treatment of epilepsy with acceptance and commitment therapy. *J Behav Med*, 31(3):225–235.

Lundh, L. (2005). The role of acceptance and mindfulness in the treatment of insomnia. *Journal of Cognitive Psychotherapy*, 19:29–39.

Luoma, J. B., Kohlenberg, B. S., Hayes, S. C., Bunting, K., and Rye, A. K. (2008). Reducing self-stigma in substance abuse through acceptance and commitment therapy: Model, manual development, and pilot outcomes. *Addiction Research and Theory*, 16:149–165.

MacCallum, R. C. and Austin, J. T. (2000). Applications of structural equation modeling in psychological research. *Annu Rev Psychol*, 51:201–226.

Macfarlane, G. J. (2008). Changing patient perceptions of their illness: can they contribute to an improved outcome for episodes of musculoskeletal pain? *Pain*, 136(1-2):1–2.

Magni, G., Moreschi, C., Rigatti-Luchini, S., and Merskey, H. (1994). Prospective study on the relationship between depressive symptoms and chronic musculoskeletal pain. *Pain*, 56(3):289–297.

Maniadakis, N. and Gray, A. (2000). The economic burden of back pain in the UK. *Pain*, 84(1):95–103.

Markland, D. (2007). The golden rule is that there are no golden rules: A commentary on paul barretts recommendations for reporting model fit in structural equation modelling. *Personality and Individual Differences*, 42(5):851–858.

Marsh, H. W. (1996). Positive and negative global self-esteem: a substantively meaningful distinction or artifactors? *J Pers Soc Psychol*, 70(4):810–819.

Marsh, H. W., Hau, K.-T., and Wen, Z. (2004). In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) findings. *Structural Equation Modeling*, 11(3):320 341.

Maruyama, G. (1998). *Basics of Structural Equation Modeling*. Thousand Oaks CA: Sage.

Masedo, A. I. and Esteve, M. R. (2007). Effects of suppression, acceptance and spontaneous coping on pain tolerance, pain intensity and distress. *Behav Res Ther*, 45(2):199–209.

Materazzo, F., Cathcart, S., and Pritchard, D. (2000). Anger, depression, and coping interactions in headache activity and adjustment: a controlled study. *J Psychosom Res*, 49(1):69–75.

Mauss, I. B. and Gross, J. J. (2004). Emotional expression andhealth: Advances in theory, assessment, and clinical applications. In Nyklicek, I., Temoshok, L. R., and Vingerhoets, A.,

editors, *Emotion suppression and cardiovascular disease: Is hiding your feelings bad for your heart?*, page 6281. New York: Brunner-Routledge.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., and Gross, J. J. (2005). The tie that binds? coherence among emotion experience, behavior, and physiology. *Emotion*, 5(2):175–190.

Maxwell, T. D., Gatchel, R. J., and Mayer, T. G. (1998). Cognitive predictors of depression in chronic low back pain: toward an inclusive model. *J Behav Med*, 21(2):131–143.

McConnell, S., Kolopack, P., and Davis, A. M. (2001). The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC): a review of its utility and measurement properties. *Arthritis Rheum*, 45(5):453–461.

McCracken, L. M. (1998). Learning to live with the pain: acceptance of pain predicts adjustment in persons with chronic pain. *Pain*, 74(1):21–27.

McCracken, L. M. (1999). Behavioral constituents of chronic pain acceptance: Results from factor analysis of the Chronic Pain Acceptance Questionnaire. *Journal of Back & Musculoskeletal Rehabilitation*, 13:93–100.

McCracken, L. M. (2005). Social context and acceptance of chronic pain: the role of solicitous and punishing responses. *Pain*, 113(1-2):155–159.

McCracken, L. M., Carson, J. W., Eccleston, C., and Keefe, F. J. (2004a). Acceptance and change in the context of chronic pain. *Pain*, 109(1-2):4–7.

McCracken, L. M. and Eccleston, C. (2003). Coping or acceptance: what to do about chronic pain? *Pain*, 105(1-2):197–204.

McCracken, L. M. and Eccleston, C. (2005). A prospective study of acceptance of pain and patient functioning with chronic pain. *Pain*, 118(1-2):164–169.

McCracken, L. M. and Eccleston, C. (2006). A comparison of the relative utility of coping and acceptance-based measures in a sample of chronic pain sufferers. *Eur J Pain*, 10(1):23–29.

McCracken, L. M., Faber, S. D., and Janeck, A. S. (1998). Pain-related anxiety predicts non-specific physical complaints in persons with chronic pain. *Behav Res Ther*, 36(6):621–630.

McCracken, L. M. and Gross, R. T. (1993). Does anxiety affect coping with chronic pain? *Clin J Pain*, 9(4):253–259.

McCracken, L. M., Gross, R. T., Aikens, J., and Carnrike, C. L. (1996). The assessment of anxiety and fear in persons with chronic pain: a comparison of instruments. *Behav Res Ther*, 34(11-12):927–933.

McCracken, L. M. and Keogh, E. (2009). Acceptance, mindfulness, and values-based action may counteract fear and avoidance of emotions in chronic pain: an analysis of anxiety sensitivity. *J Pain*, 10(4):408–415.

McCracken, L. M., MacKichan, F., and Eccleston, C. (2007a). Contextual cognitive-behavioral therapy for severely disabled chronic pain sufferers: effectiveness and clinically significant change. *Eur J Pain*, 11(3):314–322.

McCracken, L. M., Spertus, I. L., Janeck, A. S., Sinclair, D., and Wetzel, F. T. (1999). Behavioral dimensions of adjustment in persons with chronic pain: pain-related anxiety and acceptance. *Pain*, 80(1-2):283–289.

McCracken, L. M., Vowles, K., and Thompson, M. (2007b). Comment on Nicholas and Asghari: Pain 2006;124:269-79. *Pain*, 128(3):283–4; author reply 284–5.

McCracken, L. M. and Vowles, K. E. (2007). Psychological flexibility and traditional pain management strategies in relation to patient functioning with chronic pain: an examination of a revised instrument. *J Pain*, 8(9):700–707.

McCracken, L. M. and Vowles, K. E. (2008). A prospective analysis of acceptance of pain and values-based action in patients with chronic pain. *Health Psychol*, 27(2):215–220.

McCracken, L. M., Vowles, K. E., and Eccleston, C. (2004b). Acceptance of chronic pain: component analysis and a revised assessment method. *Pain*, 107(1-2):159–166.

McCracken, L. M., Vowles, K. E., and Eccleston, C. (2005). Acceptance-based treatment for persons with complex, long standing chronic pain: a preliminary analysis of treatment outcome in comparison to a waiting phase. *Behav Res Ther*, 43(10):1335–1346.

McCracken, L. M. and Yang, S.-Y. (2006). The role of values in a contextual cognitive-behavioral approach to chronic pain. *Pain*, 123(1-2):137–145.

McCracken, L. M., Zayfert, C., and Gross, R. T. (1992). The Pain Anxiety Symptoms Scale: development and validation of a scale to measure fear of pain. *Pain*, 50(1):67–73.

McDonald, R. P. and Ho, M.-H. R. (2002). Principles and practice in reporting structural equation analyses. *Psychol Methods*, 7(1):64–82.

McEwan, J. (2004). Scottish Executive Health Department report on Chronic Pain in Scotland, Chronic Pain Services in Scotland. retrieved June 29, 2006 from http://www.show.scot.nhs.uk/sehd/mels/HDL2004_48.pdf.

McIntosh, C. N. (2007). Rethinking fit assessment in structural equation modelling: A commentary and elaboration on Barrett (2007). *Personality and Individual Differences*, 42(5):859–867.

McNeil, D. W. and Rainwater, A. J. (1998). Development of the Fear of Pain Questionnaire–III. *J Behav Med*, 21(4):389–410.

McWilliams, L. A., Cox, B. J., and Enns, M. W. (2003). Mood and anxiety disorders associated with chronic pain: an examination in a nationally representative sample. *Pain*, 106(1-2):127–133.

Meagher, M. W., Arnau, R. C., and Rhudy, J. L. (2001). Pain and emotion: effects of affective picture modulation. *Psychosom Med*, 63(1):79–90.

Means-Christensen, A. J., Roy-Byrne, P. P., Sherbourne, C. D., Craske, M. G., and Stein, M. B. (2008). Relationships among pain, anxiety, and depression in primary care. *Depress Anxiety*, 25(7):593–600.

Meehl, P. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, 46(4):806–834.

Meehl, P. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality*, 60(1):117–174.

Mehling, W. E. and Krause, N. (2005). Are difficulties perceiving and expressing emotions associated with low-back pain? The relationship between lack of emotional awareness (alexithymia) and 12-month prevalence of low-back pain in 1180 urban public transit operators. *J Psychosom Res*, 58(1):73–81.

Mehling, W. E. and Krause, N. (2007). Alexithymia and 7.5-year incidence of compensated low back pain in 1207 urban public transit operators. *J Psychosom Res*, 62(6):667–674.

Meier, S. and Davis, S. (1990). Trends in reporting psychometric properties of scales used in counseling psychology research. *Journal of Counseling Psychology*, 37(1):113–115.

Melzack, R. (1975). The McGill Pain Questionnaire: major properties and scoring methods. *Pain*, 1(3):277–299.

Melzack, R. (1987). The short-form McGill Pain Questionnaire. *Pain*, 30(2):191–7.

Melzack, R. (2001). Pain and the neuromatrix in the brain. *J Dent Educ*, 65(12):1378–1382.

Melzack, R. (2005). The McGill Pain Questionnaire: from description to measurement. *Anesthesiology*, 103(1):199–202.

Melzack, R. and Katz, J. (2001). The McGill Pain Questionnaire: appraisal and current status. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 3, pages 35–52. New York: Guilford Press, 2nd edition.

Melzack, R. and Katz, J. (2004). The Gate Control Theory: Reaching for the Brain. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 1, pages 13–34. Psychology Press.

Melzack, R. and Wall, P. D. (1965). Pain mechanisms: a new theory. *Science*, 150(699):971–979.

Melzack, R. and Wall, P. D. (1970). Psychophysiology of Pain. *Int Anesthesiol Clin*, 8(1):3–34.

Merskey, H. and Bogduk, N. (1994). *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*. Seattle: IASP Press, 2nd edition. Retrieved October, 3, 2008 from `http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Definitions&Template=/CM/HTMLDisplay.cfm&ContentID=1728`.

Millan, M. J. (1999). The induction of pain: an integrative review. *Prog Neurobiol*, 57(1):1–164.

Millard, R. W. and Kinsler, B. L. (1992). Evaluation of constricted affect in chronic pain: an attempt using the Toronto Alexythymia Scale. *Pain*, 50(3):287–292.

Mollet, G. A. and Harrison, D. W. (2006). Emotion and pain: a functional cerebral systems integration. *Neuropsychol Rev*, 16(3):99–121.

Montao, D. E. and Kasprzyk, D. (2002). The theory of reasoned action and the theory of planned behavior. In Glanz, K., Rimer, B. K., and Lewis, F. M., editors, *Health Behavior and Health Education: Theory, Research and Practice*, chapter 4, pages 67–98. California: Jossey-Bass, 3 edition.

Moskowitz, J. (2008). The role of positive emotion in health: Avoiding Pollyanna and Pangloss. *Emotion Researcher*, 23(1):4–7.

Moss-Morris, R., Humphrey, K., Johnson, M. H., and Petrie, K. J. (2007). Patients' perceptions of their pain condition across a multidisciplinary pain management program: do they change and if so does it matter? *Clin J Pain*, 23(7):558–564.

Moss-Morris, R., Weinman, J., Petrie, K., Horne, R., Cameron, L., and Buick, D. (2002). The Revised Illness Perception Questionnaire (IPQ-R). *Psychology and Health*, 17:1–16.

Mossey, J. M. and Gallagher, R. M. (2004). The longitudinal occurrence and impact of comorbid chronic pain and chronic depression over two years in continuing care retirement community residents. *Pain Medicine*, 5(4):335–348.

Mulaik, S. (2009). *Linear Causal Modeling with Structural Equations*. Boca Raton: Chapman & Hall/CRC.

Mulaik, S. A. and Millsap, R. E. (2000). Doing the four-step right. *Structural Equation Modeling: A Multidisciplinary Journal*, 7(1):36–73.

Murphy, F. C., Nimmo-Smith, I., and Lawrence, A. D. (2003). Functional neuroanatomy of emotions: a meta-analysis. *Cogn Affect Behav Neurosci*, 3(3):207–233.

Murphy, H., Dickens, C., Creed, F., and Bernstein, R. (1999). Depression, illness perception and coping in rheumatoid arthritis. *J Psychosom Res*, 46(2):155–164.

Myrtek, M. (2004). *Heart and Emotion: Ambulatory Monitoring Studies in Everyday Life*. Kirkland, WA: Hogrefe & Huber Publishers.

Newman, D. A. (2009). Missing data techniques and low response rates: The role of systematic nonresponse parameters. In Lance, C. E. and Vandenberg, R. J., editors, *Statistical and*

methodological myths and urban legends: Doctrine, verity, and fable in the organizational and social sciences, chapter 1, pages 7–36. New York: Routledge.

Newth, S. and DeLongis, A. (2004). Individual differences, mood and coping with chronic pain in rheumatoid arthritis: A daily process analysis. *Psychology and Health*, 19(3):283–305.

Nicholas, M. K. and Asghari, A. (2006). Investigating acceptance in adjustment to chronic pain: is acceptance broader than we thought? *Pain*, 124(3):269–279.

Norton, P. J. and Asmundson, G. J. G. (2004). Anxiety sensitivity, fear, and avoidance behavior in headache pain. *Pain*, 111(1-2):218–223.

Nunnally, J. and Bernstein, I. (1994). *Psychometric Theory*. New York: McGraw Hill, 3 edition.

Oatley, K. and Johnson-Laird, P. N. (1987). Towards a cognitive theory of emotions. *Cognition & Emotion*, 1:29–50.

Ohayon, M. M. (2004). Specific characteristics of the pain/depression association in the general population. *J Clin Psychiatry*, 65 Suppl 12:5–9.

Okifuji, A. and Turk, D. C. (2001). Assessment of treatment outcomes in clinical practice: A survival guide. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 33, pages 639–658. New York: Guilford Press, 2nd edition.

Okifuji, A., Turk, D. C., and Curran, S. L. (1999). Anger in chronic pain: investigations of anger targets and intensity. *J Psychosom Res*, 47(1):1–12.

Ostafin, B. D. and Marlatt, G. A. (2007). Surfing the urge: Experiential acceptance moderates the relation between automatic alcohol motivation and hazardous drinking. *Journal of Social and Clinical Psychology*, 27(4):404–418.

Page, L. A., Howard, L. M., Husain, K., Tong, J., Dowson, A. J., Weinman, J., and Wessely, S. C. (2004). Psychiatric morbidity and cognitive representations of illness in chronic daily headache. *J Psychosom Res*, 57(6):549–555.

Panksepp, J. (2000). Emotions as natural kinds within the mammalian brain. In Lewis, M. and Haviland-Jones, J. M., editors, *Handbook of emotions*, pages 137–156. New York: Guilford, 2 edition.

Panksepp, J. (2008). The affective brain and core consciousness: how does neural activity generate emotional feelings? In Lewis, M. and Haviland-Jones, J. M., editors, *Handbook of emotions*, chapter 4, pages 47–67. New York: Guilford, 3 edition.

Paquet, C., Kergoat, M.-J., and Dub, L. (2005). The role of everyday emotion regulation on pain in hospitalized elderly: insights from a prospective within-day assessment. *Pain*, 115(3):355–363.

Parsons, S., Carnes, D., Pincus, T., Foster, N., Breen, A., Vogel, S., and Underwood, M. (2006). Measuring troublesomeness of chronic pain by location. *BMC Musculoskelet Disord*, 7:34.

Peng, C.-Y. J., Harwell, M., Liou, S.-M., and Ehman, L. H. (2006). Advances in missing data methods and implications for educational research. In Sawilowsky, S., editor, *Real Data Analysis*, chapter 3, pages pp. 31–78. New York: IAP.

Petrie, K. J., Cameron, L. D., Ellis, C. J., Buick, D., and Weinman, J. (2002). Changing illness perceptions after myocardial infarction: an early intervention randomized controlled trial. *Psychosom Med*, 64(4):580–586.

Petrie, K. J., Frampton, T., Large, R. G., Moss-Morris, R., Johnson, M., and Meechan, G. (2005). What do patients expect from their first visit to a pain clinic? *Clin J Pain*, 21(4):297–301.

Petrie, K. J., Jago, L. A., and Devcich, D. A. (2007). The role of illness perceptions in patients with medical conditions. *Curr Opin Psychiatry*, 20(2):163–167.

Petrie, K. J. and Weinman, J. (2006). Why illness perceptions matter. *Clin Med*, 6(6):536–539.

Petrie, K. J., Weinman, J., Sharpe, N., and Buckley, J. (1996). Role of patients' view of their illness in predicting return to work and functioning after myocardial infarction: longitudinal study. *BMJ*, 312(7040):1191–1194.

Peugh, J. L. and Enders, C. K. (2005). Using the SPSS mixed procedure to fit cross-sectional and longitudinal multilevel models. *Educational and Psychological Measurement*, 65:714–741.

Philippot, P. and Rime, B. (1997). The perception of bodily sensation during emotion: A cross-cultural perspective. *Polish Bulletin of Psychology*, 28:175–188.

Phillips, K. and Power, M. (2007). A new self-report measure of emotion regulation in adolescents: The regulation of emotions questionnaire. *Clinical Psychology & Psychotherapy*, 14(2):145 – 156.

Pimm, T. J. and Weinman, J. (1998). Applying Leventhal's self regulation model to adaptation and intervention in rheumatic disease. *Clinical Psychology and Psychotherapy*, 5(2):62–75.

Pincus, T., Vogel, S., Burton, A. K., Santos, R., and Field, A. P. (2006). Fear avoidance and prognosis in back pain: a systematic review and synthesis of current evidence. *Arthritis Rheum*, 54(12):3999–4010.

Pincus, T. and Williams, A. (1999). Models and measurements of depression in chronic pain. *J Psychosom Res*, 47(3):211–219.

PingJr, R. A. (2003). On assuring valid measures for theoretical models using survey data. *Journal of Business Research*, 57(2):125–141.

Plehn, K., Peterson, R. A., and Williams, D. A. (1998). Anxiety sensitivity: Its relationship to functional status in patients with chronic pain. *Journal of Occupational Rehabilitation*, 8(3):213–222.

Podsakoff, P. M., MacKenzie, S. B., Lee, J.-Y., and Podsakoff, N. P. (2003). Common method biases in behavioral research: a critical review of the literature and recommended remedies. *J Appl Psychol*, 88(5):879–903.

Poleshuck, E. L., Bair, M. J., Kroenke, K., Damush, T. M., Tu, W., Wu, J., Krebs, E. E., and Giles, D. E. (2009). Psychosocial stress and anxiety in musculoskeletal pain patients with and without depression. *Gen Hosp Psychiatry*, 31(2):116–122.

Porter-Moffitt, S., Gatchel, R. J., Robinson, R. C., Deschner, M., Posamentier, M., Polatin, P., and Lou, L. (2006). Biopsychosocial profiles of different pain diagnostic groups. *J Pain*, 7(5):308–318.

Power, M. J. (2006). The structure of emotion: An empirical comparison of six models. *Cognition & Emotion*, 20:694–713.

Power, M. J. (2008). Emotion regulation: "out, damned spot, out!". *Emotion Researcher*, 23(2):11–12.

Power, M. J. and Dalgleish, T. (2008). *Cognition and Emotion: from Order to Disorder*. Hove: Psychology Press, 2 edition.

Price, D. D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science*, 288(5472):1769–1772.

Price, D. D. (2002). Central neural mechanisms that interrelate sensory and affective dimensions of pain. *Mol Interv*, 2(6):392–403, 339.

Price, D. D. and Harkins, S. W. (1992). The affective-motivational dimension of pain a two-stage model. *APS Journal*, 1(4):229–239.

Price, D. D., Riley, J. L., and Wade, J. B. (2001). Psychophysical approaches to the measurement of the dimensions and stages of pain. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 4, pages 53–75. New York: Guilford Press, 2nd edition.

Quartana, P. J. and Burns, J. W. (2007). Painful consequences of anger suppression. *Emotion*, 7(2):400–414.

Quartana, P. J., Yoon, K. L., and Burns, J. W. (2007). Anger suppression, ironic processes and pain. *J Behav Med*, 30(6):455–469.

Radat, F., Lantri-Minet, M., Nachit-Ouinekh, F., Massiou, H., Lucas, C., Pradalier, A., Mercier, F., and Hasnaoui, A. E. (2009). The GRIM2005 study of migraine consultation in France. III: Psychological features of subjects with migraine. *Cephalalgia*, 29(3):338–350.

Reynolds, L., Broadbent, E., Ellis, C. J., Gamble, G., and Petrie, K. J. (2007). Patients' drawings illustrate psychological and functional status in heart failure. *J Psychosom Res*, 63(5):525–532.

Risdon, A., Eccleston, C., Crombez, G., and McCracken, L. (2003). How can we learn to live with pain? a Q-methodological analysis of the diverse understandings of acceptance of chronic pain. *Soc Sci Med*, 56(2):375–386.

Roche, B., Forsyth, J. P., and Maher, E. (2007). The impact of demand characteristics on brief acceptance- and control-based interventions for pain tolerance. *Cognitive and Behavioral Practice*, 14:381–393.

Roelofs, J., Peters, M. L., Patijn, J., Schouten, E. G. W., and Vlaeyen, J. W. S. (2004). Electronic diary assessment of pain-related fear, attention to pain, and pain intensity in chronic low back pain patients. *Pain*, 112(3):335–342.

Roland, M. and Morris, R. (1983). A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. *Spine*, 8(2):141–144.

Rollman, G. B. (2004). Ethnocultural variations in the experience of pain. In Hadjistavropoulos, T. and Craig, K. D., editors, *Pain: Psychological Perspectives*, chapter 6, pages 155–178. Psychology Press.

Romano, J. M. and Schmaling, K. B. (2001). Assessment of couples and families with chronic pain. In Turk, D. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 18, pages 346–361. New York: Guilford Press, 2nd edition.

Rosenberg, E. I., Genao, I., Chen, I., Mechaber, A. J., Wood, J. A., Faselis, C. J., Kurz, J., Menon, M., O'Rorke, J., Panda, M., Pasanen, M., Staton, L., Calleson, D., and Cykert, S. (2008). Complementary and alternative medicine use by primary care patients with chronic pain. *Pain Med*, 9(8):1065–1072.

Rosenstiel, A. K. and Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. *Pain*, 17(1):33–44.

Rudy, T. E., Kerns, R. D., and Turk, D. C. (1988). Chronic pain and depression: toward a cognitive-behavioral mediation model. *Pain*, 35(2):129–140.

Rudy, T. E., Turk, D. C., Zaki, H. S., and Curtin, H. D. (1989). An empirical taxometric alternative to traditional classification of temporomandibular disorders. *Pain*, 36(3):311–320.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychol Rev*, 110(1):145–172.

Ryan, R. M. and Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *Am Psychol*, 55(1):68–78.

Sagula, D. and Rice, K. (2004). The effectiveness of mindfulness training on the grieving process and emotional well-being of chronic pain patients. *Journal of Clinical Psychology in Medical Settings*, 11(4):333–342.

Salewski, C. (2003). Illness representations in families with a chronically ill adolescent: differences between family members and impact on patients' outcome variables. *J Health Psychol*, 8(5):587–598.

Sangha, O., Stucki, G., Liang, M. H., Fossel, A. H., and Katz, J. N. (2003). The self-administered comorbidity questionnaire: a new method to assess comorbidity for clinical and health services research. *Arthritis Rheum*, 49(2):156–63.

Sankey, H. (2008). Scientific method. In Psillos, S. and Curd, M., editors, *The Routledge Companion To Philosophy of Science*, pages 248–258. NY: Routledge.

Sayar, K., Gulec, H., and Topbas, M. (2004). Alexithymia and anger in patients with fibromyalgia. *Clin Rheumatol*, 23(5):441–448.

Scascighini, L., Toma, V., Dober-Spielmann, S., and Sprott, H. (2008). Multidisciplinary treatment for chronic pain: a systematic review of interventions and outcomes. *Rheumatology (Oxford)*, 47(5):670–678.

Scharloo, M., Kaptein, A. A., Weinman, J., Bergman, W., Vermeer, B. J., and Rooijmans, H. G. (2000). Patients' illness perceptions and coping as predictors of functional status in psoriasis: a 1-year follow-up. *Br J Dermatol*, 142(5):899–907.

Scharloo, M., Kaptein, A. A., Weinman, J., Hazes, J. M., Willems, L. N., Bergman, W., and Rooijmans, H. G. (1998). Illness perceptions, coping and functioning in patients with rheumatoid arthritis, chronic obstructive pulmonary disease and psoriasis. *J Psychosom Res*, 44(5):573–585.

Scherer, K. R. and Wallbott, H. G. (1994). Evidence for universality and cultural variation of differential emotion response patterning. *J Pers Soc Psychol*, 66(2):310–328.

Schiaffino, K. M., Shawaryn, M. A., and Blum, D. (1998). Examining the impact of illness representations on psychological adjustment to chronic illnesses. *Health Psychol*, 17(3):262–268.

Schmidt, N. B. and Cook, J. H. (1999). Effects of anxiety sensitivity on anxiety and pain during a cold pressor challenge in patients with panic disorder. *Behav Res Ther*, 37(4):313–323.

Schreiber, J. B., Nora, A., Stage, F. K., Barlow, E. A., and King, J. (2006). Reporting structural equation modeling and confirmatory factor analysis results: a review. *Journal of Educational Research*, 99(6):323–337.

Schumacker, R. E. and Lomax, R. G. (2004). *A Beginner's Guide to Structural Equation Modeling*. Mahwah, NJ: Lawrence Earlbaum Associates.

Senior, V., Marteau, T. M., Weinman, J., and the Genetic Risk Assessment for FH Trial (GRAFT) Study Group (2004). Self-reported adherence to cholesterol-lowering medication in patients with familial hypercholesterolaemia: the role of illness perceptions. *Cardiovasc Drugs Ther*, 18(6):475–481.

Sieben, J. M., Vlaeyen, J. W. S., Portegijs, P. J. M., Verbunt, J. A., van Riet-Rutgers, S., Kester, A. D. M., Korff, M. V., Arntz, A., and Knottnerus, J. A. (2005). A longitudinal study on the predictive validity of the fear-avoidance model in low back pain. *Pain*, 117(1-2):162–170.

Singer, J. D. and Willett, J. B. (2003). *Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence*. Oxford University Press.

Singer, T. and Frith, C. (2005). The painful side of empathy. *Nat Neurosci*, 8(7):845–846.

Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R. J., and Frith, C. D. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science*, 303(5661):1157–1162.

Skevington, S. M. (1995). *Psychology of Pain*. Chichester: John Wiley and Sons.

Sloan, D. M. (2004). Emotion regulation in action: emotional reactivity in experiential avoidance. *Behav Res Ther*, 42(11):1257–1270.

Smith, B. H., Penny, K. I., Purves, A. M., Munro, C., Wilson, B., Grimshaw, J., Chambers, W. A., and Smith, W. C. (1997). The chronic pain grade questionnaire: validation and reliability in postal research. *Pain*, 71(2):141–147.

Smith, B. W. and Zautra, A. J. (2008). The effects of anxiety and depression on weekly pain in women with arthritis. *Pain*, 138(2):354–361.

Smith, G. and McCarthy, D. (1995). Methodological considerations in the refinement of clinical assessment instruments. *Psychological Assessment*, 7(3):300–308.

Strand, E. B., Kerns, R. D., Christie, A., Haavik-Nilsen, K., Klokkerud, M., and Finset, A. (2007). Higher levels of pain readiness to change and more positive affect reduce pain reports–a weekly assessment study on arthritis patients. *Pain*, 127(3):204–213.

Strand, E. B., Zautra, A. J., Thoresen, M., degrd, S., Uhlig, T., and Finset, A. (2006). Positive affect as a factor of resilience in the pain-negative affect relationship in patients with rheumatoid arthritis. *J Psychosom Res*, 60(5):477–484.

Streiner, D. L. (2003). Starting at the beginning: an introduction to coefficient alpha and internal consistency. *J Pers Assess*, 80(1):99–103.

Stroud, M. W., McKnight, P. E., and Jensen, M. P. (2004). Assessment of self-reported physical activity in patients with chronic pain: development of an abbreviated Roland-Morris disability scale. *J Pain*, 5(5):257–263.

Suchdev, P. (2002). Pathophysiology of pain. In Warfield, CA; Fausett, H., editor, *Manual of Pain Management*, pages 6–12. Philadelphia: Lippincott Williams and Wilkins, 2nd edition.

Sufka, K. and Turner, D. (2005). An evolutionary account of chronic pain: integrating the natural method in evolutionary psychology. *Philosophical Psychology*, 18:243–257.

Sullivan, M. D. (2001). Assessment of psychiatric disorders. In DC, T. and R, M., editors, *Handbook of Pain Assessment*, chapter 15, pages 275–291. New York: Guilford Press, 2nd edition.

Tabachnick, B. G. and Fidell, L. S. (2001). *Using Multivariate Statistics*. Allyn and Bacon.

Tan, G., Jensen, M., Thornby, J., and Sloan, P. A. (2008). Negative emotions, pain, and functioning. *Psychological Services*, 5:26–35.

Thompson, R. A. (1994). Emotion regulation: a theme in search of definition. *Monogr Soc Res Child Dev*, 59(2-3):25–52.

Tomarken, A. J. and Waller, N. G. (2003). Potential problems with "well fitting" models. *J Abnorm Psychol*, 112(4):578–598.

Tourangeau, R., Rips, L., and Rasinski, K. (2000). *The psychology of survey response*. Cambridge, UK: Cambridge University Press.

Turk, D. C. and Dworkin, R. H. (2004). What should be the core outcomes in chronic pain clinical trials? *Arthritis Res Ther*, 6(4):151–4.

Turk, D. C., Meichenbaum, D., and Genest, M. (1983). *Pain and behavioral medicine: A cognitive-behavioral perspective*. New York: Guilford Press.

Turk, D. C. and Okifuji, A. (1997). Evaluating the role of physical, operant, cognitive, and affective factors in the pain behaviors of chronic pain patients. *Behav Modif*, 21(3):259–280.

Turk, D. C. and Okifuji, A. (2001). Matching treatment to assessment of patients with chronic pain. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 21, pages 400–414. New York: Guilford Press, 2nd edition.

Turk, D. C., Okifuji, A., and Scharff, L. (1995). Chronic pain and depression: role of perceived impact and perceived control in different age cohorts. *Pain*, 61(1):93–101.

Turk, D. C. and Rudy, T. E. (1988). Toward an empirically derived taxonomy of chronic pain patients: integration of psychological assessment data. *J Consult Clin Psychol*, 56(2):233–238.

Turk, D. C. and Rudy, T. E. (1990). The robustness of an empirically derived taxonomy of chronic pain patients. *Pain*, 43(1):27–35.

Turner, J. A. and Clancy, S. (1988). Comparison of operant behavioral and cognitive-behavioral group treatment for chronic low back pain. *J Consult Clin Psychol*, 56(2):261–266.

Ullman, J. B. (2001). Structural equation modeling. In Tabachnick, B. G. and Fidell, L. S., editors, *Using Multivariate Statistics*, chapter 14. Allyn and Bacon.

Ulrich, C. M., Wallen, G. R., Feister, A., and Grady, C. (2005). Respondent burden in clinical research: when are we asking too much of subjects? *IRB*, 27(4):17–20.

Viane, I., Crombez, G., Eccleston, C., Devulder, J., and De Corte, W. (2004). Acceptance of the unpleasant reality of chronic pain: effects upon attention to pain and engagement with daily activities. *Pain*, 112(3):282–8.

Viane, I., Crombez, G., Eccleston, C., Poppe, C., Devulder, J., Houdenhove, B. V., and Corte, W. D. (2003). Acceptance of pain is an independent predictor of mental well-being in patients with chronic pain: empirical evidence and reappraisal. *Pain*, 106(1-2):65–72.

Vlaeyen, J. W. and Crombez, G. (1999). Fear of movement/(re)injury, avoidance and pain disability in chronic low back pain patients. *Man Ther*, 4(4):187–195.

Vlaeyen, J. W., Kole-Snijders, A. M., Boeren, R. G., and 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.

Vlieger, P. D., Crombez, G., and Eccleston, C. (2006). Worrying about chronic pain. an examination of worry and problem solving in adults who identify as chronic pain sufferers. *Pain*, 120(1-2):138–144.

Von Korff, M. (2001). Epidemiological and survey methods: Assessment of chronic pain. In Turk, D. C. and Melzack, R., editors, *Handbook of Pain Assessment*, chapter 31, pages 603–618. New York: Guilford Press, 2nd edition.

Von Korff, M. and Dunn, K. M. (2008). Chronic pain reconsidered. *Pain*, 138(2):267–276.

Von Korff, M. and Miglioretti, D. L. (2005). A prognostic approach to defining chronic pain. *Pain*, 117(3):304–13.

Von Korff, M., Ormel, J., Keefe, F. J., and Dworkin, S. F. (1992). Grading the severity of chronic pain. *Pain*, 50(2):133–149.

Vowles, K. E., McCracken, L. M., and Eccleston, C. (2007a). Processes of change in treatment for chronic pain: the contributions of pain, acceptance, and catastrophizing. *Eur J Pain*, 11(7):779–787.

Vowles, K. E., McCracken, L. M., and Eccleston, C. (2008a). Patient functioning and catastrophizing in chronic pain: the mediating effects of acceptance. *Health Psychol*, 27(2 Suppl):S136–S143.

Vowles, K. E., McCracken, L. M., McLeod, C., and Eccleston, C. (2008b). The Chronic Pain Acceptance Questionnaire: confirmatory factor analysis and identification of patient subgroups. *Pain*, 140(2):284–291.

Vowles, K. E., McNeil, D. W., Gross, R. T., McDaniel, M. L., Mouse, A., Bates, M., Gallimore, P., and McCall, C. (2007b). Effects of pain acceptance and pain control strategies on physical impairment in individuals with chronic low back pain. *Behav Ther*, 38(4):412–425.

Vowles, K. E., Wetherell, J. L., and Sorrell, J. T. (2009). Targeting acceptance, mindfulness, and values-based action in chronic pain: Findings of two preliminary trials of an outpatient group-based intervention. *Cognitive and Behavioral Practice*, 16(1):49–58.

Vowles, K. E., Zvolensky, M. J., Gross, R. T., and Sperry, J. A. (2004). Pain-related anxiety in the prediction of chronic low-back pain distress. *J Behav Med*, 27(1):77–89.

Waddell, G., Newton, M., Henderson, I., Somerville, D., and Main, C. J. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*, 52(2):157–168.

Wade, J. B., Dougherty, L. M., Archer, C. R., and Price, D. D. (1996). Assessing the stages of pain processing: a multivariate analytical approach. *Pain*, 68(1):157–167.

Wade, J. B., Price, D. D., Hamer, R. M., Schwartz, S. M., and Hart, R. P. (1990). An emotional component analysis of chronic pain. *Pain*, 40(3):303–310.

Wagner, G., Koschke, M., Leuf, T., Schlsser, R., and Br, K.-J. (2009). Reduced heat pain thresholds after sad-mood induction are associated with changes in thalamic activity. *Neuropsychologia*, 47(4):980–987.

Walker, J. G., Jackson, H. J., and Littlejohn, G. O. (2004). Models of adjustment to chronic illness: Using the example of rheumatoid arthritis. *Clinical Psychology Review*, 24(4):461–488.

Watson, D. and Tellegen, A. (1985). Toward a consensual structure of mood. *Psychol Bull*, 98(2):219–235.

Watson, D., Wiese, D., Vaidya, J., and Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology*, 76(5):820–838.

Weijters, B., Geuens, M., and Schillewaert, N. (2009). The proximity effect: the role of inter-item distance on reverse-item bias. *International Journal of Research in Marketing*, 26(1):2 –12.

Weiner, B. (1985). An attributional theory of achievement motivation and emotion. *Psychol Rev*, 92(4):548–573.

Weinman, J., Petrie, K., Moss-Morris, R., and Horne, R. (1996). The Illness Perception Questionnaire: A new method for assessing illness perceptions. *Psychology and Health*, 11:431–446.

Weinman, J. and Petrie, K. J. (1997). Illness perceptions: a new paradigm for psychosomatics? *J Psychosom Res*, 42(2):113–6.

Wenzel, L., Glanz, K., and Lerman, C. (2002). Stress, coping, and health behavior. In Glanz, K., Rimer, B. K., and Lewis, F. M., editors, *Health Behavior and Health Education: Theory, Research and Practice*, chapter 10, pages 210–239. California: Jossey-Bass, 3 edition.

Werner, A., Isaksen, L. W., and Malterud, K. (2004). 'I am not the kind of woman who complains of everything': Illness stories on self and shame in women with chronic pain. *Social Science & Medicine*, 59:1035–1045.

Wicksell, R. K., Ahlqvist, J., Bring, A., Melin, L., and Olsson, G. L. (2008a). Can exposure and acceptance strategies improve functioning and life satisfaction in people with chronic pain

and whiplash-associated disorders (WAD)? a randomized controlled trial. *Cogn Behav Ther*, 37(3):169–182.

Wicksell, R. K., Melin, L., Lekander, M., and Olsson, G. L. (2009). Evaluating the effectiveness of exposure and acceptance strategies to improve functioning and quality of life in longstanding pediatric pain–a randomized controlled trial. *Pain*, 141(3):248–257.

Wicksell, R. K., Melin, L., and Olsson, G. L. (2007). Exposure and acceptance in the rehabilitation of adolescents with idiopathic chronic pain - a pilot study. *Eur J Pain*, 11(3):267–274.

Wicksell, R. K., Olsson, G. L., and Melin, L. (2008b). The Chronic Pain Acceptance Questionnaire (CPAQ)-further validation including a confirmatory factor analysis and a comparison with the Tampa Scale of Kinesiophobia. *Eur J Pain*.

Wicksell, R. K., Renflt, J., Olsson, G. L., Bond, F. W., and Melin, L. (2008c). Avoidance and cognitive fusion–central components in pain related disability? Development and preliminary validation of the Psychological Inflexibility in Pain Scale (PIPS). *Eur J Pain*, 12(4):491–500.

Wilhelm, F. H., Pfaltz, M. C., and Grossman, P. (2006). Continuous electronic data capture of physiology, behavior and experience in real life: towards ecological momentary assessment of emotion. *Interacting with Computers*, 18(2):171–186.

Wilkinson, L. and the Task Force on Statistical Inference (1999). Statistical methods in psychology journals: Guidelines and explanations. *American Psychologist*, 54(8):594–604.

Wilson, M. W., Richards, J. S., Klapow, J. C., DeVivo, M. J., and Greene, P. (2005). Cluster analysis and chronic pain: an empirical classification of pain subgroups in a spinal cord injury sample. *Rehabilitation Psychology*, 50(4):381–8.

Woby, S. R., Watson, P. J., Roach, N. K., and Urmston, M. (2004). Adjustment to chronic low back pain–the relative influence of fear-avoidance beliefs, catastrophizing, and appraisals of control. *Behav Res Ther*, 42(7):761–774.

Wright, K. D., Asmundson, G. J., and McCreary, D. R. (2001). Factorial validity of the short-form McGill pain questionnaire (SF-MPQ). *Eur J Pain*, 5(3):279–284.

Yuan, K. H. and Bentler, P. M. (2004). On chi-square difference and z tests in mean and covariance structure analysis when the base model is misspecified. *Educational and Psychological Measurement*, 64(5):737–757.

Yunus, M. B. (2008). Central sensitivity syndromes: a new paradigm and group nosology for fibromyalgia and overlapping conditions, and the related issue of disease versus illness. *Semin Arthritis Rheum*, 37(6):339–352.

Zautra, A., Smith, B., Affleck, G., and Tennen, H. (2001). Examinations of chronic pain and affect relationships: applications of a dynamic model of affect. *J Consult Clin Psychol*, 69(5):786–795.

Zautra, A. J., Johnson, L. M., and Davis, M. C. (2005). Positive affect as a source of resilience for women in chronic pain. *J Consult Clin Psychol*, 73(2):212–20.

Zautra, A. J., Parrish, B. P., Puymbroeck, C. M. V., Tennen, H., Davis, M. C., Reich, J. W., and Irwin, M. (2007). Depression history, stress, and pain in rheumatoid arthritis patients. *J Behav Med*, 30(3):187–197.

Zvolensky, M. J., Goodie, J. L., McNeil, D. W., Sperry, J. A., and Sorrell, J. T. (2001). Anxiety sensitivity in the prediction of pain-related fear and anxiety in a heterogeneous chronic pain population. *Behav Res Ther*, 39(6):683–696.