Risk and Resistance Factors in Chronic Pain

Joanne Maree Sheedy
B. Psych (Hons)
B. App. Sci (Physiotherapy)
Grad. Dip. Research Methods

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Abstract

The majority of research examining adjustment to chronic pain has focused on intra-individual risk predictors of poorer outcomes. Less research has explored ways that risk and resistance factors interact to influence pain adjustment outcomes. In the context of the current research, a risk factor is a variable that is associated with a worsened adjustment outcome; a resistance factor is a variable associated with enhanced adjustment outcomes. To address these deficits in the literature, Wallander and Varni's (Wallander et al., 1989; Wallander & Varni, 1998) generic risk-resistance model of adjustment to chronic paediatric health conditions was adapted to the chronic pain context. It offered a theory-driven approach to explore a range of effects likely relevant to chronic pain adjustment processes. Improved understanding of ways that a range of predictors directly and indirectly influence chronic pain outcomes will improve specificity of therapeutic targets.

Three studies were completed. The first explored direct and indirect influences of risk and resistance factors on pain-related disability, using pain clinic data obtained from 352 individuals. The second, qualitative study examined factors associated with improved adjustment via interviews with people perceived to be living well with chronic pain. Study Three was informed by Studies One and Two. It tested an expanded version of the model to examine direct and indirect influences of risk and resistance factors on pain-related disability and quality-of-life (QOL) in a community-based sample of 281 pain-affected adults.

Results: The qualitative study identified a range of positive processes that appeared to promote an improved capacity to live with pain. These factors were included in the Study Three model. Pain severity and pain self-efficacy were identified in both Studies One and Three as significant predictors of pain-related disability. In the Study One sample, negative affect and

partner responses to pain were also significant predictors of disability. A number of risk and resistance factors were identified as significant predictors of QOL. In all regression models, resistance factors explained additional variance in pain-related disability and QOL over and above that explained by the risk factors, highlighting that strengthening resistance factors in rehabilitation is important.

Mediation effects were explored using both single and parallel mediator models. In single mediator models, a number of pain appraisal and coping factors mediated relationships between predictors and adjustment outcomes. In parallel mediator models predicting pain-related disability, only pain self-efficacy mediated these relationships. In parallel mediator models predicting QOL, several resistance factors mediated these relationships. Moderation analyses identified that those reporting high levels of pain acceptance and values reported the lowest overall levels of pain-related disability, however the relationships between pain severity and negative affect with pain-related disability were stronger for those reporting high levels of the moderators compared to lower levels.

Conclusions: This research extends previous work by exploring direct and indirect influences of risk and resistance factors on pain-related disability and QOL. Pain severity and pain self-efficacy were critical factors associated with pain-related disability while a number of risk and resistance factors were associated with QOL. These factors all represent important therapeutic targets. Moderator analyses demonstrated some resistance factors strengthen risk-outcome relationships at the same time that they provide overall protective effects for adjustment. This highlights the importance of specific and individualised treatment plans.

Declaration

This thesis contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

Publications during enrolment

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Chapter One – Introduction

1.1 Overview

This research was designed to explore direct, moderating and mediating effects of psychosocial factors associated concurrently with adjustment to chronic pain. Chronic pain adjustment refers to the impact of pain on physical and psychological function and on well-being (Nicassio, 2011). This research aimed to explore adjustment processes by adapting a risk-resistance model of adjustment to chronic paediatric conditions (Wallander et al., 1989; Wallander & Varni, 1998). This framework was selected because of its strong theoretical underpinnings, its inclusion of a range of intra and inter-individual risk and resistance factors and because it offered clear and testable hypotheses of direct and indirect effects. A broad but testable framework that considers both direct and indirect effects is currently lacking within the chronic pain literature and offers the opportunity to expand current understanding of pain adjustment processes. Along with other key terms, the terms ‘adjustment’, ‘direct and indirect effects’ as they apply in the current research are defined below, in Section 1.6.

Three studies were completed, two quantitative and one qualitative. The first study was quantitative and served an exploratory purpose, to investigate the utility of the model in a pain clinic sample. A second qualitative study aimed to identify additional resistance factors that may improve the explanatory capacity of an expanded version of the model, which was then tested in a third quantitative study. This final study used a community-based sample. Specific detail of each of the three studies is provided below in Section 1.4. Together, the three studies aimed to improve understanding of the direct and indirect effects of a range of risk and resistance factors on chronic pain adjustment outcomes. It was intended this research would improve specificity of clinical intervention targets as well as provide a basis for further research and model development. The aim

of this first chapter is to provide the rationale, scope and need for the current research and to define key terms. An overview of chronic pain is provided and some limitations in existing models of chronic pain adjustment are described. Finally, a summary of thesis chapters is provided.

1.2 Study Rationale

Chronic pain, defined as prolonged pain of at least three months, is a common and costly health problem affecting one in five Australians (Hogg, Gibson, Helou, DeGabriele & Farrell, 2012). It is often associated with high levels of disability (Raftery et al., 2011) and psychological morbidity (Asmundson, Jacobson, Allerdings & Norton, 1996; Bair, Robinson, Katon, & Kroenke, 2003; Dersh, Gatchel, Mayer, Polatin & Temple, 2006). Considerable inter-individual variability in chronic pain adjustment has been demonstrated, with only weak links observed between pathology and disability (Andrasik, Flor & Turk, 2005). Because of this, much research has sought to identify and explore non-biological factors that contribute to chronic pain adjustment outcomes. A number of robust psychosocial predictors have been demonstrated (Turk & Okifiji, 2002). It has been suggested that adjustment to chronic pain is a process that likely involves both risk and resistance factors (Stanton, Revenson & Tennen, 2007; Walker, Jackson & Littejohn, 2004; Wallander & Varni, 1998). Risk factors are seen as factors that typically worsen the effect of an adversity. Resistance factors, on the other hand, are suggested to be those that reduce the negative impact of adverse situations (Masten & Tellegen, 2012). These terms are defined in detail in Section 1.6. The literature pertaining to their relationships with pain adjustment outcomes is provided in Chapter Two.

Over the past several decades, numerous theoretical models have been proposed to explore chronic pain adjustment processes. These models have promoted understanding of ways in which psychosocial influences may impact adjustment to chronic pain; the most commonly referenced of

these are outlined below in Section 1.7. However, it has been suggested that many existing models of chronic pain adjustment are too restricted in scope to adequately capture adjustment processes, or have been so comprehensive they have been difficult to evaluate empirically (Meredith & Strong, 2008). The current research applies Wallander and Varni's risk-resistance model of adjustment to paediatric disability (Wallander et al., 1989; Wallander & Varni, 1998) to explore chronic pain adjustment processes because it provides a theory-driven framework that examines processes likely relevant to chronic pain adjustment and provides clear, testable hypotheses. In this research it is used as the basis to explore the direct, indirect and interactive effects of a broad range of psychosocial risk and resistance factors on chronic pain outcomes.

1.3 Need for the Study

Much of the existing research exploring adjustment processes to chronic pain has occurred within the context of models that have focused on the negative effects of risk predictors on poorer outcomes (Gatchel, Peng, Peters, Fuchs & Turk, 2007). Exploration of resistance factors associated with positive adjustment to chronic pain may help promote a strength-based approach to treatment. Less research has explored factors associated with quality of life (QOL) compared to research that has explored factors associated with pain-related physical or psychological morbidity. Fewer studies still have specifically examined resistance factors associated with positive adjustment to chronic pain, despite the fact that not everyone living with chronic pain is disabled by it (Karoly & Ruehlman, 2006).

Although a large body of research has explored the influence of social experiences on pain adjustment outcomes, the majority of this research has not occurred within the context of a comprehensive, theory-driven framework. Existing models of chronic pain adjustment have tended to focus on intra-personal predictors of outcome (Jensen, Nielson & Kerns, 2003; McCracken &

Morley, 2014; Meredith, Ownsworth & Strong, 2008; Truchon, 2001; Turk, 2002a; Vlaeyen, Kole-Snijders, Boeren & van Eek, 1995). Yeung, Arewasikporn and Zautra (2012) suggested that the inclusion of social influences is critical when modelling adjustment to chronic pain because positive social experiences may facilitate adaptive responses in times of challenge. At the same time, some negative social interactions have been shown to exert a direct negative influence on measures of adjustment (Leonard, Cano & Johansen, 2006; Newton-John, 2002; Romano, Jensen, Turner, Good & Hops, 2000; Stroud, Turner, Jensen & Cardenas, 2006). Thus, the effect of social experiences on pain adjustment outcomes is likely complex.

A final limitation of existing models of adjustment to chronic pain is a focus on direct or mediated relationships between predictors and outcomes. This represents a limitation because adjustment processes to both chronic health conditions generally, and to chronic pain specifically, are suggested to be complex and likely to involve both mediator and moderator effects (Wallander & Varni, 1998; Wright, Zautra & Going, 2008). Such effects specify the mechanisms through which predictors influence outcomes and the conditions under which a predictor is likely to exert an adjustment influence (Kraemer, Stice, Kazdin, Offord & Kupfer, 2014). Conceptual and analytic clarity about the nature of risk and resistance factors and how they interact is important if models of adjustment are to be used for treatment planning. Clarity about indirect effects supports the targeting of therapeutic interventions towards individuals most likely to benefit, or towards those most at risk of adverse outcomes (Kraemer et al., 2014). Definitions of these terms are provided below in Section 1.6. Overall the aim of the current research was to improve understanding of the direct, indirect and interactive effects of intra and inter-personal psychosocial predictors on both negative and positive adjustment to chronic pain.

1.4 Scope and Design of this Research

The scope of this research was limited to adults aged 18 and over, living in Australia with any type of chronic, non-malignant pain condition. Heterogeneous samples of individuals with chronic pain were sought for the research because many people with chronic pain do not have a clear diagnosis and may never have the exact cause of their pain identified. For example, it has been reported that up to 65% of people with chronic pain lack a clear diagnosis (Blyth, March, & Cousins, 2003). It has also been suggested by some researchers that the range of psychosocial factors associated with adjustment to chronic pain may not be specific to particular diagnoses (Burke, Mathias & Denson, 2015). Exploring adjustment processes that are relevant to a broad range of people living with pain is important because findings from this type of research will provide insights relevant to the broad groups of patients most commonly encountered in clinical settings.

The overall research design was both quantitative and qualitative. Study One explored the direct and indirect effects of risk and resistance factors on pain-related disability. The study employed a quantitative design, using cross-sectional pre-existing data obtained from a multi-disciplinary pain management unit. Participants in Study One were adults with any type of chronic, non-malignant, mainly musculoskeletal pain condition, who had been referred to the pain management unit for assessment of suitability to complete a pain management program. Consequently the measures within Study One were constrained to those available in the clinical database. The model tested in Study One can be seen below in Figure 1.7.

This first study served an exploratory function, to test the proposed model with a subset of predictors available in the clinical database to explore factors associated with pain-related disability. Then, because research exploring resistance factors is more limited than that exploring risk factors, a second qualitative study was completed. This second study aimed to identify

resistance factors associated with positive adjustment to chronic pain. Few, if any, studies have explored resistance factors that promote improved adjustment in a group of individuals perceived to be functioning well despite pain. Therefore, participants in Study Two were adult patients of a single general medical practice that were perceived by treating health professionals to be coping well with pain. It was thought that these individuals might offer insights into resistance factors that may not have been previously identified. It was intended that results of Studies One and Two, together with the literature review in Chapter Two, would be used to inform selection of additional predictors for a final quantitative study, Study Three.

Study Three was planned because a range of resistance factors have been identified in the literature, in addition to those included in Study One. These factors are reviewed in Chapter Two. It was intended that this final study would replicate the design of Study One, to explore direct and indirect effects proposed by the model. Major strengths of Study Three were that it included a broad range of risk and resistance factors selected from a range of theoretical paradigms. It also included a measure of positive adjustment, QOL, as an additional outcome to pain-related disability. As an aim of Study Three was to explore positive and negative adjustment outcomes, the Study Three sample included people who were potentially functioning well despite pain as well as those experiencing pain adjustment difficulties. Participants in Study Three were recruited from exercise facilities as well as health centres and Australian online pain groups. Study Three participants were adults with any type of chronic non-malignant pain diagnosis.

1.5 Overview of Chronic Pain

1.5.1 Definition of chronic pain.

The International Association for the Study of Pain (1986) defined pain as ‘an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms

of such damage'. Implicit to this definition is the idea that the psychological aspect of the pain experience encompasses affect, behaviour and cognition (Novy, Nelson, Davis & Turk, 1995).

Acute pain is a universal experience and a biologically protective mechanism. Acute pain occurs in response to actual or potential tissue damage and is generally of short duration, consistent with the time required for tissue healing (Apkarian, Baliki & Geha, 2009). Chronic pain, on the other hand, can be experienced for months, years or decades. It is pain which persists longer than would be expected by the antecedent event. Chronic pain conditions are generally labeled by their site of injury, for example, low back pain, or by the type of condition, for example, neuropathic, arthritic, diabetic (Apkarian et al., 2009). Different types of tissue injury may contribute to a single clinical condition. For example chronic low back pain may result from a combination of joint, muscle or nerve injury and the relative contribution of each of these elements to the presentation may be indeterminable (Apkarian et al., 2009). The literature reviewed in this thesis relates only to pain associated with chronic non-malignant conditions. The majority of the reviewed literature is based on samples of individuals experiencing chronic pain as a result of musculoskeletal problems such as spinal problems or pain resulting from degenerative joint disease, such as arthritis.

1.5.2 Prevalence of chronic pain.

The prevalence of chronic pain in Australia has been reported to be around 18% (Blyth et al., 2001; Currow, Agar, Plummer, Blyth & Abernethy, 2010; Henderson, Harrison, Britt, Bayram & Miller, 2013). These figures are consistent with international prevalence rates (Reid et al., 2011). Higher pain prevalence is reported in older populations, with up to 50 per cent of adults aged over 65 years reported to experience pain on a daily basis (Yonan & Wegener, 2003). Based on current population estimates, these figures equate to at least four million Australians living with chronic non-cancer related pain in 2015 (Henderson et al., 2013).

1.5.3 Costs associated with chronic pain.

Chronic pain is associated with a high socio-economic burden. Direct costs of chronic pain include medications, operative procedures, regional anesthesia, pain management programs and allied health treatments (Annemans, 2011). Indirect costs result from work absences, reduced productivity and welfare payments (Turk, 2002b). The net economic impact of chronic pain to the Australian community was estimated in 2007 to be in excess of $34 billion (Access Economics, 2007). The individual cost of chronic pain can also be significant, with up to a 50% loss of income demonstrated for people living with rheumatoid arthritis over a nine year period (Keefe et al., 2002).

1.5.4 Mechanisms of chronic pain.

In its simplest representation, pain is experienced when a pain message is sent to the spinal cord via a peripheral nerve once a threshold level of pain receptor activation is reached. This pain message is then transmitted via the spinal cord to the cerebral cortex, where it is perceived and interpreted (Brookoff, 2000). Pain processing though, is understood to be substantially more complex than this. Multiple areas of the brain are known to be involved, including the thalamus, the posterior and anterior insulae, the secondary somatosensory cortex, the anterior cingulate cortex and the periaqueductal grey matter (Wager et al., 2013). Widespread ascending pathways from the spinal cord to the brain can also activate multiple brainstem and subcortical regions, limbic pathways, and both ipsilateral and contralateral cortical brain regions involved in pain perception (Basbaum & Fields, 1984). These pathways intermingle with regions of the brain that mediate emotions, autonomic activity, attention and localization, motor planning and cognition.

Ongoing nerve signals arising from tissue or nerve injury sites can, over time, lead to long-term changes in the central nervous system that contribute to the amplification and persistence of

chronic pain (Butler, 2000). These changes in the central nervous system are referred to as central sensitisation (Ren & Dubner, 1999). Central sensitisation involves an increase in the excitability of medullary and spinal dorsal horn neurons. As a result of increased afferent input, peptide and excitatory amino acid neurotransmitters are released in greater amounts and for longer periods than in acute injury (Ren & Dubner, 1999). Thus as the pain duration extends, the pain can change from an experience linked to pathology, to a complex condition in its own right (Fine, 2011).

Pain perception is also strongly influenced by output regulatory systems. Output systems modulating the pain experience stem from a number of brain regions and include the endogenous opiate system (Basbaum, & Fields, 1984) and brainstem centres such as the dorsal raphe nucleus and locus ceruleus. These centres in turn are influenced by cortical, subcortical, and limbic pathways. Endogenous neurotransmitters, including oxytocin, vasopressin, histamine, prolactin, and cannabinoids also play a key role in modification of the outgoing pain signal (Hainline, 2005). In addition, systems such as the reward-learning-motivation system also influence pain perception and modulation although the precise role of such systems remains unclear (Denk, McMahon & Tracey, 2014). Improved understanding of the neuro-scientific basis of chronic pain has made it increasingly clear that pain perception involves both nociception (body to brain messages) as well as top-down processing such as context, memory and cognitions. For example, fear-based appraisals of pain have been demonstrated to be strongly related to an increased perception of pain and higher levels of disability (Jackson, Wang & Fan, 2014). This research is reviewed in Chapter Two.

1.6 Definitions of Key Terms

The terms adjustment to chronic pain, adjustment, predictors, outcomes, direct effects, indirect effects, mediation and moderation are defined in order to clarify these terms and the

common ways they are operationalised in the literature. These definitions also outline the inclusion criteria used to select the literature reviewed in Chapter Two.

1.6.1 Adjustment to chronic pain.

Individuals living with chronic pain are likely to experience disruption to a number of different facets of their lives as a result of their condition. Chronic pain adjustment is most commonly reflected by the impact of pain on physical and psychological function and, less commonly, on QOL (Nicassio, 2011). In this thesis, the term adjustment refers to pain-related disability and QOL.

Measures of pain-related disability ask respondents to indicate the extent to which pain impacts on their ability to complete functional tasks such as dressing, washing, standing and walking (Kerns, Turk & Rudy, 1985; Roland & Morris, 1983). Up to 37% of those living with chronic pain have been reported to experience moderate or severe pain-related disability (Raftery et al., 2011). Pain-related interference is also a measure of the impact of pain on physical function. It is a criterion variable of some of the research reviewed in Chapter Two. Measures of pain-related interference assess more broadly the impact of pain on everyday tasks and mood (Scudds & Østbye, 2001).

Quality of life has been reported to be significantly lower in individuals with chronic pain (Raftery et al., 2011). The more specific measures of the impact of health on QOL, health-related QOL (HRQOL), are also reported in some research. The operational definition for the construct of general QOL continues to be debated. No single measure exists specific to QOL in the chronic pain context, rather, a range of validated measures have been used. Quality of life scales generally reflect a subjective evaluation of the extent to which a person is satisfied with various aspects of life.

The World Health Organisation QOL Group define QOL as:

"…an individual's perceptions of their position in life, in the context of the culture and value systems in which they live, and in relation to their goals, expectations, standards and concerns. It is a broad ranging concept, affected in a complex way by the person's health, psychological state, level of independence, social relationships and their relationship to salient features of their environment". The construct of QOL is operationalised in this research as general QOL, reflecting the above definition.

Adjustment is also operationalised in the chronic pain literature as psychological adjustment, or as symptoms of anxiety and depression. Chronic pain is strongly associated with psychological morbidity (Bair et al., 2003) with around 50% of chronic pain patients estimated to suffer co-morbid depression (Dersh et al., 2006). Elevated anxiety rates are also frequently reported (Asmundson et al., 1996; Holzberg, Robinson, Geisser, & Gremillion, 1996). Measures of psychological function are represented in the literature as both adjustment predictors and outcomes. In the current research, measures of psychological function are placed in the model as risk factors. This issue is explored in more detail in Chapter Two.

An important factor to note in relation to the current research is that despite that the hypothesised model implies directionality, in reality it represents a recursive model in which risk and resistance factors and outcomes exert reciprocal influences. Because the current research utilises cross-sectional data, no conclusions can be drawn about the direction of relationships in the model. It is therefore an important point that the term 'predictor' in this thesis refers to statistical prediction and does not imply a cause and effect relationship. Rather than attempting to establish causal relationships, the current research aimed to instead explore factors broadly associated with physical function and QOL for those living with chronic pain with a view to identifying targets for clinical intervention and informing further model development.

1.6.2 Predictors and outcomes.

The term ‘predictors’ in the current research refers to risk and resistance factors statistically associated with the measures of adjustment, not necessarily causal factors. The term ‘outcomes’ refers to measures of adjustment to chronic pain and in the current research, refers to pain-related disability and QOL. These measures represent the main criterion variables both of the reviewed literature and the quantitative studies in this thesis.

1.6.3 Risk and resistance factors.

Theorists outside of the chronic pain field have paid considerable research attention to the roles of risk and resistance factors and how they may act directly and indirectly to influence adjustment. Much of this work has explored resilience processes occurring in the context of adversity. Predictors are described as risk or resistance depending on where their influence is primarily exerted (Johnson, Wood, Gooding, Taylor & Tarrier, 2011; Masten & Tellegen, 2012; Rutter, 2012). For example a factor would be labelled resistance if an adverse situation is reduced by its presence. Conversely, a risk factor is associated with a worsened outcome (Masten & Tellegen, 2012).

Rose, Holmbeck, Coakley and Franks (2004) further describe risk and resistance factors in terms of their influence at varying levels of risk. They describe a risk factor as one that negatively influences outcomes regardless of the level of risk, and a vulnerability factor as one that increases the likelihood of a poor outcome only under adverse conditions. By contrast, they describe a resource factor as one that positively influences outcome, regardless of the level of risk, and a protective factor as one that decreases the chance of a poorer outcome under adverse conditions. Therefore, protective and vulnerability factors can both be considered moderators, as they specify the conditions under which risk predictors exert their negative effects (Holmbeck, 1997).

1.6.3.1 Direct, indirect, mediator and moderator effects.

This thesis adopted the following definitions of direct, indirect, moderator and mediator effects. The adopted definitions are consistent with those described by Holmbeck (2002), Rose and colleagues (2004) and Rutter (2009).

1.6.3.2 Direct effects.

Direct effects refers to when a predictor has an impact on an outcome variable, regardless of the level or presence of other variables. See Figure 1.1.

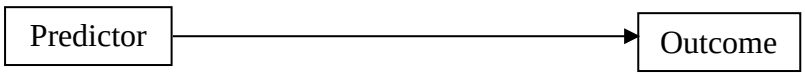

Figure 1.1 Direct effect

1.6.3.3 Indirect effects.

Indirect effects refers to when a predictor affects an intermediate variable, which then affects an outcome variable. See Figure 1.2.

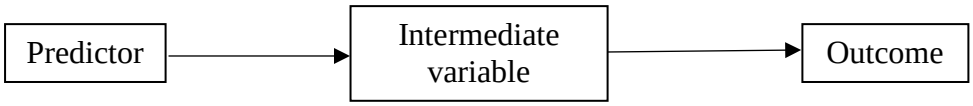

Figure 1.2 Indirect effect

1.6.3.4 Mediation effects.

Mediation effects occur when one intermediate variable explains how a predictor influences an outcome (Rutter, 2009). Mediated effects are usually only hypothesised when a predictor and outcome relationship is already established, that is, that a relationship is available to be mediated (Rose et al., 2004). Therefore, mediation requires that the predictor and outcome variables are

correlated. A predictor can have its relationship with an outcome partially or fully mediated. Partial mediation means that both the direct and indirect effects from the predictor to the outcome variable are significant. Full mediation means that the direct effect drops out of significance when the mediator is added, and that the indirect effect is significant (Baron & Kenny, 1986). Significant mediation effects mean that addressing the mediator in treatment is important, otherwise a critical link in a chain of influence is missed (Kraemer, Wilson, Fairburn, & Agras, 2002; Stanton, Revenson & Tennen., 2007). Mediation is depicted below in Figure 1.3.

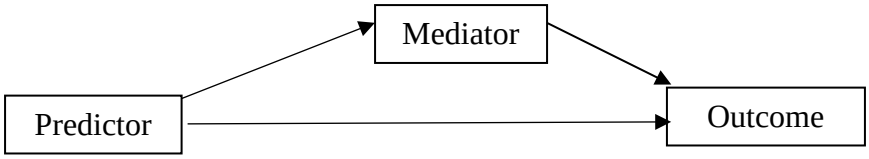

Figure 1.3 Representation of a mediator relationship

1.6.3.5 Moderation effects.

Moderators are those variables that cause another variable to operate differently on an outcome (Rutter, 2009). That is, moderators are variables that influence the strength or direction of a relationship between a predictor and an outcome (Rose et al., 2004). Moderator effects may be explored when it is noted that the influence of one predictor on an outcome varies according to the level of other variables, or when the literature reports mixed findings for the relationship between predictor and outcome measure. Exploration of moderator effects promote understanding of the conditions under which certain predictors influence outcomes (Rose et al., 2004). See Figure 1.4.

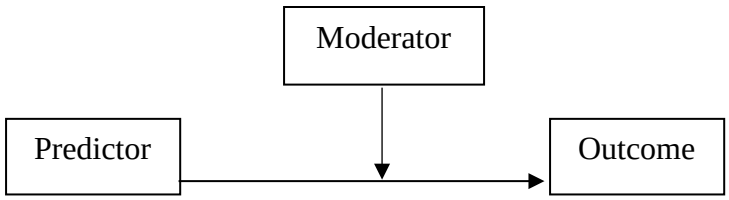

Figure 1.4 Representation of a moderator effect

1.7 Conceptual Models of Chronic Pain Adjustment

Multiple models of adjustment to chronic pain have been proposed. One of the most commonly referenced is the biopsychosocial model of chronic pain (Gatchel et al., 2007). In general, biopsychosocial models of chronic health conditions propose that adjustment is the result of a complex interaction of biological, psychological, and social factors (Gatchel et al., 2007). In the context of chronic pain, the biological component alone encompasses a large number of factors that include inflammatory, neurological and genetic (Gatchel et al., 2007).

Because of the complexity of such processes, and because links between physical parameters and outcomes in chronic pain are often weak (Andrasik et al., 2005; Brown, Nicassio & Wallston, 1989; Keefe, Brown, Wallston & Caldwell, 1989), a large body of research addressing chronic pain adjustment processes has focused only on psychosocial predictors of pain adjustment. This literature presents theories that stand alone from biological influences (McCracken & Morley, 2014). A number of psychosocial explanatory models have been proposed. It is in this area of research that this thesis is focused.

This section provides an overview of the most prevalent psychosocial models of chronic pain adjustment. In general, these models have offered theory-driven heuristics for understanding adjustment and possible points for clinical intervention. Where these models have been explored empirically, this evidence is presented in Chapter Two. Strengths and deficits of these models are

reviewed below. For the purposes of organising this literature, the review of existing models of chronic pain adjustment firstly presents a number of models arising from the stress and coping framework (Lazarus & Folkman, 1984). Several other models such as the psychological flexibility model of chronic pain (McCracken & Morley, 2014) and the motivational model of chronic pain (Jensen et al., 2003) are also reviewed.

1.7.1 Models arising from the stress and coping framework.

Lazarus and Folkman's transactional model of stress and coping (1984) has given rise to a number of different psychosocial models of pain adjustment. According to this framework, a transaction occurs between the individual and the environment upon encountering life events. Stress arises when the demands of the event are perceived to exceed the individual's coping resources. Coping refers to cognitive responses and behaviours adopted in the face of a stressor. The stress and coping framework (Lazarus & Folkman, 1984) proposes that, upon encountering a stressor, a primary appraisal process occurs during which the threat potential of the stressor is assessed. A secondary appraisal process is then suggested to occur, during which the availability of internal and external resources is reviewed. Lazarus and Folkman's (1984) theory of stress and coping depicts relationships between predictors and outcomes as dynamic and reciprocal. This model is shown in Figure 1.5 below.

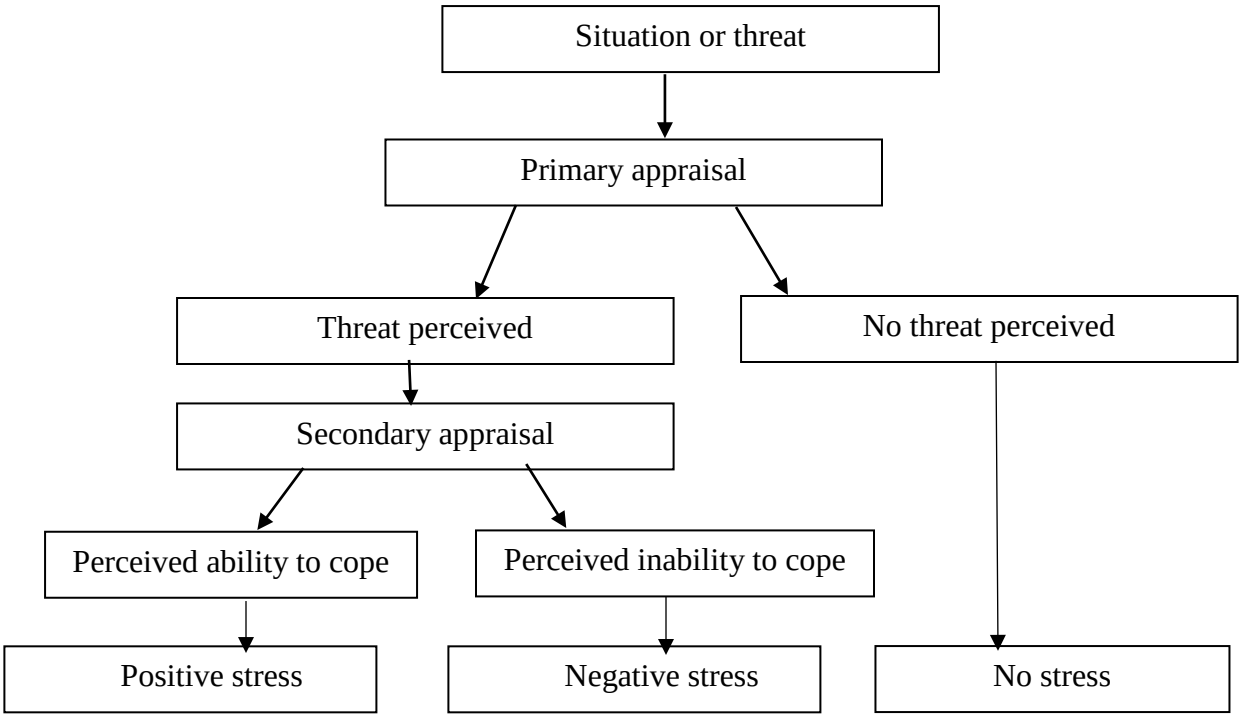

Figure 1.5 Lazarus and Folkman's (1984) transactional model of stress and coping

1.7.1.1 Fear-avoidance model of chronic pain.

The fear-avoidance model of chronic pain adjustment (Vlaeyen, Kole-Snijders, Rotteveel, Ruesink & Heuts, 1995b) is a widely researched model of adjustment to chronic pain. It is a simple and recursive cognitive-behavioural model that has provided useful clinical utility. The fear-avoidance model proposes that pain interacts with catastrophising and negative affect to produce fear-avoidance which subsequently leads to avoidance behaviours and disability. Robust links have been established between fear-avoidance and pain-related disability (Linton, 2000; Nieto, Miro & Huguet, 2013; Picavet, Vlaeyen & Schouten, 2002; Samwel, Kraaimaat, Crul & Evers, 2002), but several criticisms have been made of the fear-avoidance model (Vlaeyen et al., 1995b). The first is that the temporal sequence of the paths proposed by the model has not been empirically supported (Wideman, Adams & Sullivan, 2009). It has also been suggested that model focus on threat appraisals of pain for explaining pain-related physical function may limit its predictive capacity as

it may neglect other important intra-individual and contextual risk and protective factors (Wideman et al; 2013). Additionally, the assumption of the fear-avoidance model that fearful thoughts of pain are based in psychopathology goes against the observation that such thoughts may be quite normal and culturally endorsed (Crombez, Eccleston, Van Damme, Vlaeyen & Karoly, 2012). A final criticism of the fear-avoidance is that it may have underestimated the impact of pain intensity on avoidance behaviours (Crombez et al., 2012).

1.7.1.2 Diathesis–stress model of chronic pain.

In order to expand the scope of the fear-avoidance model, Turk (2002) introduced post-traumatic stress to this model. This 'diathesis-stress' model is also cognitive-behavioural, based on the stress and coping framework (Lazarus & Folkman, 1984). It also proposes a sequential process to explain adjustment to chronic pain following injury. 'Diathesis' refers to any biological or psychological characteristic that increases an individual's chance of developing a disorder (Turk, 2002). The model proposes that the injury itself, combined with anxiety sensitivity, together influence appraisal processes such as catastrophising, fear-avoidance and pain self-efficacy. The combination of these factors is proposed to influence pain avoidant behaviours that contribute to disability. The diathesis-stress model suggests that pain behaviour is elicited by organic factors, but is maintained by reinforcing environmental effects (Turk, 2002). However, given it is an extension of the fear-avoidance model, similar limitations to those listed above are also applicable to this model.

1.7.1.3 Stress-process model of chronic pain.

In another cognitive-behavioural approach, Truchon (2001) proposed the stress-process model of chronic pain. This model proposes that stressor appraisals and stressful life events together influence level of emotional distress and degree of avoidance coping, which both then

directly influence level of pain-related disability. This model offers a theory-driven representation of risk influences of adjustment and has been shown to have moderate explanatory power for pain-related disability (Truchon et al., 2010). This model does not explore indirect effects, nor does it include social factors.

1.7.1.4 Attachment-diathesis model of chronic pain.

Meredith and colleagues (2008) argued that attachment theory provides an additional explanatory framework for understanding adjustment to chronic pain. Their attachment-diathesis model proposes that pain interacts with individual attachment style to influence cognitive appraisals of coping resources and ultimately, adjustment. This model represents an intra-individual model of chronic pain adjustment and as a whole has not yet been empirically investigated.

1.7.2 Other models of chronic pain adjustment.

1.7.2.1 Psychological flexibility model of chronic pain.

Psychological flexibility is defined as the capacity to develop improved awareness of thoughts, direct experience of feelings and the ability to direct behavioural efforts towards valued life goals (Hayes, Luoma, Bond, Masuda & Lillis, 2006; McCracken & Morley, 2014). The concept of psychological flexibility arises from acceptance and commitment therapy (Hayes, Strosahl & Wilson, 1999), an approach that emphasises acceptance of thoughts and experiences rather than efforts to change them. Both acceptance and commitment therapy and the psychological flexibility model of chronic pain consist of various components including acceptance, values-based behaviour, present-focused awareness and reduction of self-identification with thoughts (McCracken & Morley, 2014). This model focuses on positive psychological processes that promote improved adjustment to chronic pain. A large volume of research supports the efficacy of

the processes within the model for pain rehabilitation outcomes (McCracken, 1998; McCracken & Thompson, 2011; Veehof, Oskam, Schreurs & Bohlmeijer, 2011; Vowles, McCracken & O'Brien, 2011; Vowles, Sowden & Ashworth, 2014). This research is reviewed in Chapter Two.

1.7.2.2 Motivational model of chronic pain.

Referencing existing models of health behaviour change, Jensen and colleagues (2003) proposed a motivational model of chronic pain. This model suggests that a person assesses the importance of a behaviour change that will improve pain coping, and that this appraisal, together with self-efficacy beliefs, influences a person's stage of readiness to change. Readiness to change in turn influences capacity to adopt positive pain coping strategies such as exercise, relaxation and task persistence. This model focuses on intra-individual predictors of adjustment.

1.7.2.3 Model of resilient functioning in chronic pain.

In response to some the above noted limitations, Yeung, Arewasikporn and Zautra (2012) proposed a conceptually broader adjustment model. Similar to the current research, these authors suggested that resilient functioning in chronic pain is the outcome of an interplay between inter and intra-personal resilience and vulnerability factors. Their model of resilient functioning breaks these two factors down into stable and modifiable predictors. Specific modifiable resilience predictors include approach coping, benefit-finding and family support. Stable resilience predictors include trait optimism, resilience and social networks. Stable vulnerability predictors in this model are trait neuroticism, history of affective disorders and childhood trauma. Modifiable vulnerability predictors are anxiety and depression symptoms, catastrophising and negative familial exchanges. This model is inclusive of both intra and inter-individual adjustment predictors, but in its current form lacks specific hypotheses about how these predictors might interact. As yet, the model its entirety has not been explored empirically.

1.7.3 Summary of existing models of adjustment to chronic pain.

Multiple models of adjustment have been proposed to guide chronic pain research and treatment. The majority of these focus on intra-individual risk and resistance factors associated with adjustment and offer valuable theoretical heuristics for understanding chronic pain adjustment processes. Simmonds, Moseley and Vlaeyen (2008) have argued that integrating knowledge from a range of perspectives may be important in order to progress understanding of adjustment processes. This highlights the need for a comprehensive model of chronic pain adjustment that can be empirically explored. A comprehensive framework is needed to organise and understand the existing research, integrate competing perspectives and further probe the ways in which predictors interact to influence outcomes. Improved understanding of these effects would enhance the development of clinical interventions by allowing influential predictors from varying research and therapeutic paradigms to be targeted.

1.8 Risk-Resistance Models

One method to facilitate this exploration is a risk-resistance approach. Within this approach, adjustment is understood to be the outcome of a complex interplay of a number of different factors (Wallander et al., 1989; Wallander & Varni, 1998). Risk-resistance models have been utilised to explore adjustment and resilience in the areas of post-disaster research (Benight, 2012), developmental psychology (Olsson, Bond, Burns, Vella-Brodrick & Sawyer, 2003), paediatric chronic conditions (Wallander et al., 1989; Wallander & Varni, 1998, McLean, Harvey, Pallant, Bartlett & Mutimer, 2004, McLean, Harvey & Mutimer, 2014) and to a small extent, adjustment to chronic pain (Alschuler, Kratz & Ehde, 2016; Furlong, Zautra, Puente, Lopez-Lopez & Valero, 2010; Libby & Glenwick, 2010; Sturgeon, Zautra and Arewasikporn, 2014; Yeung et al., 2012; Wright et al., 2008). Of the above research, Wallander and Varni's risk-resistance model of

adjustment to chronic paediatric conditions (Wallander et al., 1989; Wallander & Varni, 1998) is notable because it offers a comprehensive, theory-driven and empirically validated approach for exploring adjustment processes. A particular advantage of this framework is that it proposes testable hypotheses likely to be relevant to chronic pain adjustment processes.

Wallander and Varni (1998, Wallander et al., 1989) referenced Lazarus and Folkman's (1984) stress and coping framework in developing their model aimed at exploring relationships between risk and resistance factors that influence adjustment to paediatric disability. Their model was intended to be generic and applicable across a range of paediatric conditions. Wallander and Varni's model (Wallander et al., 1989; Wallander & Varni, 1998) proposes that risk and protective factors exert direct effects on adjustment independent of other predictor variables. The model also proposes a number of complex interactions between variables, such as mediator and moderator effects (Bretherton & McLean, 2014; McLean et al., 2014; McLean et al., 2004). This model has been used to model adjustment in a range of settings that include congenital physical disability (Wallander et al., 1989), African American adolescents with intellectual disability (Frison, Wallander & Browne, 1998), obstetric brachial plexus injury (McLean et al., 2004), paediatric sleep disturbance (Moore, Gordon & McLean, 2012) and psychological adjustment in older people (Bretherton & McLean, 2014).

Categories of risk predictors in Wallander and Varni's original model of child adjustment (Wallander et al., 1989; Wallander & Varni, 1998) are disease parameters (for example condition severity), functional independence and psychosocial stressors (for example, daily hassles). Categories of resistance factors were stress-processing variables (appraisal and coping factors), social-ecological factors (for example family support) and intrapersonal factors (for example, temperament). Because of the comprehensive nature and the clarity offered by the model in terms of hypothesised effects, the current research adapted Wallander and Varni's risk-resistance model

(Wallander et al., 1989; Wallander & Varni, 1998) to the chronic pain context using existing literature. Risk categories in the current model are condition parameters (perceived pain severity), stress-processing factors (catastrophising and fear-avoidance), intrapersonal factors (anxiety and depression) and social-ecological factors (punishing and solicitous partner responses to pain). Resistance categories in the current model are stress-processing factors (pain self-efficacy and pain acceptance), personal factors (optimism and positive affect) and social-ecological factors (perceived social support). Wallander and Varni's (Wallander et al., 1989; Wallander & Varni, 1998) original model is depicted in Figure 1.6. Following that, in Figure 1.7, the framework is outlined again, adapted for the current research. The model tested in Study One was further refined for Study Three according to the results of Study Two, the qualitative research.

Wallander's model (Wallander et al., 1989; Wallander & Varni, 1998) firstly proposes that each risk and resistance predictor will exert a direct negative or positive effect, respectively, on adjustment. Secondly, mediator effects are proposed in which some or all of the direct relationship between the social-ecological and intrapersonal factors and adjustment is mediated by the stress-processing factors. Thirdly, the model proposes moderator effects where the relationship between risk factors and adjustment varies with, or is moderated by, levels of the resistance factors. A final indirect effect, consistent with the stress and coping framework (Lazarus & Folkman, 1984) is also proposed. Stress-processing factors, both risk and resistance are hypothesised to mediate the relationship between perceived pain intensity and adjustment. The literature review of factors potentially influencing pain adjustment outcomes is reviewed in Chapter Two.

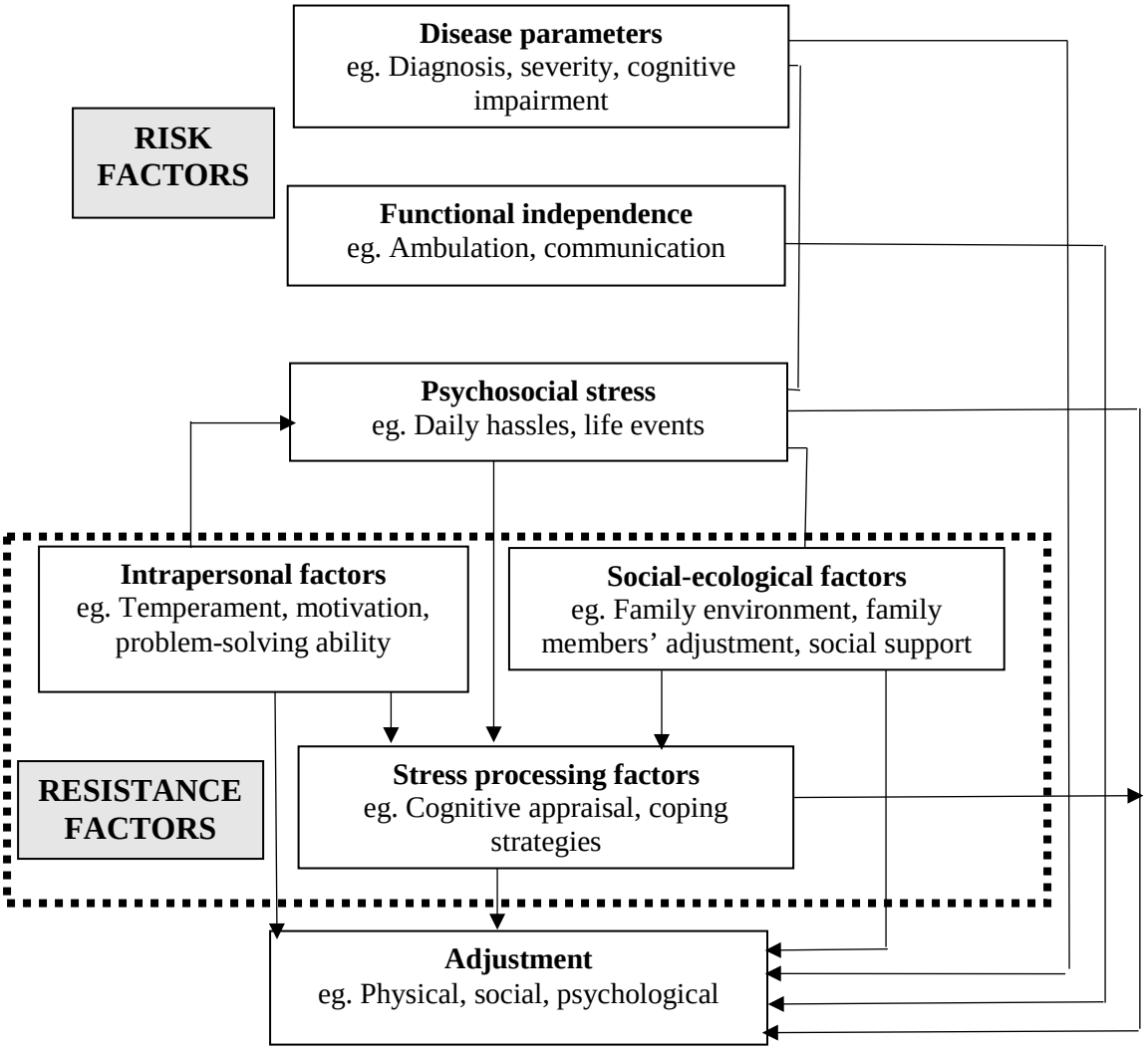

Figure 1.6 Model of child adjustment to paediatric chronic physical disorders (Wallander et al., 1989; Wallander & Varni, 1998)

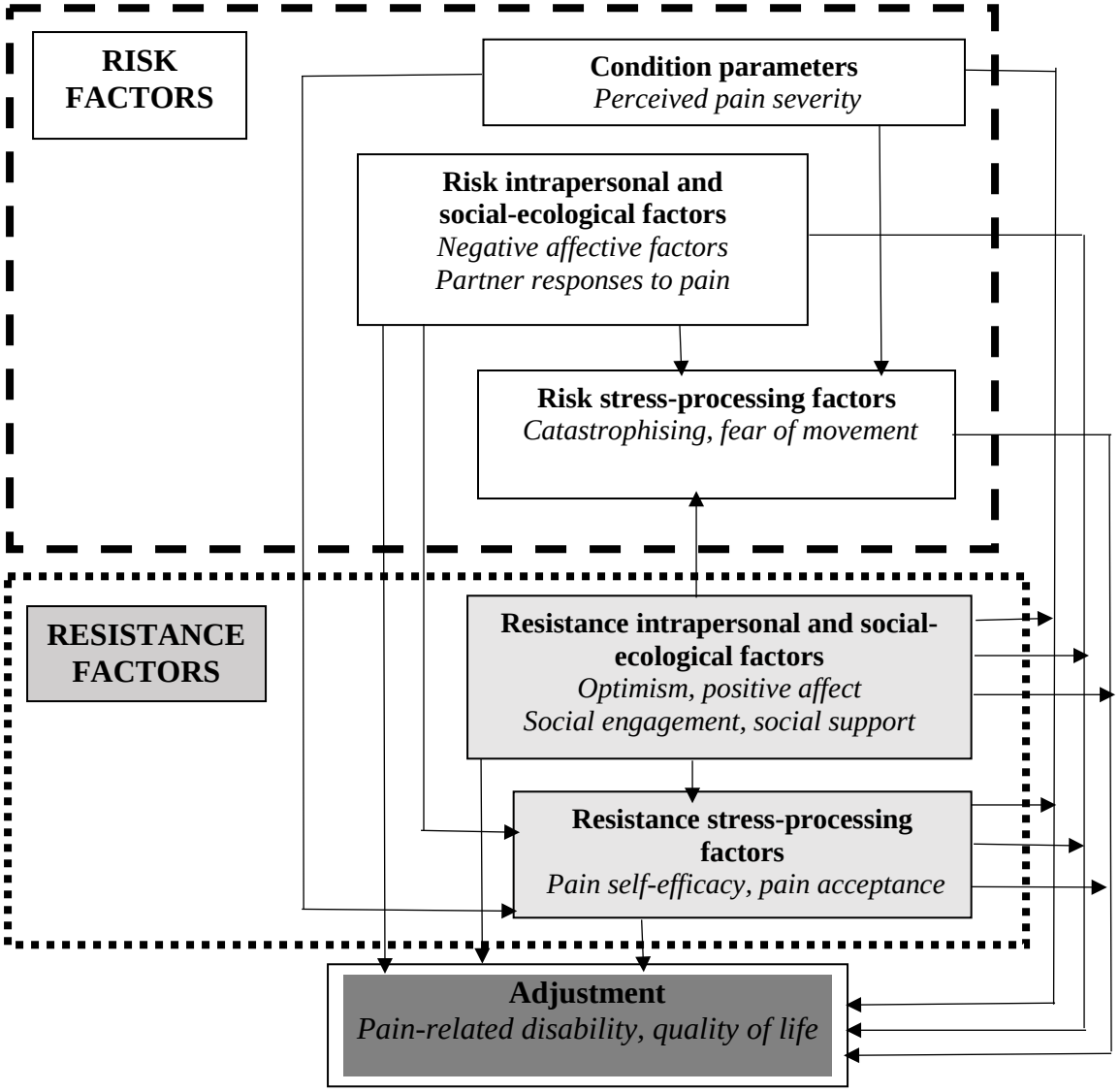

Figure 1.7 Proposed model of adjustment to chronic pain (adapted with permission from Wallander et al., 1989; Wallander & Varni, 1998).

Note 1: Study hypotheses are indicated by arrows.
Note 2: Existing literature was used to guide placement of variables within the model, hence stress-processing and social-ecological variables are placed as both risk and resistance factors.

1.9 Overview of the Thesis

This thesis is organised into five sections: introduction, literature review, methodology and results of each of the three studies, followed by discussion and conclusion. The thesis addresses these sections in nine chapters. Chapter One provides an introduction to the research. It details the research problem and provides an overview of the structure of the studies and the significance of the research. This chapter also reviews the prevalence, associated disability, costs and mechanisms of chronic pain. The theoretical literature related to models of adjustment to chronic pain is then presented.

A salient point from this chapter is that pathology is often not commensurate with disability, meaning that factors other than physical ones can make a large contribution to the individual variations in pain adjustment. This chapter highlights some of the limitations of existing models of chronic pain adjustment and outlines why the application of a risk-resistance approach to analysis of adjustment to chronic pain may offer additional insights for tailoring clinical interventions. The final part of this first chapter provides an overview of the structure of the thesis.

Chapter Two reviews the current literature regarding risk and resistance factors relevant to adjustment to chronic pain. These factors are categorised as risk or resistance factors and are reviewed in the context of each category of the conceptual model in which they are placed. Chapter Two reviews previous research identifying direct and indirect effects of risk and resistance factors on chronic pain adjustment outcomes. Where the models presented in Chapter One have been investigated empirically, these results are also presented under the relevant headings in this chapter. The chapter concludes with the identification of the research questions of the first study.

Chapter Three begins with an introduction to Study One. The aims, research questions and hypotheses are outlined. Study One methodology is described including information about the Study One participants, measures, method of data collection, and statistical analysis.

Chapter Four contains the results of Study One. Descriptive statistics for the study variables are documented and analyses of direct and indirect effects of the risk and resistance factors on chronic pain adjustment are reported. Implications for Study Three are discussed.

Chapter Five outlines the qualitative methodology, participants, measures, method of data collection, and analytical approach of Study Two. Chapter Six provides results of Study Two and discusses implications for Study Three.

Chapter Seven describes the methodology, participants, measures, method of data collection, and analytical approach of Study Three. Chapter Eight provides the analyses and results of Study Three.

Chapter Nine provides the discussion and outlines the findings of the research in light of current literature, highlights the contribution of this research to the body of literature, discusses limitations and offers insights for further research.

Chapter Two – Literature Review

2.1 Introduction

A large volume of literature has established that genetic, biological and psychosocial factors all influence physical function and quality of life (QOL) for those with chronic pain (Gatchel et al., 2007). The current research is guided by the literature that has explored psychosocial predictors of pain-related physical function and QOL or health related QOL (HRQOL). An adapted version of Wallander's risk-resistance model of adjustment to chronic paediatric health conditions (Wallander et al., 1989; Wallander & Varni, 1998) is used to integrate the findings of this large body of research and to explore the inter-relationships of risk and resistance predictors with chronic pain outcomes.

The original model and the adapted version can be seen in Figures 1.6 and 1.7 in Chapter One. Risk and resistance factors were included in the current model on the basis of their demonstrated relationships with outcomes, and because they represent targets of therapeutic intervention. Within the risk and resistance conceptual framework, adjustment is seen as a complex product of inter-relationships between some, if not all, risk and resistance factors. The current research aims to explore these inter-relationships and their resultant impact on pain adjustment outcomes.

This literature review is structured according to the categories in the adapted model. The first section of the literature review addresses risk factors. Categories of risk factors in the current model are condition parameters, stress-processing factors, intrapersonal factors and social-ecological factors. The second section of the literature review addresses resistance factors. Resistance categories in the adapted model comprise stress-processing factors, intrapersonal factors and social-ecological factors.

Wallander's original model of adjustment (Wallander et al., 1989; Wallander & Varni, 1998) firstly proposes that each risk and resistance factor will exert a direct negative or positive

effect, respectively, on adjustment. Secondly, mediator effects are proposed in which some or all of the direct relationships between condition parameters and risk and resistance intrapersonal and social-ecological and factors and adjustment is mediated by the stress-processing factors. Thirdly, the model proposes moderator effects where the relationship between the risk factors and adjustment varies with, or is moderated by, levels of the resistance factors.

These hypothesised direct and indirect effects are explored more specifically in the introduction to the risk and resistance sections of this review. It is clear from the broad body of literature pertaining to risk-resistance approaches as well as that considering direct effects of risk factors on adjustment outcomes, that consideration must also be given to their association with the other risk and resistance factors. Consequently, this literature review considers the theoretical and empirical literature related to the direct effects of each risk factor on the proposed outcomes, their inter-relationships and indirect relationships between risk factors and outcomes. This chapter concludes with the identification of the research questions.

2.1.1 Selection of literature.

Extensive literature searches were conducted using the main psychological and medical research databases (PubMed, PsycINFO and Medline). A selection of keywords was used for each risk and resistance factor with each potential outcome, for example, pain severity, disability, pain-related interference, QOL, catastrophising, depression, anxiety, mediation, mediator, moderation, moderator. Related articles were also sourced using the reference lists of other studies. Manual searches of journal editions were completed where specific relevant topics were published in a single volume.

Articles were reviewed if the research included participants with any type of chronic, non-malignant pain diagnoses with a duration of more than three months. The majority of included

studies in this review are based on samples individuals with chronic musculoskeletal pain. Two types of research are predominant in the current review - cross-sectional and longitudinal or prospective cohort studies. This latter type of research explores the capacity of a predictor measured at an earlier time point to explain variance in an outcome measured at a later point. The majority of reviewed studies are cross-sectional, based on self-report measures of both predictors and outcomes. However, in a small number of studies the criterion variable for pain-related physical function was an actual physical test, such as a sit to stand or timed walk test. A number of meta-analyses and systematic reviews were included in the current review. Where these were referenced, the evidence from them is presented first in each relevant section. If a single study that was reviewed as part of a meta-analysis was subsequently reviewed individually, this was noted.

The predominant focus of this literature review is on clinical, not experimental or laboratory-based research. However, where results of a laboratory-based study clearly informed the findings of clinical research, these studies were also included. Where evidence for a predictor specific to the context of chronic non-malignant pain was lacking, the current research was guided by literature from the broader fields of chronic health conditions. Strong links are recognised to exist between many psychosocial risk and resistance factors and chronic pain outcomes and most studies included in the review were based on multivariate analyses that examined associations between a number of predictors and outcomes. For the purposes of this review, these factors were somewhat artificially separated and considered as predictors that acted independent of other influences.

2.1.2 Grading of research quality.

Multiple approaches exist for grading the quality of research evidence. The approach used to rate the constituent papers included in the systematic reviews and meta-analyses contained in

the current review varied. These were described where details of the adopted rating system were provided. Examples of rating systems used were the Quality in Prognosis Studies (QUIPS; Hayden, Cote & Bombardier, 2006), the Scottish Intercollegiate Guidelines Network methodology checklist for cohort studies (SIGN; Harbour, Lowe & Twaddle, 2011) and guidelines provided by the Cochrane Collaboration (Higgins & Altman, 2008) and the Jadad scale (Jadad, 1996). Generally speaking, grading scales assess the quality of research by checking sources of potential research bias across different research domains. Criteria include participation bias, measurement issues, consideration and control of confounding variables, approach to statistical analysis and how well the study addresses the research question. Studies are generally rated high, medium or low quality according to the criteria provided by the referenced grading system.

2.2 Risk Factors

The current model includes four categories of risk factors proposed to negatively influence adjustment to chronic pain. These are condition parameters, intrapersonal, social-ecological and stress-processing factors. In Wallander's original model (Wallander et al., 1989; Wallander & Varni, 1998) condition parameters refers to aspects of the actual condition that influence adjustment outcomes. In the current model, this category includes only perceived pain severity. Intrapersonal risk factors refer to aspects of personal experience that potentially contribute to worsened adjustment outcomes. Depression and anxiety are included in the current model in this category. Social-ecological factors are defined as the social and environmental factors that influence adjustment processes. Negative and solicitous spousal responses to pain have been linked to higher levels of pain-related disability (Leonard et al., 2006, Newton-John, 2002) and are thus placed in the current model as social-ecological risk factors. Stress-processing factors are defined as appraisals of circumstances and cognitive and behavioural efforts aimed at managing stressors.

Represented in this category are catastrophising and fear-avoidance. Referencing Wallander and Varni's original model (Wallander et al., 1989; Wallander & Varni, 1998), the current model proposes direct and indirect effects relevant to risk factors. Firstly, that risk factors act directly to negatively influence adjustment outcomes. Secondly, risk stress-processing factors are hypothesised to mediate relationships between pain, intrapersonal factors and social-ecological factors and adjustment outcomes.

2.2.1 Condition parameters - pain severity.

Substantial inter-individual variation is observed between perceived pain severity and physical function in those with chronic pain (Rainville, Ahern, Phalen, Childs & Sutherland, 1992). Aside from the pathology related to the pain, some psychological factors have been suggested to strongly influence pain perception. These include fearful pain appraisals, the meaning attributed to the pain (Arntz & Claassens, 2004) and anxiety and depression (Gore et al., 2005; Smedstad, Vaglum, Kvien & Moum, 1995). Because of the strong links noted between pain perception and psychosocial factors, debate exists as to whether pain severity is best seen as a predictor or outcome measure and it is considered in the literature from both perspectives. Despite this, substantial evidence shows that higher pain severity is strongly associated with poorer physical function and lowered QOL, even after adjusting for other closely linked variables such as negative affect (NA). This relationship has been observed in both community based and pain clinic samples. This literature is reviewed in the next sections. Pharmaceutical trials showing that reductions in pain severity produce substantial functional and QOL gains for those with chronic pain have provided further evidence of the influence of pain severity on physical function (Moore et al., 2010). For this reason and because it was of interest to explore indirect ways in which perceived pain severity may influence adjustment, pain severity was included in the current model as a risk factor.

2.2.1.1 Empirical studies of pain severity and physical function: direct effects in cross-sectional research.

Consistent with the hypothesis of the current model, studies using cross-sectional designs in community-based and primary care samples of people with chronic pain have repeatedly demonstrated that higher pain severity is linked with greater pain-related disability, although the reported strength of this association has varied. For example, in a large Irish prevalence study, Raftery and colleagues (2011) assessed predictors of pain-related interference in a heterogeneous sample of 438 adult patients of general medical practices with a mean age of 50 years. Pain-related interference was assessed with the Chronic Pain Grade Questionnaire (Smith et al., 1997) which measures both pain severity and pain-related interference in two subscales. Pain severity, depression and illness perceptions together explained 67% of variance in the measure of interference. Of the significant predictors, pain severity was the strongest.

A further study in a large heterogeneous sample of 2618 American primary care pain patients (Arnow et al., 2011) also reported strong links between pain severity and pain-related interference. Mean age of this sample was 55 years. Pain-related interference was assessed with the Chronic Pain Grade Questionnaire (Smith et al., 1997). Predictors in the regression model included pain severity, age, gender, depression and catastrophising. All predictors except age and gender were significantly associated with interference. Together, the model explained 51% of the variance in pain-related interference and pain severity displayed the strongest relationship with interference. Other research using cross-sectional data from primary care settings has replicated these findings in specific patient populations that have included whiplash associated disorders (Nieto et al., 2013), chronic low back pain (Hung, Liu & Fu, 2015) and idiopathic neck pain (Thompson, Urmston, Oldham & Woby, 2010).

Also demonstrating a robust relationship between pain severity and disability, Alcantara and colleagues (2010) explored predictors of pain-related disability in 115 workers with chronic musculoskeletal pain. Average age of this sample was 41 years and around half of the participants reported moderate severity pain. Predictors in the regression model included pain severity, pain beliefs and depression. Pain-related disability was measured by a Brazilian version of the Roland-Morris Questionnaire (Roland-Morris, 1983). In the multivariate analysis, pain severity was the strongest predictor of disability.

Pain has also been shown to predict physical function in older adults. For example, Chan, Hadjistavropoulos, Carleton and Hadjistavropoulos (2012) investigated predictors of pain-related interference in a community based heterogeneous sample of 109 older adults with a mean age of 76 years. Significant predictors of pain-related interference were pain severity, depression symptoms, coping self-statements and guarding. Of these predictors, pain severity demonstrated the strongest relationship with pain-related interference.

Similarly, using regression analysis, Silva, Queirós, Cerqueira and Rocha (2014) found that pain severity was a robust predictor of physical function in a community-based sample of 254 older adults. The majority of the sample (81%) reported pain, and of these, 84% reported a pain duration of more than six months. Disability was measured by the World Health Organisation Disability Schedule Version 2.0 (Ustun, Kostanjsek, Chatterji & Rehm, 2010). This scale provides a global assessment of functional impairment. Predictors included pain severity, age, sex, education, comorbid health conditions, depression and physical activity. Pain severity and frequency, depression, physical activity, education and chronic conditions together explained 44% of the variance in functional impairment. Of these predictors, pain severity was the strongest, explaining 27% of the variance in this outcome. Similar results were noted in a group of 204 community-based patients aged older than 50 years with musculoskeletal pain (Silva et al., 2013). In this

sample, pain severity was the strongest predictor of different aspects of physical function that including getting around, self-care, household activities and work, as well as total disability score.

Contrasting with the above results, a smaller number of studies based on cross-sectional data from primary care settings have reported a significant but less robust relationship between pain severity and disability. For example, Denison, Asenlof and Lindberg (2004) investigated relationships between self-efficacy, fear-avoidance, pain severity and pain-related disability in two primary health care samples of 210 and 161 participants with subacute, chronic or recurring musculoskeletal pain. Participants were people seeking treatment at physiotherapy departments within three primary health care units and one occupational health care organisation in rural Sweden. Mean ages of the two groups were similar (45 and 47 years) and median pain duration in both groups was 12 months. Approximately 37% of the participants in each group were on sick leave at the time the research was conducted. Pain severity explained a small but significant proportion of the variance in disability scores in one sample only. Pain self-efficacy and, to a lesser extent, fear-avoidance were both stronger predictors of pain-related disability. These authors postulated that these findings may have been attributable to the higher relative level of function of these samples. That is, in the setting of relatively low disability levels, cognitive predictors exerted a stronger influence on physical function than pain severity.

In contrast to findings from primary care or community-based samples of people with chronic pain, mixed results regarding the strength of the relationship between pain severity and functional impairment have been found in cross-sectional research using pain clinic samples. Providing evidence for pain severity as a robust predictor of disability in a pain clinic sample, Newton-John, Mason and Hunter (2014) examined pain severity, catastrophising, fear-avoidance and a measure of resilience as predictors of pain-related disability in a heterogeneous group of 101 individuals recruited from an Australian pain clinic with a mean age of 43 years, and mean pain

duration of 34 months. All predictors were significantly associated with disability, but the relationship was strongest for pain severity.

Contrasting with these results, Esteve, Ramırez-Maestre and Lopez-Martınez (2007) found that pain severity and pain acceptance explained pain-related functional impairment in a heterogeneous sample of 117 Spanish pain clinic patients, but pain acceptance was a stronger predictor. Structural equations modelling was used in this study to explore predictors of disability that included coping style, pain acceptance, catastrophising and resourcefulness beliefs. Similarly, Moix, Kovacs, Martín, Plana and Royuela (2011) examined the influence of pain severity, catastrophising, anger, anxiety, and depression on pain-related disability in 123 pain clinic patients with chronic low back pain. In the regression analysis, pain severity together with trait anxiety, previous failed back surgery and education level were significant predictors of disability, explaining 41% of the variance. However, trait anxiety was a stronger predictor of disability than pain severity. Consistent with the results, Nicholas, Coulston, Asghari and Malhi (2009) found, in a heterogeneous sample of 812 Australian pain clinic patients, that although pain severity was significantly associated with disability, pain self-efficacy and fear-avoidance were stronger predictors. Similarly, Crombez, Vlaeyen, Heuts and Lysens (1999) found that fear-avoidance, not pain severity, predicted pain-related disability in 104 patients with low back pain attending a pain rehabilitation centre.

The reasons why the strength of the association between pain and disability should differ between primary care and community based samples of pain patients and pain clinic samples remains unclear. It is possible this relationship varies according to pain duration although there is little evidence to support longer pain durations in pain clinic patients compared to community-based samples. For example, mean pain duration in Australian pain clinic normative data was approximately seven years (Nicholas, Asghari & Blyth, 2008). This compared to an average pain

duration of five and eight years respectively reported in two community-based samples (Alcantara et al., 2010; Raftery et al., 2011).

Another possible explanation may be the clinical characteristics of the samples. Patients tend to be referred to specialist pain clinics when their function is especially adversely affected (Turk & Okifuji, 2002). Therefore, pain clinic samples likely represent individuals who are particularly disabled by pain. Pain clinic data supports this assertion. For example, normative data obtained from a sample of 5941 patients attending an Australian pain management unit (Nicholas et al., 2008) demonstrated that pain clinic patients report high levels of pain severity, functional impairment and psychological distress.

Research that has explored differences between pain clinic and community-based individuals with pain also supports these contentions. For example, Kung, Gibson and Helme (2000) compared a sample of 150 pain clinic patients to 193 community-based older adults with chronic pain and found that pain clinic patients were more likely to have pain related to non-musculoskeletal conditions, a pain history of less than two years and high ratings of pain intensity and depression. Similarly, Crook, Tunks, Rideout and Browne (1986) found that demographic variables and length of pain history were similar in pain clinic and primary care patients, but pain clinic patients were more likely to have had work-related accidents, experience more constant pain and report higher levels of disability and psychosocial distress. The generally weaker relationship between pain severity and physical function in pain clinic samples suggests that when psychological distress and fearful pain appraisals are high, that these psychological factors may exert a stronger influence on physical function than pain itself.

2.2.1.2 Empirical studies of pain severity and physical function: direct effects in longitudinal research.

Also supporting the hypothesis of the current model that pain severity is linked to physical function in those with chronic pain, a large volume of longitudinal research has reported strong associations between pain severity measured at the time of pain onset with later disability. For example, in a sample of 488 primary care patients with low back pain, higher pain severity at an initial medical visit conferred a 12% increase in risk for poor functional outcome six months later (Campbell, Foster, Thomas & Dunn, 2013). Similar results were reported by Sieben and colleagues (2005) who found that, in 222 primary care patients with acute onset low back pain, pain severity, a previous history of low back pain and NA were all significant predictors of later disability.

Grotle, Foster, Dunn and Croft (2010) similarly found that early pain severity levels predicted later disability in primary care patients. The sample comprised two different groups; the first consisted of 258 individuals with acute or subacute low back pain. The second was 668 individuals with chronic low back pain. All participants were followed up 12 months after an initial assessment and prognostic indicators of disability were compared between groups. At baseline the chronic back pain group reported significantly higher levels of pain, catastrophising, anxiety and depression compared to the acute/subacute group. A significantly higher proportion of the chronic group (23%) also reported work absences due to back pain compared to the acute pain group (15%). Despite these differences, multivariate regression models showed that predictors of disability at 12 months were similar in both groups. Specifically, these predictors were not working, higher pain levels and catastrophising. The only between-groups difference noted was that fear-avoidance was a significant predictor of disability in the chronic but not acute back pain group. The strength of the association between pain severity and disability was stronger

than that observed for catastrophising, suggesting that earlier stage pain severity may be a more important predictor of later disability than the pain appraisal processes.

Much of the longitudinal research investigating pain severity as a predictor of later functional impairment has been conducted in samples of injured workers. Turner, Franklin and Turk (2000) conducted a systematic review of this research. Twenty studies of both longitudinal and cross-sectional design were included. No formal quality rating of included studies was reported. Commonly examined endpoints were presence or absence of a work disability and time to return to work. The most consistent predictors of later disability were pain severity and initial disability levels. However the authors cautioned that several factors, such as variability in the statistical methods and measures may have reduced the reliability of the results. Similar results were reported by Walton et al. (2013) in a meta-analysis of predictors of later stage functional impairment and pain in people with whiplash-associated disorders. Variables robust to publication bias that were significantly associated with poorer later stage outcome were high initial pain severity, disability scores and grade of injury, being female, headache at time of injury and lower education levels.

A further systematic review of predictors of outcomes in people with low back pain also supported a link between earlier stage pain severity and return-to-work rates (Verkerk, Luijsterburg, Miedema, Pool-Goudzwaard & Koes, 2012). Fourteen studies were included with methodological quality assessed by the Quality in Prognosis Studies (QUIPS) (Hayden et al., 2006). Methodological issues included lack of information about non-responders and lack of accounting for measurement confounds. Of the included studies, eight were prospective cohort studies. The remainder were randomised controlled trials, case series and correlational designs. No association was found between early pain severity and later disability, but in two of three studies

that were rated as high quality, limited evidence supported links between lower baseline pain and improved return-to-work rates.

2.2.1.3 Empirical studies of pain severity and quality of life: direct effects in cross-sectional research.

Higher pain severity has also been linked to poorer QOL in cross-sectional research using heterogeneous samples of individuals with chronic pain. For example, Merlijn and colleagues (2006) assessed links between pain and QOL in a community-based sample of 194 adolescents with chronic pain. Quality of life was measured with the Quality of Life Questionnaire for Adolescents with Chronic Pain (Merlijn et al., 2002) which consists of six domains; Psychological Functioning, Functional Status, Physical Status, Social Functioning, Satisfaction with Life in General, and Satisfaction with Health. Pain severity was negatively correlated with all QOL domains at moderate strength except for social functioning and was a significant predictor of physical QOL in regression models. However, in most regression models, a measure of perceived vulnerability was a stronger predictor of QOL than pain severity.

Similarly, Lamé, Peters, Vlaeyen, Kleefand and Patijn (2005) investigated the relative influence of pain severity and pain beliefs (coping and catastrophising) on QOL in a heterogeneous group of 1208 chronic pain patients of a multidisciplinary pain treatment centre. Health-related QOL was measured with the Dutch version of the Short Form-36 (SF-36; Ware, Snow, Kosinkio & Gandek, 1993). The SF-36 contains 36 items, measuring eight domains of HRQOL, physical, social, role limitations physical, role limitations emotional, mental health, vitality, bodily pain and general health perception. Pain severity was a significant predictor of all eight domains of HRQOL, although the strength of its association with these domains was less than that observed for catastrophising, age and education. An association between pain severity and QOL have also been demonstrated in patients with fibromyalgia. Moore et al. (2010) pooled data from four separate

studies to investigate the relationship between changes in pain severity and QOL. Total sample size was 2575. Results indicated that reductions in pain severity of between 50 and 30 % were associated with statistically significant improvements in QOL.

In a study of 290 Iranian pain clinic patients, Yazdi-Ravandi and colleagues (2013) found that, together with coping self-efficacy, pain intensity was a significant predictor of QOL. Quality of life was assessed using the World Health Organization WHOQOL-BREF (EuroQol Group, 1990) which provides scores across four domains - physical, psychological, social and environmental. Pain intensity was a significant predictor only of the physical and psychological QOL subscales but in these models was a stronger predictor than coping self-efficacy.

Partly contrasting with these results, Wahl and colleagues (2009) examined the relationship between chronic pain and QOL in a community-based sample of people with chronic pain using a Norwegian version of the Quality of Life Scale (QOLS; Burckhardt, & Anderson, 2003) which is composed of 16 items across varying domains. A total scale score provides an indication of respondents' overall satisfaction with QOL. Chronic pain was strongly significantly associated with lowered QOL in bivariate analyses, but was not significantly associated with QOL in multivariate regression models once other variables such as stress-related symptoms, fatigue, and subjective health were adjusted for. These latter factors all showed highly significant, direct associations with QOL.

Reporting results consistent with those of Wahl and colleagues (2009), Lee, Chronister and Bishop (2008) explored pain severity and other psychosocial factors as predictors of QOL in a sample of 171 Canadian patients of pain rehabilitation centres. Quality of life was measured with the WHOQOL-BREF (EuroQol Group, 1990). In regression models pain severity was only weakly associated with lowered QOL in each of the four domains and was not a significant predictor of overall QOL. The strongest predictors for overall QOL were depression and coping approaches.

2.2.1.4 Empirical studies of pain severity and quality of life: direct effects in longitudinal research.

Providing further support for the model hypothesis that greater pain severity would be directly linked to poorer QOL, pain severity has also been demonstrated to predict later QOL in longitudinal research. For example, Nolet and colleagues (2014, 2015) investigated the associations between baseline pain severity in the neck and low back with HRQOL measured six months later in 1100 Canadian adults. Health-related QOL was measured by the SF-36 (Ware et al., 1993). In both studies, the chances of worsened perceptions of physical health six months later were significantly higher for those with neck pain compared to those without. A dose relationship was also identified in which the risk of a worsened HRQOL outcome worsened as perceived pain increased.

2.2.1.5 Summary of evidence pertaining to pain severity.

The contention of the current model is that pain severity will be directly associated with pain adjustment outcomes is supported by the literature. Pain severity is represented in the literature as both a predictor and an outcome measure and strong links are known to exist between pain severity and other risk factors for adjustment such as catastrophising and NA. In summary, a large volume of research has demonstrated consistently strong links between pain severity and pain-related physical function. These links are evident in cross-sectional as well as longitudinal research, in samples of different ages and in community-based, primary care and pain clinic samples. The strength of the association between pain severity and pain-related function in pain clinic samples appears to be less robust that than observed in community-based and primary care samples. The exact reasons for this difference are unclear but may relate to sample characteristics. Evidence from multiple single studies also exists to support a negative relationship between pain severity and QOL but the evidence base is comparatively small.

2.2.2 Intrapersonal factors – depression.

Depression is a common comorbid condition with chronic pain. The prevalence of major depression in individuals with chronic pain is estimated to range between 20 and 54% which is substantially higher than in the general population (Campbell, Clauw & Keefe, 2003; Wilson, Eriksson, D'Eon, Mikail & Emery, 2002). A meta-analysis of psychological problems commonly experienced by individuals with chronic pain reported a large effect size for the difference in level of depression symptoms for those with pain compared to healthy controls (Burke et al., 2015) indicating that symptoms of depression in the pain groups were consistently higher than the non-pain affected control groups.

There is some evidence to suggest that depression can precede chronic pain, be a consequence of it, and is also a biological relative (Dersh, Polatin & Gatchel, 2001). Accordingly, depression is also considered in the research both as a risk predictor and as an outcome measure. Fishbain and colleagues (1997) summarised a number of theories that have attempted to explain links observed between depression and pain-related disability. The antecedent hypothesis suggests that depression precedes pain and the consequence hypothesis that depression is a consequence of pain (Fishbain, Cutler, Rosomoff & Rosomoff, 1997). The scar hypothesis (Cadoret, Winokur, Langbehn, Troughton, Yates & Stewart, 1996) suggests that some people have a genetic predisposition to recurrent depression and that depressive episodes experienced before the onset of chronic pain predispose to depression in the setting of chronic pain. The common pathogenetic mechanisms hypothesis suggests that depression and pain share similar biological underpinnings. The cognitive-behavioral mediation model (Magni, Moreschi, Rigatti-Luchini & Merskey, 1994) hypothesises that pain appraisals such as decreased self-control mediate the relationship between pain and depression.

While a comprehensive review of the above theories is beyond the scope of this thesis, there is evidence to support all of them. In their literature review of 82 related studies, Fishbain and colleagues (1997) concluded that the majority of studies supported both the scar hypothesis and the cognitive mediation model. That is, previous episodes of depression can predispose to further depression in the setting of chronic pain and that these episodes are likely mediated by negative pain appraisals. A substantial volume of research has also supported the idea both pain and depression may be mediated by similar neurochemical mechanisms such as serotonin, norepinephrine, substance P and corticotrophin-releasing factor (Campbell et al., 2003; Fishbain et al; 1997). Shackman and colleagues (2011) presented evidence of anatomical links between pain and depression and suggested that nociceptive and affective pathways coincide anatomically. Further support for the idea of common biological pathways mediating pain and depression can be found in evidence that antidepressants have been found to be effective for the treatment of some forms of chronic pain (Dersh et al., 2001).

Depression is also thought to produce a deficit of pain inhibition via contribution to dysfunction of the endogenous pain inhibitory system. This hypothesis was confirmed in a study of 32 patients with fibromyalgia by Ang and colleagues (2011) that investigated depression as a moderator of the relationship between pain and a measure of central sensitisation called the spinal nociceptive flexion reflex (NFR). The NFR response time is an indicator of the degree of pain required to stimulate a central nervous system response and has been reported to be lower in people with fibromyalgia due to abnormalities in central pain processing (Staud & Domingo, 2001). Patients participated in an invoked pain trial three times over 12 weeks. Ang and colleagues (2011) hypothesised that individuals with fibromyalgia and depression compared to those without depression would exhibit significantly lowered NFR thresholds. Their results supported this hypothesis. This suggests that depression may reduce pain inhibition, and may contribute to pain-

related disability indirectly via this pathway. Depression is placed in the current model as a risk factor because a substantial volume of literature shows that depression is associated with poorer physical function and reduced QOL in those with chronic pain although the likelihood of a bi-directional relationship between these two factors is acknowledged.

2.2.2.1 Empirical studies of depression and physical function: direct effects in cross-sectional research.

Multiple studies have demonstrated strong links between depression and pain-related disability. These findings have been reported in pain clinic, primary care and community-based samples of individuals with chronic pain. Bair, Robinson, Katon and Kroenke (2003) reviewed the literature pertaining to the relationship between depression and chronic pain outcomes. Six studies included in the review assessed the relationship between depression and functional outcomes, of these three using community-based or primary care samples were cross-sectional in design. All three studies identified a significant association between higher levels of depression and poorer functional outcomes. Depression has also been shown to be a significant predictor of pain-related disability in a community-based of 428 adults with chronic pain of mixed diagnoses (Raftery et al., 2011).

More recent research has reported similar findings. For example, in 225 pain clinic patients with chronic low back pain depression was reported to explain the highest proportion of variance a measure of pain-related disability in a regression model that also included pain severity (Nicholas et al., 2009). Borsbo, Peolsson and Gerdle (2009) identified significant links between depression and elevated levels of pain-related disability in 433 rehabilitation patients with chronic pain of mixed diagnoses. Scores on validated measures of depression, catastrophising, pain intensity and pain duration were used to classify participants into four clusters. Higher scores on measures of both depression and catastrophising were associated with significantly higher pain-related

disability. In a regression analysis, depression scores displayed the strongest positive relationship with pain-related disability.

Similar links have been reported in research from primary care and community-based settings. For example, in a sample of 2618 primary care patients, Arnow and colleagues (2011) found that depression predicted pain-related interference. This study used a regression-based approach and their model adjusted for age, gender and pain intensity. Both depression and catastrophising were uniquely and independently related to interference. Similarly, Bair, Wu, Damush, Sutherland and Kroenke (2008) noted significant links between depression and poorer function in 500 primary care patients with chronic musculoskeletal disorders. Scores on validated measures of depression and anxiety were used to dichotomize participants as having or not having clinical depression and anxiety. Participants were then classified into groups according to the presence or absence of pain, depression, and/or anxiety. Significantly greater pain interference was noted for individuals with depression or both depression and anxiety compared to participants who did not meet criteria for these conditions. Depression has also been shown to be a significant predictor of pain-related disability in a community-based of 428 adults with chronic pain of mixed diagnoses (Raftery et al., 2011).

2.2.2.2 Empirical studies of depression and physical function: direct effects in longitudinal research.

Depression has been shown to predict poorer pain-related function in longitudinal research. For example, depression was identified as the strongest predictor of disability in a systematic review of prospective research examining psychological factors associated with chronicity in low back pain (Pincus et al., 2002). Twenty-five studies were included, only six of which met the authors' criteria for acceptable methodology. Study quality was rated according to a number of criteria including exclusion and inclusion criteria, dropout rates, quality of psychological

measurement, and statistical considerations, such as sample size and use of statistical tests. The majority of included studies were based on pain clinic samples, however a small number of studies used primary care data. Outcomes included disability, return to work and sick leave taken. Psychological distress was a significant predictor of poorer functional outcome, especially in primary care samples. This finding was made on the basis of two high and two acceptable quality studies and the effect was independent of other predictors, such as pain and function at baseline.

A large American national health survey also demonstrated a longitudinal association between depression and pain-related disability. The initial sample size in this study consisted of 91,347 individuals with chronic pain, with 55,690 of participants followed up at two years (Meyer, Cooper & Raspe, 2007). Participants were randomly selected primary care patients of American health management organisations. Depression symptoms at baseline increased the odds of reporting pain-related disability two years later and these findings were independent of socio-demographic characteristics and baseline medical and functional status. Demonstrating the bi-directional nature of the relationship between depression and pain-related disability, this study also found that disabling low back pain at baseline increased the odds of depressive symptoms after two years to a similar degree.

Linton's (2000) review of prospective research reported Level A evidence for depression as risk factor for the development of poorer outcomes in people with neck and back pain. Thirty-seven studies were included; Level A evidence was concluded where at least two good quality prospective studies supported the findings. A range of outcomes were included such as return to work and perceived pain severity. Four studies examined the outcome of pain-related disability; of these, three reported significant links between earlier depression and the development of later pain-related disability.

In an effort to better understand the temporal relationships between depression, anxiety and pain-related disability, Lerman, Rudich, Brill, Shalev and Shahar (2015) asked 428 people with chronic pain of mixed diagnoses recruited from two pain clinics to complete measures for depression, state anxiety and pain-related disability at four different times, with an average of five months between. They used a cross-lagged, structural equation modelling approach to test the longitudinal associations between the variables. Earlier levels of depression and anxiety significantly predicted later pain-related disability and pain. However, neither pain nor pain-related disability significantly predicted depression or anxiety, suggesting that depression and anxiety may worsen pain-related disability but that disability does not contribute to depression and anxiety in those with chronic pain.

Two other studies adopting longitudinal designs that identified a cumulative risk associated with depression and catastrophising provided support for these constructs as distinct risk factors. Bergbom and colleagues (2011) employed cluster analysis to explore psychological profiles as predictors of disability in 297 physiotherapy patients recruited from primary care settings. Patients had musculoskeletal pain at varying body sites and one third of the sample reported a pain duration of less than three months. Measures were taken at treatment outset and were repeated six months later. Participants with high depression and catastrophising reported significantly higher levels of disability than those in the lower risk clusters and those in intermediate risk clusters reported more disability than those in the low-risk cluster.

Researchers from the same group (Linton et al., 2011) provided further evidence for a cumulative risk associated with elevated rates of both depression and catastrophising. Two groups of patients comprised the study sample. All had sub-acute, not chronic pain at the time of the initial measures and were followed up one year later. A Swedish sample had varying musculoskeletal pain problems, the second sample were Australian physiotherapy patients with an episode of low

back pain. Total sample size comprised 632 individuals. Participants were classified according to their catastrophising and depression scores into four clusters; high catastrophising, high depression, low catastrophising, high depression, high catastrophising, low depression and low catastrophising, low depression. Odds ratios were calculated to estimate the likelihood of reporting higher depression and greater functional impairment scores at 1 year follow-up. Results indicated that the high depression groups at baseline had significantly increased risk for poorer function at follow-up compared to the low depression groups. Additionally, when high catastrophising was added as a risk factor the chance of a poorer functional outcomes significantly increased. These results highlight that depression and catastrophising represent independent risk factors for poorer functional outcome for those with chronic musculoskeletal pain.

Some research has identified a bidirectional relationship between depression and pain-related disability. For example, Arola, Nicholls, Mallen and Thomas (2010) explored temporal relationships between depression and pain-related interference in 4234 English adults with osteoarthritis who completed an initial health survey as well as a follow up three years later. Depression and anxiety at baseline predicted new onset pain-related interference at three years, but pain interference at baseline also increased risk for depression and anxiety three years later suggesting a bidirectional relationship between depression and disability.

Other research in primary care settings has failed to identify prior depression as a risk factor in multivariate models predicting pain-related function. In a large sample of 1591 English primary care patients, Foster, Thomas, Bishop, Dunn and Main (2010) analysed predictors of pain-related disability from measures taken at an initial consultation. Eight hundred and ten patients also provided data six months later. Less than one quarter of the sample had chronic pain at presentation. Disability was measured with the Roland-Morris Disability Questionnaire (Roland & Morris, 1983). Mean disability levels at baseline were approximately a third of the maximum scale score.

Various predictors of outcome were measured that included depression and anxiety, catastrophising, pain self-efficacy and illness beliefs. Moderate positive bivariate associations between depression and disability were noted but in a final regression model depression was not a significant predictor of disability measured at six months. Instead pain duration at presentation, illness identity, personal control and pain self-efficacy predicted disability. These results do not discount that a relationship was still identified between depression and later disability. They may perhaps be explained by the high proportion of participants with acute pain on initial presentation or by the inclusion of other measures such as illness perceptions in the predictive model.

Lastly, conflicting evidence for the relationship between depression and later pain-related function were reported in a recent systematic review that explored predictors of treatment outcome in fibromyalgia and chronic widespread pain. Only weak evidence supported that prior depression predicted poorer post treatment physical function (de Rooij et al., 2013). Fourteen studies were included in this review, most of which used samples from hospital based fibromyalgia treatment clinics. In order to be included studies had to have a longitudinal design with at least one follow up measurement obtained following a multidisciplinary clinical interventions with at least two treatment components. Study quality was assessed according to the Hayden criteria (Hayden et al., 2006) which assess five areas of potential bias - participation, attrition, measurement, confounding and analysis. The risk of bias is rated as low, moderate or high. Six studies were considered to be of high quality and eight were rated low quality. Due to the heterogeneity of included studies in terms of design and inclusion of predictors, treatment and outcomes, pooling of data to estimate effect sizes was not possible. Instead the authors reported they conducted a qualitative data synthesis instead. It is possible these finding may be attributable to the specific diagnostic profile of the included studies.

2.2.2.3 Empirical studies of depression and quality of life: direct effects in cross-sectional research.

Borsbo and colleagues (2009), described above, also reported significant links between depression and poorer overall QOL in 433 rehabilitation patients with chronic pain of mixed diagnoses. Quality of Life was measured with the QOLS (Burckhardt, Archenholtz & Bjelle, 1992). Higher scores on measures of both depression and catastrophising were associated with significantly lower QOL. In a regression analysis, depression scores displayed the strongest negative relationship with QOL.

Much of the research exploring links between depression and QOL has examined not overall QOL but HRQOL as the outcome, with several studies noting negative associations. For example, Outcalt and colleagues (2015) found depression was independently negatively associated with both HRQOL and disability in a sample of 250 Veterans Affairs primary care patients with moderate to severe chronic musculoskeletal pain. Multiple measures of HRQOL were used in this study and significant negative associations between depression and all measured domains. Scholich, Hallner, Wittenberg, Hasenbring & Rusu (2012) also reported moderate strength negative correlations between depression and HRQOL in 52 patients with chronic pain recruited from a German orthopaedic clinic.

Negative associations between depression and HRQOL were reported by Elliott, Renier and Palcher (2003) who examined the relationship between depression and HRQOL in a group of 242 heterogeneous pain clinic patients with a mean age of 46 years. Depression was assessed in an interview and categorised as major, mild or no depression according to number of depression symptoms identified in the Diagnostic and Statistical Manual, Fourth Edition (American Psychiatric Association, 1994). Health-related QOL was measured with the Short-Form 36 (SF 36; Ware, Snow, Kosinkio & Gandek, 1993) which assesses eight domains that include physical and

social function and vitality. No significant differences were in physical component score of the SF-36 for those with major depression compared to those without major depression. However, individuals with major depression reported significantly lower mental component scores than those with mild or no depression. Similarly, in a sample of 451 women with fibromyalgia, Soriano-Maldonado and colleagues (2015) also identified links between depression and HRQOL which was measured with the SF-36 (Ware et al., 1993). Individuals with severe depression reported significantly poorer mental component scores compared to those with mild depression but no association between depression and the physical component score was noted. No other research was identified that examined depression as a predictor of overall QOL in mixed samples of pain patients, potentially because of a potential overlap with affective subscales of QOL measures.

2.2.2.4 Summary of evidence pertaining to depression.

The above evidence supports the inclusion in the current model of depression as a risk factor for pain adjustment as the literature demonstrates depression is consistently associated with greater functional impairment. A smaller amount of research was identified that has demonstrated negative links exist between depression and QOL or HRQOL for those with chronic pain. Although results of one study refuted the idea of a bi-directional relationship between depression and pain-related disability, other research has suggested a two-way relationship exists between these two factors.

2.2.3 Intrapersonal factors – anxiety.

Anxiety is proposed in the current model to be directly associated with higher pain-related disability and lower QOL as well as indirectly linked with these outcomes via stress-processing factors. Anxiety disorders, such as generalized anxiety disorder, post-traumatic stress disorder, panic disorder and social anxiety disorder (Asmundson & Katz, 2009) are frequently seen as co-morbid conditions with chronic pain. Prevalence rates for anxiety disorders are reported to be

around 33% in those with chronic pain compared to 17% in the general population (Asmundson & Katz, 2009), although Kroenke and colleagues (2013) reported a rate of 45% in a heterogeneous sample of 250 primary care patients with chronic musculoskeletal pain. Two main hypotheses have been suggested to explain the elevated anxiety levels in chronic pain (Asmundson & Katz, 2009). The first suggests that the physiological, affective or behavioural components of anxiety and pain mutually reinforce the other. The second holds that, as for depression, some of these maintenance factors share a common vulnerability factor, possibly genetically influenced (Asmundson & Katz, 2009).

There are several factors associated with the experience of anxiety that directly impact on the experience of chronic pain. Heightened anxiety is associated with cognitive biases such as increased attention to threat related stimuli and the tendency to interpret ambiguous stimuli as threatening (Jordan & Okifiji, 2011). Anxiety can produce increased physiological arousal and muscle tension, which can increase pain states (Turk & Okifuji, 2002) and lead to activity avoidance (Gatchel et al., 2007). It has been suggested that one possible mechanism of co-occurrence is anxiety sensitivity (Greenberg & Burns, 2003), which is associated with higher disability and pain severity levels in chronic pain (McCracken & Gross, 1998). Anxiety sensitivity is linked with increased fear-avoidance beliefs and a greater tendency to negatively interpret bodily sensations (Keogh, Hamid, Hamid & Ellery, 2004), which in turn further contribute to pain-related disability. This highlights the reciprocity of the relationships between affect and pain appraisal both of which are strongly linked to pain-related function.

Anxiety may also affect pain-related physical function indirectly, via its influence on other risk predictors. For example, anxiety has been linked to increased pain levels. A meta-analysis that examined the relationships between pre-operative anxiety, catastrophising and chronic post-surgical pain included 25 studies. Of the included research that examined outcomes in

musculoskeletal surgery, 67% reported significant associations between anxiety and post-surgical pain (Theunissen, Peters, Bruce, Gramke & Marcus, 2012). However, some caution was suggested in interpretation of these results due to the heterogeneity of measurement instruments used.

2.2.3.1 Empirical studies of anxiety and physical function: direct effects in cross-sectional research.

Less research attention has been paid to the influence of elevated anxiety levels on pain outcomes compared to research that has investigated depression. This may be partly because pain research has largely focused instead on the influence on pain outcomes of other anxiety-related constructs such as catastrophising and fear-avoidance. Nonetheless, some evidence exists to demonstrate significant positive links between increased anxiety and pain-related disability in those with chronic low back pain. For example, McWilliams, Cox and Enns (2003) explored links between anxiety disorders and chronic pain in a large American sample (n=5877). Participants completed a diagnostic interview of mood disorders that also provided self-reports of pain and disability. The presence of more than one anxiety disorder was significantly associated with increased pain-related disability.

Multiple other studies have examined state or trait anxiety levels as multivariate predictors of pain-related disability with mixed results reported. For example, Moix and colleagues (2011) investigated pain severity, trait and state anxiety, catastrophising, anger, depression and demographic variables as multivariate predictors of pain-related disability in 122 pain clinic patients with low back pain. Their sample was predominantly female and had a mean age of 50.4 years. Trait and state anxiety were measured with the State Trait Anxiety Inventory, which is a questionnaire composed of two subscales that measure state and trait anxiety (Spielberger, 1985). Together pain severity, state anxiety, previous failed back surgery and higher education levels

predicted 49% of the variance in disability. State anxiety was the predictor most strongly associated with disability.

Contrasting with these results, Meredith, Strong and Feeney (2008) found anxiety was not a significant predictor of pain-related disability in a regression analysis that also included perceived pain severity and pain self-efficacy as predictors. The study sample comprised a heterogeneous sample of 132 pain clinic patients with a mean age of 38.81 years. Anxiety levels were measured by the Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995). In this model, both pain self-efficacy and, to a lesser extent, pain severity predicted disability.

Sullivan, Stanish, Waite, Sullivan and Tripp (1998) also examined multivariate prediction of pain-related disability in a regression model that included catastrophising, anxiety and depression as predictors. The sample included 86 pain clinic patients with a mean age of 36 years. Anxiety was measured by the State-Trait Anxiety Inventory (Spielberger, 1985). Anxiety and depression did not contribute unique variance to the prediction of disability over and above that explained by catastrophising suggesting a degree of construct overlap between these variables.

2.2.3.2 Empirical studies of anxiety and physical function: direct effects in longitudinal research.

The question of whether anxiety causes pain-related disability or vice versa was partly addressed by the cross-lagged structural equations modelling investigation described in the above section addressing the relationship between depression and physical function (Lerman et al., 2015). This study used a heterogeneous group of pain patients to test the longitudinal associations between the variables and found that earlier levels of anxiety significantly predicted later pain-related disability and pain, but neither pain nor pain-related disability significantly predicted depression or anxiety.

2.2.3.3 Empirical studies of anxiety and quality of life: direct effects in cross-sectional research.

Cross-sectional research has demonstrated significant links between higher anxiety levels and poorer QOL in those with chronic pain. Kroenke and colleagues (2013) examined the association between anxiety disorders and QOL outcomes in 250 primary care patients with moderate to severe musculoskeletal pain. The sample had a mean age of approximately 55 years and almost half of the participants reported at least one anxiety disorder. Compared to the patients not meeting criteria for an anxiety disorder, screen positive patients reported significantly worse scores for all domains of HRQOL and these scores progressively worsened as the number of anxiety conditions increased.

Scholich and colleagues (2012) also reported a robust negative relationship between anxiety and HRQOL in a study of 52 patients of an orthopaedic inpatient unit with chronic low back pain. Correlations between trait anxiety and other predictors of adjustment such as depression, catastrophising and avoidance behaviours and HRQOL were examined. Measures were made at two time points in the patients' care trajectory. The first was at the time of onset of an acute exacerbation of pain; the second occurred six months later. Of all predictors, trait anxiety demonstrated the strongest correlation with HRQOL at both assessment times.

Similar results were reported by Keeley and colleagues (2008). These authors examined predictors of HRQOL in a sample of 93 patients. Average age of this sample was approximately 40 years. Predictors included depression, anxiety, social stresses and fear-avoidance beliefs. Participants were individuals with chronic low back pain who had been recently referred to an orthopaedic outpatient clinic. Participants were assessed at the time of initial referral and then again six months later. Both anxiety and depression in this sample were significantly associated with HRQOL at six months, although the strength of this association was greater for depression than

anxiety. A combined anxiety and depression score, together with fear-avoidance beliefs and social stresses explained 55% of the variance in HRQOL at six months after initial assessment.

2.2.3.4 Summary of evidence pertaining to anxiety.

Compared to depression, less research has investigated the links between anxiety and chronic pain outcomes. Despite this, there is consistent evidence that prevalence rates for anxiety disorders in those with chronic pain are significantly elevated and that heightened anxiety is associated with poorer functional outcomes and lowered HRQOL. This may be in part due to a stronger tendency for anxious individuals to experience cognitive pain-related biases such as increased attention to threat-related stimuli. Anxiety may also influence pain outcomes indirectly by increasing perceived pain severity. It is possible that, like depression, anxiety and chronic pain may share some common maintaining mechanisms, one of which is suggested to be a genetic predisposition towards anxiety sensitivity. Some of the conflicting findings in relation to anxiety and pain-related disability may relate to variations in anxiety assessment tools or sample variability. It appears likely that some degree of statistical overlap exists between measures of anxiety, depression and catastrophising.

2.2.4 Social-ecological risk factors - partner responses to pain.

In qualitative chronic pain research a range of negative social experiences has been reported to be associated with chronic pain including negative partner and family responses to pain and invalidation (Bunzli, Watkins, Smith, Schütze & O'Sullivan, 2013; Osborn & Smith, 1998; Silva, Sampaio, Mancini, Luz & Alcântara, 2011; Smith & Osborn, 2007; Snelgrove & Liossi, 2013; Warwick, Joseph, Cordle & Ashworth, 2004). The quantitative research related to negative social interactions in the context of chronic pain has focused mainly on the influence of negative partner

responses to pain. The influence of these experiences on pain adjustment outcomes have been explored in a large body of research with both negative and positive effects reported.

The spousal responses to pain research sits within the broader field of literature that has examined dyadic coping responses to chronic health conditions. While it is beyond the scope of this review to explore this literature in its entirety, as a whole it has indicated that marriage in itself confers substantial health benefits, with lowered mortality and morbidity rates reported for married compared to non-married individuals (Kiecolt-Glaser & Newton, 2001). It is unclear whether these differences can be attributed to a selection bias, whereby healthier individuals tend to get married, whether it is marriage itself that is associated with healthier lifestyle choices, or a combination of the two (Newton-John, 2013). It has also been suggested that it is not marriage per se that confers health benefits but more specifically, the nature of the partner relationship that may be associated with benefit or disadvantage. For example, improved adjustment outcomes for those affected by chronic health conditions have been linked to supportive and collaborative spousal responses rather than overprotective, hostile or dismissive ones (Berg & Upchurch, 2007; Newton-John, 2013).

The research that has specifically explored the relationship between couples' interactions and adjustment to chronic pain has largely considered these interactions from two theoretical perspectives - operant and cognitive behavioural (Leonard et al; 2006). The operant perspective suggests that expressions of pain or pain behaviours are either reinforced or punished by partners (Fordyce, Fowler & DeLateur, 1968). In a review of the literature pertaining to partner responses to pain, Newton-John (2002) noted that according to behavioural theory, spousal reinforcement of pain behaviours can occur across three domains; positive and negative reinforcement of pain behavior and insufficient reinforcement or discouragement of well behaviours. The cognitive-behavioural perspective proposes that the responses of a person living with pain may be influenced by their spouses', friends' or family's beliefs about pain (Cano, Miller & Loree, 2009; Leonard et

al; 2006). This perspective also emphasises that spouses are automatically interpreting each other's behaviour during interactions and these interpretations influence both future reciprocal behaviours and the emotional nature of the relationship (Newton-John, 2002).

In his review of literature, Newton-John (2002) noted that research and clinical work relating to solicitous (overly supportive) partner responses to pain has been heavily influenced by use of the Multidimensional Pain Inventory (MPI; Kerns, Turk & Rudy, 1985) which has been the predominant self-report tool used to assess spousal responses. This scale classifies spousal responses to pain behaviours into either solicitous, punishing (negative) or distracting. Newton-John (2002) stated that even when alternative assessment methods were used, such as diary recording, the same approach was adopted to classify these interactions. Newton-John (2002) summarized several limitations in much of the research up to this point. Firstly he noted that there are issues with the operationalization of the construct of solicitous responses as it is hard to define a priori which behaviours will be perceived or will function in this manner. Secondly, Newton-John (2002) highlighted that use of the MPI (Kerns et al., 1985) has restricted analysis of couple interactions to three responses, when in reality a spouse may react in a multitude of ways to their partner's pain and this may vary according to circumstance. Lastly, Newton-John (2002) highlighted that other contextual factors may influence the perception and impact of such responses and these factors vary across couple dyads. For example, marital satisfaction has also been reported to moderate the relationship between solicitous spouse responses and disability (Turk, Kerns & Rosenberg, 1992).

More recently, Cano and Williams (2010) suggested that current understanding of the influence of spousal responses on pain adjustment outcomes may be enhanced by adopting other perspectives such as Reis and Shaver's (1988) interpersonal process model of intimacy. According to this model, intimacy in couples develops when emotional self-disclosure of one partner is met

with an empathic responses from the other. Reis and Shaver's (1988) model proposes that interactions characterised by hostility produce feelings of rejection and these disrupt emotion regulation, thus that punishing spousal responses may be linked to higher levels of NA. Although this is not hypothesised by the current model, the idea that negative social exchanges may influence pain adjustment indirectly via NA represent a potential avenue for future pain research.

Solicitous and punishing spousal responses have been linked with other psychosocial and pain-related factors. For example, punishing spousal responses are associated with greater perceived pain severity (Cano, Gillis, Geisser & Foran, 2004; Flor, Turk & Rudy, 1989; Leonard et al., 2006). In patients reporting lower social support, catastrophising was linked to a perception of a greater level of punishing spousal responses to pain (Buenaver, Edwards & Haythornthwaite, 2007). These findings suggest that multiple pain-related factors may inter-relate to influence the impact of partner responses on adjustment to chronic pain.

2.2.4.1 Empirical studies of partner responses to pain and physical function: direct effects in cross-sectional research.

As noted above, most of the research that has examined spousal responses to partner's pain has measured these with the MPI (Kerns et al., 1985) which classifies partner responses to pain into three categories; overly supportive (solicitous), negative (punishing) and distracting. Of the three MPI partner responses, solicitous and punishing, in particular, have been linked to greater pain related disability (Esteve-Zarazaga & Ramírez-Maestre, 2008; Flor, Kerns & Turk, 1987; López-Martínez, Stroud, Turner, Jensen & Cardenas, 2006; Romano, Jensen, Turner, Good & Hops, 2000).

Leonard and colleagues' (2006) review of research that has explored the relationships between marital satisfaction and spousal responses to pain with chronic pain adjustment outcomes included 74 studies. Outcomes included pain-related disability and psychological distress. Each of

the included articles was rated for quality and strength of the findings. Quality ratings (adequate, good and superior) were assigned according to adequacy of measurement tools used, sample size, use of control groups, and analyses. Strength ratings of the association between variables were based on Cohen's effect sizes (Cohen, 1988).

Eight of the included studies in this review (Leonard et al., 2006) examined the relationship between punishing responses to pain and pain-related disability. The authors rated three of these studies as superior in quality, with the remaining five rated good quality. Of the three studies rated superior, two reported no effect of punishing responses to pain on pain-related disability. The third study rated superior quality reported a weak positive effect. Of the five studies rated as good quality, three reported a moderate positive effect, one a strong effect and one study a moderate negative relationship. Taken together, these results suggest that punishing partner responses may be associated with increased pain-related disability, but the strength of this relationship appears to be moderate rather than strong. More high quality research is needed to establish these links.

Five studies included in this same review (Leonard et al., 2006) investigated the relationship between solicitous partner responses and disability. All but one of these studies were rated moderate quality, the fifth was rated good quality. Mixed results were reported. The only high quality study (Turk, Kerns & Rosenberg, 1992) reported a weak positive association between solicitous responses and disability. Of the four studies rated as moderate quality, three reported positive associations of moderate to strong grade. A final study rated moderate in quality reported a significant inverse relationship of moderate strength. That is, in this study when solicitous responses were higher, disability levels were lower. Together, these results offer some support for a positive relationship between solicitous partner responses to pain and pain-related disability but more high quality research is needed to substantiate these findings.

An earlier review of the literature pertaining to solicitous partner responses to pain (Newton-John, 2002) also included some of the research reviewed by Leonard and colleagues (2006). Newton-John (2002) concluded that solicitous spousal behaviours were associated with increased disability but noted the strength of this relationships appeared to be influenced by other factors such as marital satisfaction or patient's mood.

Less research has examined distracting spousal responses as a predictor of disability than has investigated solicitous or punishing responses. In the review by Leonard et al. (2006), only three relevant studies were identified. All three were rated good or moderate quality. Results were contrasting. A study by Williamson, Robinson and Melamed (1997), rated moderate quality, found that ratings of distracting spouse responses were positively related to interference in 52 patients with rheumatoid arthritis. Somewhat contrasting results were reported by Turk and colleagues (1992) in a study that was rated as superior quality (Leonard et al., 2006). Data from 148 married pain clinic patients and their spouses showed that distracting spouse responses were positively related to disability but only in couples reporting high marital satisfaction. On the other hand, in a sample of patients with rheumatoid arthritis and fibromyalgia, Nicassio and Radojevic (1993) found that attempts by family members to engage patients in recreational activities were linked to decreased disability levels. Because of these mixed results, distracting spousal responses was not included as a risk factor in the current model.

Since publication of the review by Leonard and colleagues (2006) several other studies were identified that have explored the relationship between spousal responses to pain and pain-related physical function. A study by Cano and colleagues (2004) explored links between punishing spousal responses, marital satisfaction, pain, disability and affective distress in 110 pain clinic patients with musculoskeletal conditions and their spouses. Specifically, this study investigated whether negative partner responses to pain and marital satisfaction contributed uniquely to

explaining variance in depression and anxiety symptoms over that explained by pain severity and disability. On a bivariate level, punishing spousal responses were positively associated with disability at weak strength.

In a study of 94 pain clinic patients, Raichle, Romano and Jensen (2011) used the Spouse Response Inventory (Schwartz, Jensen & Romano, 2005) to assess the relationships between spousal responses to pain, depression and pain-related function. This latter outcome was assessed using the Sickness Impact Profile (Bergner, Bobbitt, Carter & Gilson, 1981). These authors also aimed to assess the potential moderating influence of marital satisfaction on these relationships. They reported that both negative and solicitous responses was related to poorer physical function but that marital satisfaction did not moderate these relationships.

Campbell, Jordan and Dunn (2012) investigated associations between pain, disability, relationship quality (cohesion, consensus, and satisfaction) and solicitous and punishing partner responses to pain as well as the influence of symptoms of depression on these associations. The sample comprised 174 primary care patients with low back pain. Regression analysis was used to test the associations. In order to explore the effect of level of depression on the relationships examined in the models, the sample was dichotomized into high and low depression according to their scores on this measure. In the overall sample, punishing, but not solicitous responses, were significantly associated with disability. Suggesting that partner responses to pain may exert differential effects according to the characteristics of the person in receipt, the relationship between solicitous responses and disability relationship was only significant in those with lower levels of depression.

Consistent with the contention of Newton-John (2002), the idea that reactions to spousal support may vary according to personal differences of the pain-affected person has also been explored in other research. For example, Martire, Stephens and Schulz (2011) examined reactions

to spousal support in individuals according to the degree to which they valued being independent. Differential moderating effects of spousal (physical) support on self-efficacy were found for individuals who strongly valued independence compared to those who did not. Those who did not value independence demonstrated higher self-efficacy as a result of spousal support whereas those who valued independence showed increased depression and disability. Taken together, these results suggest that spousal support exerts differential effects according to personal attributes or affective experiences of the person receiving support.

There is also some evidence to suggest that the effect of spousal responses to pain on disability may vary by gender. For example, female spouses have been reported to be more likely to respond solicitously than their male counterparts (Newton-John & Williams, 2006) and solicitous responses are associated differentially with chronic pain adjustment outcomes according to gender (Fillingim, Doleys, Edwards & Lowery, 2003). No studies directly examining the relationship between spousal responses to pain and QOL were identified.

2.2.4.2 Summary of evidence pertaining to partner responses.

Punishing spousal responses to pain have been linked to poorer physical function in the setting of chronic pain. The evidence supporting the relationship between solicitous or distracting spousal responses and disability appears to be inconsistent. It is possible the relationship between punishing and solicitous partner responses to pain and disability may be mediated or moderated by other factors, such as marital satisfaction. Spousal responses to pain also interact with other potential predictors such as pain severity and catastrophising. It is also possible that some of the direct effects of spousal responses to pain may be expressed differently between males and females. The relationship between spousal responses to pain and QOL is at present unknown.

2.2.5 Stress-processing factors – catastrophising.

Catastrophising is defined as an exaggerated negative mental outlook that arises during, or in anticipation of, a pain experience (Sullivan et al; 2001). Catastrophising has been suggested to exert its effects on pain outcomes via several mechanisms including alteration in neural circuits related to pain-related attention and creation of a negative emotional state that contributes to a sense of helplessness (Lumley et al., 2011). Although catastrophising has a robust evidence base linking it to poorer physical function in those with chronic pain, there continues to be debate in the literature about how catastrophising is best defined (Severeijns, Vlaeyen & van den Hout, 2004). Some authors have suggested catastrophising functions as a way of garnering social support or empathy (Savard & Crombez, 2006; Sullivan, 2012). Most commonly, however, and in the current research, it is seen as a pain appraisal process, where fearful thoughts about pain lead to negative emotions (Arnow et al., 2011). In support of catastrophising as an appraisal process, Severeijns and colleagues (2004) argued that the research findings presented in support of the interpersonal aspects of catastrophising (Sullivan, Adams, Sullivan & Williams, 2004) can also be explained by an appraisal model. As catastrophising is placed in the current research as a pain appraisal process, the empirical evidence for its influence on function and QOL is considered from this perspective.

As well as its links to disability, catastrophising is strongly associated with other risk predictors. For example, catastrophising has been reported to account for up to 31% of the variance in pain severity scores (Sullivan et al., 2001). These findings are consistent across a range of chronic pain conditions including whiplash-associated disorders (Nieto, Miro, Huguet & Saldan, 2011), osteoarthritis (Somers et al., 2009), rheumatoid arthritis (Keefe et al., 1989), idiopathic neck pain (Thompson et al., 2010) and fibromyalgia (Martin et al., 1996). In further support of links between catastrophising and pain severity, a recent meta-analysis exploring prediction of post-surgical pain included 29 studies and found that around half the these reported a significant

association between pre-operative catastrophising and the development of post-surgical pain (Theunissen, Peters, Bruce, Gramke & Marcus, 2012). This association was reported to be stronger for musculoskeletal compared to other types of surgery suggesting that there may be some aspect associated with musculoskeletal injuries that particularly predisposes people to make catastrophic pain interpretations.

Catastrophising is also associated with a range of negative affective experiences that include depression, anxiety and possibly anger (Quartana, Campbell & Edwards, 2009). There is some evidence to suggest that catastrophising does not contribute uniquely to explaining variance in adjustment outcomes, over that explained by NA. For example, Hirsh, George, Riley and Robinson (2007) found, in 152 American pain clinic patients that catastrophising explained minimal unique variance in a measure of pain above and beyond that explained by negative mood. This suggests the possibility that catastrophising and NA influence chronic pain outcomes by way of an interactive relationship, an idea that is tested by the current model. Evidence for these links is discussed below in Section 2.2.5.4.

There is also evidence that catastrophising interacts with social experiences. Lackner and Gurtman (2004) found that high catastrophising patients tended to have an interpersonal style demanding support and the pain behaviours of high catastrophisers have been shown to be more sensitive to the presence of another person when compared to low catastrophisers (Sullivan et al., 2004). Marital satisfaction has also been reported to moderate the effects of catastrophising on adjustment to chronic pain (Holtzman & Delongis, 2007).

2.2.5.1 Empirical studies of catastrophising and physical function: direct effects in cross-sectional research.

A large volume of research in cross-sectional pain clinic data has demonstrated that catastrophising is associated with poorer physical function, supporting its place in the current

model as a risk factor. For example, Sullivan, Stanish, Waite and Tripp (1998) investigated catastrophising as a predictor of disability in 86 people with soft -tissue injuries of the neck, shoulders or back following car accidents or workplace injuries in an American pain clinic sample. In a regression analysis, catastrophising explained unique variance in a measure of pain-related disability, over and above that accounted for by pain severity, depression and anxiety.

Similarly, Woby, Watson, Roach and Urmston (2005) showed, in 86 pain clinic patients with low back pain, that catastrophising, together with pain self-efficacy, explained an additional 8% of the variance in disability, after controlling for the influence of demographics, pain severity and measures of coping. However, contrasting with these results, Turner, Jensen and Romano (2000) compared the predictive capacity of catastrophising, beliefs about pain and coping for explaining variance in a measure of disability in a heterogeneous sample of 169 pain clinic patients. Only beliefs and coping, but not catastrophising, made independent and statistically significant contributions to explaining disability in this sample.

A systematic review of cross-sectional research that examined psychosocial predictors of chronic pain adjustment in disabled individuals also established strong and significant links between catastrophising and pain-related physical function (Jensen, Moore, Bockow, Ehde & Engel, 2011). This review study included 29 studies and included individuals with spinal cord injury, amputation, cerebral palsy, multiple sclerosis and muscular dystrophy. Study quality was not rated, perhaps because almost all studies were of cross-sectional design. Examined outcomes were pain-related physical function, psychological distress and pain severity. Twenty-two studies examined the link between catastrophising and the above outcomes with positive and significant associations noted. It important to note issues of generalisability though as the adjustment experiences of disabled individuals may differ from those with chronic pain without disabilities.

A number of other cross-sectional studies in non-pain clinic samples have also reported strong links between catastrophising and pain-related physical function. For example, after adjusting for age, gender and pain severity, Arnow and colleagues (2011) found that both catastrophising and depression were significant predictors of pain-related disability in a sample of 2618 American primary care patients with chronic pain, but the relationship was stronger for catastrophising. Similarly, in a sample of 297 physiotherapy patients seeking treatment for chronic musculoskeletal chronic pain conditions, higher catastrophising was associated with greater pain-related disability (Bergbom, Boersma, Overmeer & Linton, 2011). The association between catastrophising and pain-related disability has also been observed after controlling for neuroticism in 80 patients with fibromyalgia (Martin et al., 1996) and coping style in 147 patients with whiplash disorder (Nieto et al., 2011).

Supporting the idea of potential measurement overlap between catastrophising, anxiety and depression, a later study by Truchon, Cote, Fillion, Arsenault and Clermont (2008) investigated predictors of pain-related disability using structural equations modelling. Predictors were anxiety, depression, anger, catastrophising, coping and life events. The sample consisted of 439 French-speaking Canadian workers who were on sick leave due to low back pain and who were in receipt of workers' compensation benefits. The measurement model in this study, which is based on a factor analysis, contained a number of latent variables that were representative of related constructs. The latent variables were emotional distress (anger, anxiety and depression), coping, life events and pain appraisal (catastrophising, fear-avoidance and perceived pain control). Rather than catastrophising being linked to the latent variable that represented pain appraisals in the factor analysis, the catastrophising items mapped more strongly to the latent variable representing emotional distress. In this model, negative life events and pain appraisals predicted emotional distress and avoidance coping which in turn predicted 53% of the variance in disability. Avoidance

coping was a stronger predictor of disability than was emotional distress. These results highlight the inter-relatedness of catastrophising, anxiety and depression.

2.2.5.2 Empirical studies of catastrophising and physical function: direct effects in longitudinal research.

Focusing on research with a longitudinal design, Wertli and colleagues (2014) completed a systematic review that examined the influence of catastrophising on the development of disability in acute, sub-acute and chronic low back pain. This review included 19 publications based on 16 studies. Most of these were rated of at least moderate quality according to the Scottish Intercollegiate Guidelines Network (SIGN) methodology checklist for cohort studies which was the rating system adopted by the authors (Harbour, Lowe & Twaddle, 2011). Only four studies were based on samples of participants with chronic low back pain. The samples of these four chronic pain studies varied substantially but included participants of a Canadian work hardening program (Dozois, Dobson, Wong, Hughes & Long, 1996), patients of British general medical practices (Grotle et al, 2010), American Workers' Compensation claimants (Chibnall & Tait, 2009) and Swiss physiotherapy patients (Mannion, Muntener, Taimela & Dvorak, 1999). Sample sizes ranged from 1885 to 253. Mean ages of the sample ranged from 32 to 46 years.

Only one study, based on an American sample of 374 Worker's Compensation claimants (n = 374, Chibnall & Tait, 2009), was rated as high quality. This study primarily aimed to explore demographic predictors of poor long term adjustment to chronic pain but also analysed baseline levels of catastrophising and disability as potential predictors of pain-related disability measured six years after claim settlement. A logistic regression model was employed that stratified pain-related disability into high or less than high levels. High disability levels were scores equal to or greater than 45 on the Pain Disability Index (maximal score is 70, Tait, Chibnall & Krause, 1990) Potential predictors included race, gender, age, composite socio-economic and workers'

compensation factors derived from earlier analyses and high or less than high levels of pain, catastrophising and disability reported at baseline. Significant predictors of long-term disability were higher levels of catastrophising and disability at baseline, together with lower socio-economic status.

Catastrophising was also associated with greater disability at nine months follow-up for those in the work hardening program (Dozois et al., 1996). Similar associations were found at 12 months for the British general practice cohort (Grotle et al., 2010). In the group of Swiss physiotherapy patients (Mannion et al., 1999), reduction in catastrophising over time was also associated with a reduction in disability. Of the five studies in this systematic review that applied cut-off values to measures of catastrophising, four of these reported that high catastrophisers experienced a worse functional outcome compared with those who were classified as low catastrophisers. Combined, these results emphasise the importance of targeting catastrophising in treatment to reduce pain-related disability.

An earlier systematic review by Pincus, Burton, Vogel & Field (2002) also provided evidence for catastrophising as an early predictor of later disability in people with low back pain. Twenty-five studies were identified that included psychological factors as predictors at baseline but only six of these met eligibility criteria for methodology, measurement and analysis. Main outcomes included return to work status or disability. Only one study rated acceptable quality identified catastrophising as an early predictor of later stage disability. The five other studies that examined cognitive factors did not meet eligibility criteria for quality.

2.2.5.3 Empirical studies of catastrophising and quality of life: direct effects in cross-sectional research.

The relationship between catastrophising and QOL has been examined less frequently, however several studies have demonstrated significant links between these two constructs. For

example, a significant negative association was found between catastrophising and both mental and physical HRQOL in 401 Chinese patients with chronic musculoskeletal pain recruited from pain clinics (Wong et al; 2014). Health-related QOL was measured in this study with the Short-Form 12 (SF-12; Ware et al., 1993). The SF-12 provides two physical and mental component summary scores indicative of mental and physical HRQOL. Structural equations modelling was used to explore a model in which catastrophising sequentially influenced pain sensitivity and pain-related fear, which in turn influenced mental and physical HRQOL. Bivariate relationships were not reported. The best fitting model was one that explained physical rather than mental HRQOL. This suggested that fear-based pain cognitions may relate more to physical rather than mental HRQOL. Lamé, Peters, Vlaeyen, Kleef & Patijn (2005) also found significant links between catastrophising and HRQOL in a group of 1208 Dutch pain clinic patients. Health-related QOL was measured with a Dutch version of the SF-36 (Aaronson et al., 1992). In regression models, catastrophising was a highly significant predictor of all domains of HRQOL.

Exploring differential effects of catastrophising on QOL in differing psychosocial patient profiles, Börsbo, Peolsson and Gerdle (2008) classified participants into high or low groups according to their scores on measures of catastrophising, depression and pain. Participants were 275 pain clinic patients with whiplash-associated disorders. Subjects were classified “high” if their score on each measure was higher than or equal to the median. Quality of life was measured with three different scales, the SF-36 (Ware et al., 1993), the Life Satisfaction Questionnaire (LiSat-11; Fugl-Meyer, Melin, & Fugl-Meyer, 2002) and the European Quality of Life instrument (EuroQol Group, 1990). Different score patterns on QOL variables were assessed according to their subgroups on scores of pain, depression, and catastrophising. Participants reporting high pain scores had lowest scores on physical measures and those with high depression reported the lowest QOL. When participants reported either high pain or depression scores or both, the impact of

catastrophising on QOL was low. However, its negative influence on QOL increased in the setting of low pain and depression. Thus, the results showed that catastrophising exerted less impact on well-being when pain and depression levels were high.

Psychosocial predictors of HRQOL were examined in a group of 47 patients using data obtained from a randomized controlled trial that investigated treatment outcomes in whiplash (Kall 2009). Health-related QOL was measured with the SF-12 (Ware et al., 1993). A composite outcome score was used to reflect the combined SF-12 scales of mental and physical HRQOL. Predictors included catastrophising, fear of movement, pain severity, depression, disability and pain self-efficacy. Catastrophising was moderately negatively correlated with HRQOL but the only significant predictor of HRQOL in the regression model was pain self-efficacy (Kall, 2009). Taken together, these results suggest catastrophising is negatively related to HRQOL but may not be a significant predictor of HRQOL in the multivariate setting, especially when depression and pain levels are high.

The extent to which catastrophising is a construct that is distinct from other risk predictors such as depression and anxiety has been of some research interest in models predicting QOL. Addressing this issue, Börsbo, Gerdle and Peolsson (2010) examined predictors of QOL in 433 chronic pain patients recruited from Swedish rehabilitation departments. The sample included 47 patients with spinal cord injury-related pain, 150 with whiplash disorders, and 236 with fibromyalgia. Quality of life was measured with the QOLS, (Burckhardt et al., 1992) described above. Catastrophising was strongly correlated with depression, anxiety, and catastrophizing and in a principal components analysis loaded on the same factor suggesting a degree of variance overlap between these measures. This factor was strongly negatively related to QOL. Partial least squares regression demonstrated that of a range of predictors that included pain–related variables,

catastrophising, depression, anxiety and pain self-efficacy, only depression and pain self-efficacy were significant predictors of QOL.

2.2.5.4 Catastrophising as a mediator.

The current model hypothesises that pain severity, intrapersonal and social-ecological factors may influence pain-related disability or QOL indirectly via an association with catastrophising. Despite the bivariate links established between catastrophising and pain severity (Theunissen et al., 2012), anxiety and depression (Quartana et al., 2009) and social factors (Sullivan et al., 2004), so far, only a small number of studies have formally explored whether catastrophising mediates the relationships between these latter variables and disability or QOL. Guite, McCue, Sherker, Sherry and Rose (2011) examined whether catastrophising mediated the relationship between pain severity and pain-related disability in a sample of 138 adolescents with chronic musculoskeletal pain. Regression analysis and bootstrapped estimates of indirect effects were used to test mediation. Catastrophising partially mediated the pain-disability relationship. That is, pain severity was associated with disability partly indirectly via an association with increased catastrophising.

Ullrich, Jensen, Loeser and Cardenas (2007) also investigated catastrophising as a mediator of the relationships between pain, affective distress and pain-related interference in 237 people with chronic pain related to spinal cord injuries. These authors hypothesised that pain and affective distress would firstly interact to influence pain-related interference. They then examined a moderated mediation model in which catastrophising was hypothesised to mediate the relationship between the pain-distress interaction term and disability. A regression-based approach and the Baron and Kenny (1986) guidelines for mediation were used.

A significant interaction term was found that showed that the negative relationship between pain and interference was strongest for individuals with high levels of affective distress. Significance levels for the regression weights for the simple slopes were not reported, meaning that it was not possible to tell if the relationship between pain and interference was significant at high or low levels of affective distress, or only at higher levels. Regression models then showed that when catastrophising was controlled for, the relationship between the interaction term and pain-related interference became non-significant. Because pain, affective distress and the interaction term were all significant predictors of catastrophising in another regression model, the authors concluded that catastrophising partially mediated the relationship between the interaction term and pain-related interference. These results indicate that together pain and affect distress increase catastrophising which in turn influences pain-related interference. Although these results may not necessarily be generalisable to pain-affected adults without a traumatic injury, they are still notable for demonstrating the influence of interactive effects psychosocial predictors on pain-related function.

In partial contrast to these results, Mun, Okun and Karoly (2014) found that catastrophising did not mediate the relationship between pain and physical and emotional function in 335 college students with chronic pain. This study used structural equations modelling to investigate both catastrophising and mindfulness as mediators. The outcome measure was a latent variable that assessed both pain-related interference and emotional burden. Although moderate and statistically significant correlations were noted between catastrophising and pain-related impairment, path analysis and bootstrapped indirect effects established that pain severity did not influence pain-related impairment through an association with higher catastrophising. Instead, pain severity reduced participant's reported levels of mindfulness which in turn reduced reported levels of catastrophising. Reasons to explain these findings are unclear. It is possible that the outcome

measure which incorporated indicators of both physical function and emotional burden may have influenced the findings. Considered together, the above results suggest that one way pain increases pain-related disability may via an association with catastrophising which highlights catastrophising as an important therapeutic target.

Very little research was identified that has explored catastrophising as a mediator of relationships between pain, affective or social factors and QOL. However, one study that found catastrophising mediated the relationship between social support and QOL in people with inflammatory bowel disease (Katz et al., 2016) suggests that positive social experiences may influence QOL indirectly through an association with lowered catastrophising.

Most commonly, catastrophising has been examined as a mediator of the relationship between pain severity and NA, with this hypothesis supported in 669 pain clinic patients (Wood, Nicholas, Blyth, Asghari & Gibson, 2013), and in 310 people with arthritic knee pain (Wade, Riddle, Price & Dumenci, 2011). Catastrophising has also been shown to mediate the relationships between pain severity and both negative and PA in women with fibromyalgia and osteoarthritis (Sturgeon et al., 2014). In a recent study by Hood and colleagues (2012), the relationship between optimism, catastrophising and pain severity was investigated in a sample of 114 healthy subjects. Optimism indirectly decreased perceived pain severity by decreasing catastrophising. Catastrophising has also been shown to mediate treatment related change in a randomized controlled trial of 211 patients of Dutch rehabilitation centres (Smeets, Vlaeyen, Kester & Knottnerus, 2006) and in patients with chronic low back pain (Leeuw et al., 2008).

2.2.5.5 Summary of evidence pertaining to catastrophising.

The contention of the current model that catastrophising is linked to greater pain-related disability and lower QOL is supported by a large volume of research. These findings have been

replicated across varying chronic pain conditions in community-based and pain clinic samples. Catastrophising is strongly associated with other predictors such as pain severity and NA. Some research has demonstrated that a degree of construct overlap may exist between catastrophising and other risk factors such as depression and anxiety. Evidence that pain catastrophising may mediate the relationships between pain severity, intrapersonal and social-ecological factors and pain adjustment outcomes has only been explored in a small number of studies, with results generally supporting this contention of the current model.

2.2.6 Stress-processing factors – fear-avoidance.

The current model hypothesises that fear-avoidance will be directly associated with increased pain-related disability and lower QOL. A number of different terms are linked to the construct of 'fear-avoidance' in the literature including 'fear of movement' and 'fear of pain'. These terms refer to fear of certain pain-related movements and activity avoidance as a result of pain. To reduce confusion, all these terms in the current research are collectively referred to as 'fear-avoidance'. Like catastrophising, fear-avoidance and related terms are generally seen as anxiety-based pain appraisal processes (Turk & Wilson, 2010). Fear-avoidance has been hypothesised to drive pain-avoidant behaviours that create a cycle of disability (Zale, Lange, Fields & Ditre, 2013). The fear-avoidance model of pain is a highly referenced model of pain-related function (Lethem, Slade, Troup & Bentley, 1983; Vlaeyen, Kole-Snijders, Boeren & van Eek, 1995) that proposes catastrophising acts as an antecedent to fear-avoidance which then creates pain-related disability. Fear-avoidance has also been suggested to interact with other fear-related pain experiences (Vlaeyen & Linton, 2000). For example, fear-avoidance has been shown to predict pain severity (Bean, Johnson & Kydd, 2014). Fear-avoidance currently constitutes a key

intervention goal for pain rehabilitation programs where it is addressed through approaches such as graded exposure, education and thought challenging (Turk & Wilson, 2010).

Turk's (2002) diathesis-stress model of chronic pain offers further insights into ways that fear-avoidance, catastrophising and pain self-efficacy may inter-relate to influence pain-related disability. In this model, the diathesis is seen as a psychological vulnerability such as a tendency towards fear-based pain appraisals and an avoidant coping style (Turk, 2002). These diatheses are conceptualized as characteristics of an individual that pre-date the onset of chronic pain and are then activated by the stress of the chronic condition. In this model, some of the negative adjustment effects of fear-avoidance and catastrophising are proposed to be diminished by pain self-efficacy. The empirical evidence for this model is presented in the next section.

2.2.6.1 Empirical studies of fear-avoidance and physical function: cross-sectional research.

A large volume of cross-sectional research supports the contention of the current model that fear-avoidance is negatively associated with pain-related disability. Vlaeyen and Linton (2000) summarised much of the earlier research exploring these links. They described six studies that used both community-based and pain clinic samples and varying measures of both fear-avoidance and pain-related disability. Despite this measurement variability, moderate strength positive correlations between fear-avoidance and disability were evident in all studies. In the multivariate setting several of these studies identified fear-avoidance as a stronger predictor of pain-related disability than pain severity. For example, these findings were reported in 129 pain clinic patients with low back pain (Vlaeyen et al., 1995b), in 35 pain clinic patients with back pain (Crombez et al., 1999) and in a small sample of 35 pain clinic patients of mixed diagnoses (McCracken Gross, Aikens & Carnrike, 1996).

Further support for links between fear-avoidance and pain-related disability were provided in a recent meta-analysis of cross-sectional studies that included 46 studies based on both acute and chronic pain samples. Three of the included studies were conducted with children. Total sample size included in this review was 579 (Zale et al., 2013). The authors did not provide details of how study quality was assessed nor rated. Effect sizes were estimated by calculating average sample size-weighted correlations. Where correlations between multiple measures were reported, composite formulas were used to generate a single effect size for fear of movement on pain-related function in each sample. A significant positive relationship was observed between fear of movement and disability, with moderate to large effects sizes noted that were stable across different measurement scales.

A number of studies have since been published that have also demonstrated significant associations between fear-avoidance and pain-related disability in cross-sectional data. For example, a study of two groups of 88 pain patients, one with complex regional pain syndrome and the other with low back pain, reported a moderate positive correlation between these two factors (Bean et al., 2014). However, a regression analysis in this same research found that only pain severity and depression were significant unique predictors of pain-related disability suggesting that these latter factors are stronger predictors. Reporting similar results, Howell, Hudes, Vernon and Soave (2012) reported a strong and positive correlation between fear-avoidance and pain-related disability in a sample of 35 pain clinic patients but noted that in the multivariate setting, NA and pain severity were stronger predictors of pain-related disability.

2.2.6.2 Empirical studies of fear-avoidance and physical function: longitudinal research.

The fear-avoidance model (Lethem et al., 1983; Vlaeyen et al., 1995a) proposes that catastrophising contributes to fear-avoidance, which over time then creates elevated levels of pain-

related disability. Therefore, a large volume of research has investigated the predictive capacity of earlier fear-avoidance to predict later stage disability. Several review articles have summarised results of this body of research. Mixed results have been reported. A recent systematic review (Wertli et al., 2014) failed to establish significant links between earlier stage fear-avoidance and later stage pain-related disability. Twenty-one prospective cohort studies were included and outcomes included self-reported disability and return-to-work rates. Most studies were based on primary care samples, however five studies, all with large sample sizes of more than 300 people, used insurance company data. All included studies were rated moderate or high quality. Total sample size of the review comprised 9177 participants. Study quality was assessed according to the (SIGN) methodology checklist for cohort studies (Harbour et al., 2011). Consistent evidence was found for the predictive value of fear-avoidance beliefs for disability in the setting of subacute low back pain. However, fear-avoidance beliefs in the setting of acute and chronic low back pain were generally not predictive of later disability. Pincus, Vogel, Burton, Santos and Field (2006) also conducted a small systematic literature review that examined early fear of movement as a predictor of later functional and psychological outcomes in those with low back pain. The authors rated study quality according to criteria they stated were derived from the principles of evidence-based medicine. These included study exclusion and inclusion criteria, dropout rates, quality of measurement and statistical considerations, such as sample size and appropriateness of analysis.

Nine studies of variable quality were included. Only one study was rated as high quality. Lower quality studies were marred by late recruitment, small sample sizes and high rates of participants lost to follow-up. While several studies found statistically significant links between fear-avoidance and later disability, other studies reported this relationship was lost in the multivariate context, or weakened as time extended from baseline. The authors concluded that there is some evidence linking early fear of pain or movement to later pain-related disability, but that

more evidence supported links between early affective distress and later stage disability. However, Pincus and colleagues (2006) cautioned that the reliability of the results was limited by the relatively poor quality of the included studies.

Contrasting with these results, Linton (2000) reviewed 37 studies that examined the role of psychological factors in the development and maintenance of chronic neck and low back pain. Included research examined outcomes in acute, subacute and chronic pain. Samples were recruited from pain clinics, primary care settings, the community and workplaces. Only four studies specifically examined the role of fear-avoidance (Hasenbring, Marienfeld, Kuhlendahl & Soyka, 1994; Klenerman et al., 1995; Linton, Buer, Vlaeyen & Hellsing, 1999; Linton & Hallden, 1998). Three of these studies examined prediction of chronic pain from acute onset pain. All four studies found that fear-avoidance was a stable early predictor of later pain-related disability.

Two studies were identified that have specifically examined evidence for the temporal sequence of relationships proposed by the fear-avoidance model. Leeuw and colleagues (2007) conducted a prospective study based on data obtained from a Dutch population-based study of musculoskeletal injuries. The sample consisted of 152 people with chronic low back pain who completed both a baseline assessment and a follow-up questionnaire six months later. Mediation effects were tested using a regression approach and guidelines provided by Baron and Kenny (1986). Both catastrophising and fear-avoidance were cross-sectionally related to each other and to functional disability at moderate strength. However, catastrophising did not predict later disability which questioned the validity of the fear-avoidance model. Because the prerequisite of a direct relationship between pain catastrophising and disability was not met, mediation by fear-avoidance was not tested.

More recently, Wideman, Adams and Sullivan (2009) conducted a prospective analysis of the relationships proposed by the fear-avoidance model. This study was based on a sample of 121

individuals with work-related musculoskeletal injury who reported high baseline scores of both catastrophising and fear-avoidance. Participants completed a 10 week community-based rehabilitation intervention, with return-to-work status assessed four weeks after program completion. Contrary to the relationships proposed by the theoretical model, early changes in catastrophising were not associated with later changes in fear-avoidance, depression or pain severity. Significant predictors of return-to-work were early change in catastrophising and late change in fear-avoidance and pain severity. Because the fear-avoidance model proposes that early changes in catastrophising predict later changes in fear-avoidance, the authors concluded that their results failed to support the model. However, they suggested that the adopted time frames may have been a potential confounder, as it was possible catastrophising influenced fear-avoidance over a different time interval than that assessed.

In summary, it appears that a strong bivariate relationship exists between fear-avoidance and disability. However, mixed evidence exists to support the relationship between early stage fear-avoidance and later stage disability. The reported strength of fear-avoidance as a predictor of disability in the multivariate setting has also been variable. Evidence in support of the temporal sequence of relationships proposed by the fear-avoidance model (Lethem et al., 1983; Vlaeyen et al., 1995) is also lacking. Thus, the literature supports the contention of the current model that fear-avoidance will directly associated with pain-related disability but its strength as predictor in the multivariate risk-resistance model remains unclear.

2.2.6.3 Empirical studies of fear of movement and quality of life: direct effects in cross-sectional and longitudinal research.

Only a small amount of research was identified that has examined the relationship between fear-avoidance and QOL for those with chronic pain. One study established a significant positive relationship between these two variables. Abbott, Tyni-Lenné and Hedlund (2010) examined fear-

avoidance as a predictor of HRQOL in 107 patients with back pain scheduled for lumbar fusion surgery. Mean age of this sample was 51 years and most participants reported a pain duration of more than two years. Health-related QOL was measured with the European Quality of Life Questionnaire (EuroQol Group, 1990). This scale provides a total scale score. A moderate and significant association was noted between fear-avoidance and HRQOL.

Also supporting a negative relationship between fear-avoidance and HRQOL, Kovacs and colleagues (2007) reported a moderate negative correlation between fear-avoidance and the physical component score of the SF-12 in a group of institutionalised elderly adults with chronic pain of mixed diagnoses. However, the association with the mental component score was weak. In the multivariate context, fear-avoidance beliefs predicted only a small proportion of unique variance in the physical component score of the SF-12 over that explained by pain severity and perceived disability. The same research group analysed cross-sectional associations between fear-avoidance and HRQOL in a mixed sample of 209 primary care pain patients, approximately half of whom had chronic pain. Health-related QOL was measured with the SF-12 (Ware et al., 1993). Fear-avoidance beliefs at the time of initial assessment were negatively associated at moderate strength with both the mental and physical component scores of the SF-12 and were significantly associated with the SF-12 physical component score in a regression analysis that also included disability levels and presence of leg pain as predictors.

Reporting similar results in longitudinal research Orenius et al (2012) found that level of fear-avoidance measured at the start of a pain management program that was conducted over a period of six months did not predict changes in HRQOL. This sample consisted of 111 individuals with chronic pain of mixed diagnoses with a mean age of 45 years. Differential findings were reported for symptoms of anxiety and depression measured at baseline. Specifically, anxiety at baseline predicted a significant decrease of HRQOL, while depression symptoms at baseline

predicted a significant increase in HRQOL. These results suggest that, in order to improve HRQOL, it may be more important to address anxiety than fear-avoidance.

2.2.6.4 Fear-avoidance as a predictor of disability compared to catastrophising.

Because fear-avoidance and catastrophising both represent fear-based appraisals of pain, the extent to which they are distinct constructs is important (Campbell et al., 2013, Sullivan et al., 2001). Results of several studies support that these two constructs are related but distinct. For example, using structural equations modelling Cook, Brawer and Vowles (2006) reported that measures of catastrophising and fear-avoidance loaded on separate latent factors in the confirmatory factor analysis suggesting they are distinct. Further evidence for the distinctiveness of these constructs was provided in a study that examined prediction of pain severity and disability six months after whiplash injury (Nieto, et al., 2013). These authors reported variance inflation factors and tolerance statistics for fear-avoidance and catastrophising from their regression models that reflected no issues with multi-collinearity, also suggesting these predictors are distinct.

However, in contrast to these results, Dehghani, Sharpe & Nicholas (2010) used structural equations modelling to evaluate Turk's (2000) diathesis-stress model of chronic pain in a sample of 207 pain clinic patients with a mean age of 45 years. Catastrophising, fear-avoidance and fear of pain all mapped to a single latent variable representing fear-based pain appraisals indicating that in this sample, these variables demonstrate a high degree of shared variance. Support for the theoretical model was provided by the good fit of the model to the data. Supporting links between fear-based pain appraisals and pain-related disability, the overall model explained 41% of the variance in a measure of pain-related disability.

The extent to which fear-avoidance explains variance in measures of disability over that explained by catastrophising has also been investigated in multiple studies. For example, Cook and

colleagues (2006, described above) used cross-sectional data and structural equations modelling to explore the fear-avoidance model of pain. Their sample comprised a heterogeneous group of 483 pain clinic patients. Both fear-avoidance and catastrophising were significantly and independently associated with pain-related disability but this relationship was stronger for fear-avoidance.

Contrasting with these results Thompson and colleagues (2010) explored predictors of disability in 124 physiotherapy patients with chronic whiplash disorder and found that catastrophising and pain self-efficacy but not fear-avoidance were significant predictors of disability. Similarly, Somers and colleagues (2009) examined the predictive capacity of fear-avoidance compared to catastrophising for pain-related disability and various measured walking speeds in 106 community-based individuals with osteoarthritis of the knee. For all outcomes except fast walking speed, fear-avoidance did not explain any additional variance in the outcome over that explained by pain severity and catastrophising. However, in the model predicting fast walking speed both fear-avoidance and catastrophising were independently associated with walking speed at the same magnitude. The reasons the explanatory capacity of fear-avoidance varies compared with catastrophising is unclear but may relate to sample characteristics, such as diagnosis or reported level of disability. Taken together, these results provide support for catastrophising and fear-avoidance as separate constructs but the extent to which fear-avoidance explains disability after adjusting for the effects of catastrophising may vary according to the specific characteristics of the sample. Assessment and treatment of both constructs may be important when addressing functional impairment for those with chronic pain.

2.2.6.5 Fear-avoidance as a mediator.

Fear-avoidance is placed in the model as a stress-processing factor and is hypothesised to mediate relationships between condition parameters, intrapersonal and social-ecological factors

with disability and QOL. Several studies were identified that compared the mediating effects of fear-avoidance and self-efficacy in the relationship between pain severity and disability in 184 patients with chronic low back pain (Costa, Maher, McAuley, Hancock & Smeets, 2011). Both self-efficacy and fear-avoidance partially mediated this relationship at the time of pain onset, but only improvements in self-efficacy mediated the relationship between changes in pain and changes in disability over a 12 month period. This suggests that self-efficacy is a key change mechanism for disability in those with chronic pain. Fear-avoidance was also shown to mediate the pain severity–interference relationship in 67 individuals with low back pain recruited from the general community (Gay, Horn, Bishop, Robinson & Bialosky, 2015).

A later study by Kamper and colleagues (2012) found that fear-avoidance partially mediated the relationship between pain and disability in 205 patients with neck pain after a car accident and this effect remained unchanged with time. Gheldof and colleagues (2006) also reported results confirming a mediating role of fear-avoidance in the relationship between NA and disability in a sample of 890 workers. No other research was identified that examined fear-avoidance as a mediator of the relationships between other predictors, such as anxiety or depression, with disability.

As the fear-avoidance model of pain proposes that fear-avoidance mediates the relationship between catastrophising and pain-related disability this hypothesis has been examined in a number of studies. Mixed results have been reported. Using structural equations modelling, Cook et al. (2006) reported good fit for a model in which catastrophising predicted disability and depression both directly and indirectly, via fear-avoidance in a heterogeneous pain clinic sample of 469 people, thus supporting the fear-avoidance model. However, in another study of 147 rehabilitation patients with neck pain (Nieto et al., 2013), fear of movement, initial pain severity and initial disability were all significant predictors of disability six months after injury but fear-avoidance did not

mediate the relationship between catastrophising and disability. These findings suggest that the explanatory capacity and validity of the fear-avoidance model varies according to different samples of pain patients. Lee and colleagues (2015) recently published a meta-analysis of 12 studies that explored mediators of the pain-disability relationship in adults with neck and back pain. This review concluded that pain self-efficacy, psychological distress and fear-avoidance all mediated the pain-disability relationship but catastrophising did not.

2.2.6.6 Summary of evidence pertaining to fear-avoidance.

Strong bivariate links are demonstrated between fear-avoidance and disability in cross-sectional research based on heterogeneous groups of chronic pain patients. Some evidence exists to demonstrate that fear-avoidance is predictive of return to work rates in the setting of sub-acute back pain. However, the predictive capacity of fear-avoidance for later disability appears to be limited in the setting of chronic low back pain. In the very small amount of research that has examined the relationship between fear-avoidance and HRQOL, a significant and negative relationship has been identified.

A small number of studies have established fear-avoidance as a mediator in the relationships between both pain severity and catastrophising with pain-related disability, but no studies were found to demonstrate its mediation effects within other contexts. Although fear-avoidance and catastrophising are both fear based pain appraisal processes, there is evidence they are distinct constructs which is an important consideration for the empirical work to follow in Study Three. In research that has examined fear-avoidance and catastrophising as simultaneous predictors of pain-related disability, fear-avoidance has been shown in pain clinic samples to be a stronger predictor. The converse has been noted in community-based or primary care samples.

2.3 Resistance Factors

The current model identifies three categories of resistance factors hypothesised to be associated with improved pain adjustment outcomes; intrapersonal and social-ecological factors and stress-processing factors such as pain appraisals or coping strategies. Optimism and positive affect (PA) are represented in the personal resistance factors category. Social support is represented with the category of resistance social-ecological factors. Represented in the stress-processing category are pain self-efficacy and pain acceptance.

The current model proposes both direct and indirect effects relevant to resistance factors. Firstly, the model proposes that all resistance factors exert direct positive effects on adjustment. Secondly, stress-processing resistance factors are hypothesised to mediate relationships between condition parameters, intrapersonal and social-ecological factors in the model with adjustment. Finally, the model suggests that resistance factors moderate the relationships between condition parameters, intrapersonal and social-ecological factors in the model with adjustment. This latter hypothesis is consistent with both Rose and colleagues (2004) and Holmbeck (1997) who suggested that resistance factors can act as moderators that specify the conditions under which a risk factor exerts a negative influence. According to Rose et al. (2004), resistance factors can act as either resource or protective factors. A resource factor is one that exerts a positive influence on outcomes regardless of the level of risk, while a protective factor exerts a positive influence on outcomes only in the setting of a risk factor. Thus, protective factors can act as moderators of risk-adjustment relationships. Therefore, this literature review considers evidence for direct and moderator effects of resistance factors, as well as mediator effects of resistance stress-processing factors.

2.3.1 Intrapersonal factors – positive affect.

Positive affect has been defined as the emotional response to pleasurable events and includes happiness, contentment and enthusiasm (Pressman & Cohen, 2005). The research examining the effects of PA on health generally divides the concept into stable, disposition-like PA, trait PA, and shorter duration positive emotional experiences that are classified as state PA (Pressman & Cohen, 2005). It has been suggested that there is a heritable component to trait PA (Pressman & Cohen, 2005).

A moderate amount of research has investigated the relationship between PA and general health outcomes. For the most part, these results have been positive. For example, Pressman and Cohen (2005) found a generally protective effect of PA on longevity but noted that these effects were most consistently observed in community dwelling adults aged over 55 years. A more recent meta-analysis (Chida & Steptoe, 2008) of 54 studies in both healthy and chronically unwell samples, found that positive psychological states were associated with significantly lowered mortality. This effect remained significant even after the influence of NA was controlled for. While it makes sense that people experiencing better health may feel positive emotional states more often, even research investigating the relationship of baseline PA and subsequent health events demonstrates that PA is linked to significantly lowered chance of subsequent health events such as stroke and cardiac disease (Pressman & Cohen, 2005) suggesting a positive causal influence of PA on health.

Specifically how PA influences health remains unclear, although several mechanisms have been postulated. It has been suggested that PA may act as a motivator to seek growth, social connections and creativity and may drive healthier lifestyle choices (Finan & Garland, 2015; Frederickson, 2001). Positive affect is also linked to a range of other positive psychosocial factors such as optimism and self-efficacy (Finan & Garland, 2015; Steptoe, Dockray & Wardle, 2009).

Research investigating the biological processes underlying PA suggests that it positively influences the neuroendocrine, autonomic and immune systems (Dockray & Steptoe, 2010). Levels of cortisol, a hormone that is elevated in times of stress, have been shown to be lower for those with greater PA. Higher levels of PA have also been linked to improved cardiovascular function and better sleep (Dockray & Steptoe, 2010). These biological links have been shown to be independent of NA, indicating that PA has distinctly beneficial biological correlates (Steptoe et al., 2009).

While PA has been demonstrated to offer protective effects on adjustment in general, it has been noted that these effects may be particularly important in the setting of adversity (Bonanno & Diminich, 2013). This suggests that PA may function as a protective factor in the context of chronic pain and may provide a moderating effect on the relationship between risk predictors and chronic pain outcomes. Fredrikson's Broaden and Build theory of positive emotions (Fredrikson, 1998) suggests that unlike negative emotions, which focus attention specifically on dealing with threat, positive emotions produce more broad-ranging thoughts and behaviours that are not geared to safety or survival. The Broaden and Build theory (Fredrikson, 1998) suggests that over time, positive affective experiences collectively become significant resources for well-being (Cohn, Fredrickson, Brown, Mikels & Conway, 2009).

Finan and Garland (2015) summarised much of the literature pertaining to PA and pain. In their review of over 100 studies, these authors concluded that there is evidence to show that PA exerts a protective effect of on the perception of experimentally induced pain in healthy subjects. That is, in conditions of high PA, pain perception is lowered. A smaller number of studies have also illustrated that PA provides a similar protective effect on pain perception in subjects experiencing chronic pain in everyday life (for example, Finan, Quartana & Smith, 2013; Litt, Shafer & Napolitano, 2004; Strand et al., 2007). High levels of PA have also been shown to confer a protective effect on pain perception in adults post stroke (Berges, Seale & Ostir, 2011). In this

study of 917 adults aged 50 and over, high PA at discharge from inpatient rehabilitation was associated with lower pain ratings at three months post discharge after controlling for pain, NA at discharge and demographic characteristics.

A longstanding debate has existed within the literature regarding whether NA and PA represent opposite ends of a bipolar continuum or whether they are distinct affective experiences with only a small degree of overlap. This distinction is important because if NA and PA are polar ends of the same spectrum, then reducing NA should produce gains in PA. However, if they are separate experiences, both NA and PA may need to be differentially targeted in treatment (Finan & Garland, 2015). Some cross-sectional research has demonstrated relatively small correlations between these two measures (Finan, Quartana & Smith, 2013, Seebach et al., 2012; Zautra et al., 1995), suggesting that NA and PA are separate affective states. However, other studies have highlighted that the degree of shared variance between measures of PA and NA may depend to some extent on the temporal stability of the measure. That is, whether it is measuring trait or state affect (Finan & Garland, 2015).

Davis, Zautra, and Smith's (2004) Dynamic Model of Affect (DMA) provides further insights into the ways that PA and NA may interact to influence pain adjustment and coping. The DMA proposes that in normal settings, the relationship between positive and negative emotions is largely independent. However, in stressful circumstances, such as living with chronic pain, the differentiation between these two states is diminished. The DMA thus proposes that this leaves people with chronic pain at much higher risk of being unable to sustain positive affective states, which creates a vulnerability to poorer outcomes.

2.3.1.1 Empirical studies of positive affect and physical function: direct effects.

Although a number of studies have examined links between PA, pain severity and social and emotional functioning, relatively few studies have examined the direct effect of PA on pain-related physical function as is proposed by the current model. Two recent studies have explored the association between PA and pain-related interference using multi-level modelling, with both of these reporting positive associations. The first study explored links between sleep, PA and pain-related interference in 220 adults with fibromyalgia (Kothari, Davis, Yeung & Tennen, 2015). Analysis of electronic diary reports showed that pain and PA mediated the relationship between sleep quality and activity interference. Early-morning reports of poor sleep predicted increased pain and lower levels of PA at late-morning, which, in turn, predicted elevated end-of-day activity interference. Mun, Karoly and Akun (2015) also used multilevel modelling to explore links between affective states, pain severity and interference with work goal pursuit and work goal progress in a community sample of 132 adults who completed a 21-day phone diaries. Positive affect was weakly negatively correlated with pain-related interference and weakly positively associated with work goal progress. Morning pain severity and PA influenced evening work goal progress indirectly via perception of pain's interference with work goal pursuit in the afternoon. This latter finding suggests that the influence of PA on pain-related function may be more complex than a simple direct relationship and may instead be mediated by other factors.

Zautra and colleagues (1995) examined activity limitation, pain severity and coping as predictors of positive and NA in three separate samples of adults with rheumatoid arthritis and reported weak negative correlations between PA and activity limitation. Only two other studies were identified that have investigated the direct relationship between PA and physical function in chronic pain samples. Both identified positive effects. A study of 141 patients undergoing spinal surgery for degenerative conditions demonstrated that pre-operative PA did not predict post-

operative function. Instead this study found that PA measured six weeks post-operatively predicted functional status at this same time point (Seebach et al., 2012). This suggests PA does not exert a positive influence pre-emptively but rather provides a concurrent positive influence on physical function. In support of this idea, positive and significant associations have also been noted between PA and measures of actual physical performance. In a sample of 397 individuals with painful osteoarthritis of the knee, those with high PA walked significantly more steps per day than those with low PA (White et al., 2012), suggesting that PA confers a directly protective effect on physical function.

However, a positive outlook has been described as an important factor in promoting adjustment to pain in qualitative research. In a community-based sample of four men and six women interviewed by West, Stuart, Foster and Usher (2012) participants identified that looking for the positives in life helped them cope better with pain. The same authors examined resilience in families with a chronic pain affected member and reported that factors associated with resilience included a positive outlook, patience and tolerance (West, Buettner, Stuart, Foster & Usher, 2012).

2.3.1.2 Empirical studies of positive affect and quality of life: direct effects in cross-sectional research.

Few studies were identified that directly explored the relationship between PA and QOL in pain-affected samples. Research in other chronic illness populations however has demonstrated that PA is linked to improved QOL. For example, Eaton, Bradley and Morrissey (2014) found that PA was significantly associated with QOL and mediated the relationship between gratitude and forgiveness with QOL in 327 people with either arthritis, chronic obstructive lung disease or diabetes. This demonstrates that PA may act as a mechanism through which other positive processes may be associated with improved adjustment to chronic conditions. Similarly, in a study of 215 obese outpatients of Portugese hospitals, Vilhena, Pais-Ribeiro, Silva, Cardoso and

Mendonça (2014) found a strong and significant positive relationship existed between PA and a measure of general well-being. Finally, a systematic review of research investigating links between psychological factors and QOL in people with spinal cord injuries also reported consistent positive links between PA and QOL (Van Leeuwen, Kraaijeveld, Lindeman & Post, 2012). Together, these results suggest that PA confers a protective effect on adjustment outcomes in those with general health problems. This supports the contention of the current model that PA will exert a direct positive effect on chronic pain adjustment outcomes.

2.3.1.3 Positive affect as a moderator.

A hypothesis of the current model is that PA will moderate the relationships between risk predictors and physical function and QOL. No studies were found that have directly examined this hypothesis, however, elevated PA has been shown to attenuate the strength of the relationship between pain and NA. For example, Zautra, Johnson and Davis (2005) investigated the relationship between affect and pain in 124 women with either fibromyalgia or osteoarthritis over a period of 12 weeks using multi-level modelling. Their results indicated that greater PA across the period of data collection was associated with less increase in NA when pain was high or when interpersonal stresses occurred. Participants with higher levels of PA were also less likely to report increased pain over the study duration. These results suggest that PA may exert an important buffering influence on the negative relationship between chronic pain and NA. Such findings are consistent with the moderating role of PA depicted in the current model.

Research has also demonstrated that PA may influence the extent to which fear of pain is generalised to non-painful stimuli. This introduces the possibility that PA may influence function indirectly, by dampening the negative relationship between fear of movement and physical function. For example, in a laboratory-based study of healthy participants, those with low levels of

PA demonstrated impaired safety learning when the painful stimulus was ceased compared to those with higher levels (Meulders, Meulders & Vlaeyen, 2014). A later study building on this work, also in healthy participants, established that experimentally induced PA inhibited acquisition of links between pain and an unconditioned stimulus that was similar to the original painful stimulus, thereby inhibiting generalization of fear learning (Geschwind, Meulders, Peters, Vlaeyen & Meulders, 2015). These findings suggest that PA may provide a protective influence on function by reducing the extent to which fear of pain is generalised to neutral environmental cues and that pain-related fear is extinguished more effectively when PA is high. Therefore, it is possible that interventions aimed at increasing PA may have a protective effect on function indirectly, by reducing the generalisability of fear of movement.

2.3.1.4 Summary of evidence pertaining to positive affect.

It has been suggested that PA may influence health indirectly via a variety of mechanisms that may include increased motivation for healthy behaviours, improved confidence for managing pain and the promotion of better social connections. Evidence also exists to demonstrate links between PA and reduced levels of biological markers of stress, such as cortisol, cardiovascular reactivity and inflammatory indicators. Some research suggests that NA and PA represent different affective experiences and should be targeted separately in clinical intervention. However, other research has showed that differentiation between NA and PA is substantially lessened during times of elevated pain, suggesting that NA and PA may be more closely related. A small number of studies have reported a direct positive relationship between PA and physical function. Positive affect appears to moderate the relationship between pain and NA. It is possible that any positive effects of PA on pain-related function and QOL may also be exerted indirectly via a moderating

effect on the relationships between other risk predictors and function or may be mediated by fearful appraisals of pain.

2.3.2 Intrapersonal factors – optimism.

There is growing research interest in the role of optimism as a potentially protective factor in chronic pain. Optimism has been defined as a mood or attitude associated with a positive expectation about the future (Tiger, 1979). It is generally seen as incorporating cognitive, affective and motivational aspects (Peterson, 2000). There are two main ways optimism is operationalized in the literature, as 'optimistic explanatory style' and as 'dispositional optimism' (Seligman, 2012). Optimistic explanatory style refers to a tendency to see negative events as unstable and specific (Seligman, 2012), that is, to think that things will soon improve and that good is still present in life even when difficulties arise. That is, those with an optimistic explanatory style therefore, acknowledge negative events but think about them constructively (Seligman, 2012). The second way optimism is operationalized in the literature is as dispositional optimism, a construct that arises from the work of Carver and Scheier (1999; Carver, Scheier, Miller & Fulford, 2009; Carver, Scheier & Segerstrom, 2010). Carver and colleagues (2010) describe dispositional optimism as a broad and generalized sense of confidence about the future that pertains to life and not to a specific context. It is this type of optimism that is measured in the current research.

There is good evidence that higher levels of optimism are associated with an improved sense of well-being (Scheier et al., 2001) and that optimists in general experience lower levels of depression and increased perceptions of social support (Seligman, 2012). In relation to health outcomes and behaviours, there is evidence that more optimistic individuals are less likely to perceive barriers to health goals and are more likely to continue with efforts towards goal attainment even when fatigue levels are higher (Affleck et al., 2001).

Several mechanisms have been suggested to explain the observed links between optimism and improved health outcomes in the setting of chronic pain. Optimists may exhibit less negative expectancies of pain and less fearful appraisals (Hanssen, Peters, Vlaeyen, Meevissen & Vancleef, 2012). It has also been suggested that optimists as compared to pessimists tend to use more active and problem focused coping strategies (Gatchel et al., 2007) and may be more likely to seek social support (Scheier, Weintraub & Carver, 1986). Such observations are consistent with a hypothesis of the current model that suggests optimism may influence pain adjustment outcomes indirectly, by decreasing or increasing respectively, levels of risk and resistance appraisal and coping factors such as catastrophising and acceptance.

Links have also been established between optimism and pain, as well as with other risk factors. A recent review of literature related to pain and optimism (Goodin & Bulls, 2013) highlighted that a higher levels of optimism were related to decreased pain sensitivity but noted that the vast majority of this research used cross-sectional data, meaning no conclusions could be drawn about optimism as a causative factor associated with lowered pain sensitivity. However, in a group of 79 healthy Dutch volunteers a causative influence of optimism on lowered pain perception was demonstrated (Hanssen et al., 2012). The investigators induced an optimistic state by asking participants to picture an ideal future self, and then measured pain catastrophising and pain ratings during an experimentally induced pain condition. Increased optimism was associated with lowered pain perception and this relationship was mediated by catastrophising. That is, optimism decreased pain perception indirectly, by reducing levels of catastrophising. In another study of 134 patients with early stage rheumatoid arthritis followed up over one year, optimism was associated with less anxiety and depression at baseline, suggesting a protective effect of optimism on mood (Treharne, Lyons, Booth & Kitas, 2007).

2.3.2.1 Empirical studies of optimism and physical function: direct effects.

A large body of research has examined the relationship between optimism and physical health with positive associations generally reported (Seligman, 2012). However, debate is ongoing in this area as other research has reported no effect or a negative influence of optimism on health (Coyne & Tennen, 2010; Schofield et al., 2004). Seligman (2012) suggested that the influence of optimism on health may depend on the stage or type of the health problem. For example, the effects of optimism may wane in the setting of chronic, intractable illness. It has also been reported that optimism may affect health outcomes differentially according to the extent to which individuals believe their disease process to be controllable using self-care strategies (Fournier, Ridder & Bensing, 2002).

To address some of these reported inconsistencies in the literature, Rasmussen, Scheier and Greenhouse (2009) conducted a meta-analysis investigating the relationship between optimism and physical health. This review included 86 studies that examined mortality, cardiovascular outcomes, physiological markers, cancer, pregnancy and pain outcomes. Included studies were either cross-sectional or longitudinal in design and used a measure of dispositional optimism as well as a physical health outcome. To be included, studies also needed to report the sample size and include an effect size statistic. Fourteen studies used pain-affected samples. However, of these, only one study (Fournier et al., 2002) examined physical function as an end point. This study reported a moderate positive effect size of optimism on physical function. The remaining studies examined the association between pain, fatigue or physical symptoms.

The overall reported effect size for the influence of optimism on physical health outcomes across all studies was small but positive. Effects sizes were larger for studies based on subjective, rather than objective, measures of physical health. These results demonstrate that optimism is linked to better health outcomes although the possibility of a self-report bias was evident. However,

in the context of the current research and Lazarus and Folkman's theory of stress and coping, where perception of an experience is critical in determining the degree to an event it is seen as stressful, self-report remains an appropriate and valid method of assessment of subjective experience.

Of research not included in the above review that has examined the role of optimism as a possible protective factor for physical function in the context of chronic pain, some affirmative results have been reported. Cruz-Almeida and colleagues (2013) explored the psychological profiles and pain characteristics of individuals with knee osteo-arthritis. The predominantly female sample consisted of 194 individuals with a mean age of 57 years. Pain duration was not reported. Participants were clustered into four groups by their psychological profiles. The cluster showing high optimism also displayed low levels of NA, anger and depression and demonstrated the lowest levels of self-reported pain-related disability.

Research has also identified a direct protective effect of optimism on physical task performance in the setting of chronic pain. A study of 488 community-dwelling older adults with knee pain examined optimism as a predictor of timed performance on four physical tasks of daily living (walking, lifting an object, climbing stairs, and getting into and out of a car) and distance walked in a timed walk test (Brenes, Rapp, Rejeski, & Miller, 2002). After controlling for demographic and health variables, optimism was significantly related to improved performance only on the walking task, while pessimism was significantly related to worse performance on all four tasks. This suggests that the negative effects of pessimism are exerted more generally on physical function than the positive effects of optimism.

Contrasting with these results that identified a direct positive influence of optimism on pain-related disability, only weak negative direct links between optimism and physical function were identified by Wright and colleagues (2011) in a study of 89 patients who were overweight with musculoskeletal pain. Reported pain duration in this sample was approximately 12 years.

Additionally, optimism was not a significant predictor in a regression model predicting pain-related disability that also included pain acceptance as a predictor, suggesting pain acceptance may be more strongly associated with pain-related disability than optimism.

Similarly, Ramirez-Maestre, Esteve and López (2012) found that a relationship existed not between optimism and physical function, but rather between optimism and coping style. These authors investigated the relationships between optimism, pessimism, active and passive coping and a range of chronic pain outcomes that included physical function and physical impairment. Their sample comprised 96 patients with mixed pain diagnoses recruited from a Spanish pain management unit who reported a mean pain duration of nine years. Using structural equations modelling, they found empirical support for a model that placed optimism and pessimism as predictors of active and passive coping respectively. Active coping was then significantly and negatively associated with impairment, and positively associated with physical function.

Such links are well supported by a large number of studies in both the pain and non-chronic pain literature. The review by Goodin and Bulls (2013) reported that optimism has been shown to be associated with lower levels of catastrophising as well as higher levels of pain acceptance. Reviewing the general health literature, Nes and Segerstrom (2006) concluded that dispositional optimism was positively associated with approach coping strategies aimed at managing stressors. These observed links between optimism and pain appraisal and coping processes are consistent with the mediation hypothesis of the current model that proposes that optimism influences adjustment indirectly by increasing positive pain appraisal and coping strategies and by decreasing negative ones.

2.3.2.2 Empirical studies of optimism and quality of life: direct effects.

Of the research that has investigated the relationship between optimism and QOL in individuals with chronic pain, positive associations have generally been identified. A Greek study of 96 patients with musculoskeletal pain (Tsakogia, Lyrakos, Damigos, Mayreas & Dimoliatis, 2011) investigated optimism, sleep difficulties and pain severity and duration as predictors of HRQOL, using the Short-Form 12 (SF-12; Ware et al., 1993). The SF-12 provides two physical and mental component summary scores indicative of mental and physical HRQOL. Two regression models were explored using physical and mental HRQOL as the outcomes. In the model predicting physical HRQOL, pain severity and duration and optimism were significant predictors, explaining 27% of the variance in this outcome. Of these three significant predictors, optimism demonstrated the lowest regression weight. However, in the model predicting mental HRQOL, optimism was the only significant predictor and explained 24% of the variance in this outcome.

Treharne and colleagues (2007) reported that both optimism and pessimism, measured at baseline, were significant predictors examined factors associated with QOL in a longitudinal study of in 134 patients with rheumatoid arthritis. Quality of life was measured with the QOLS (Burckhardt et al., 1992). Participants in this study were grouped into those with short, medium and longer duration disease. Disease duration did not related to perceived QOL but potential changes in the relationship between optimism and QOL according to pain duration was not reported. An earlier study by the same group (Treharne, Kitas, Lyons & Booth, 2005) compared psychosocial predictors of QOL in three groups of individuals with rheumatoid arthritis of varying duration. Although only small sample sizes of the groups were noted ranging between 47 and 55 people, optimism was associated with increased QOL in all three groups.

2.3.2.3 Optimism as a moderator.

The idea that optimism may moderate or buffer risk-adjustment relationships is consistent with two major theories of stress and coping. Lazarus and Folkman's (1984) theory proposes that individual differences may influence secondary appraisal of a stressor and subsequently the degree to which an event is perceived as stressful. Hobfoll's (1989) Conservation of Resources theory also proposes that dispositional traits may act as personal resources to reduce stress. Consistent with this idea, optimism has been demonstrated to act as a moderator of the relationship between hopelessness and suicidal ideation (Hirsch & Conner, 2006), negative life events and suicidal ideation and attempts (Hirsch, Wolford, LaLonde, Brunk & Morris, 2007), on the relationship between stress and immune responses in women with breast cancer (Ah, Kang & Carpenter, 2007) and between illness perceptions and well-being in patients with Parkinson's disease (Hurt, Burn, Hindle, Samuel, Wilson & Brown, 2014).

However, only one study was identified that has investigated optimism as a moderator of negative relationships between risk predictors and adjustment outcomes for those living with chronic pain. In a sample of 141 chronic pain patients of an American pain management centre, dispositional optimism moderated the association between pain severity and pain-related interference (Cannella, Lobel, Glass, Lokshina & Graham, 2007). An interesting interaction was noted, although the specifics of it were not probed using simple slopes analysis. Overall the individuals with low optimism reported the greatest interference by pain but strength of the relationship between pain severity and interference increased at higher levels of optimism. These authors of this study postulated that although greater pain severity is associated with more functional impairment overall, pain appears to exert a stronger negative influence on the activity levels of people that have a positive outlook on life than on those with a less positive outlook.

However some inconsistencies are also noted in the literature. For example, Wong and Fielding (2007) established that optimism mediated, rather than moderated, the relationship between pain and QOL in Chinese lung cancer patients. Their predominantly male sample consisted of 334 individuals all with a diagnosis of lung cancer and a mean age of approximately 65 years. Optimism was assessed with a single item scale that asked participants to rate their general attitude to life as either optimistic or pessimistic on a 10 point scale. Quality of life was assessed with a Chinese version of the Functional Assessment of Cancer Treatment scale (ref) which consists of four subscales; physical, functional emotional and social well-being. Mediation was assessed using regression analysis according to the guidelines provided by Baron and Kenny (1986). Moderation was assessed using interaction calculations via regression analysis, however, specific details about the statistical approach such as variable centring (Aitken & West, 1991) were not provided.

Optimism mediated, but did not moderate the relationship between pain and QOL. That is, pain was associated with reduced optimism which in turn reduced reported QOL. This finding is not surprising, given the likely eroding effect of pain on optimism. However, several factors about this research make the results difficult to generalise to populations experiencing benign, not malignant, sources of chronic pain. Firstly, the relationship between optimism and QOL for cancer patients may be quite different to that experienced by those with benign pain conditions. This is because the process of being diagnosed with a malignancy and undergoing treatment for this may exert particular effects on an individual’s outlook and level of optimism. Secondly, the assessment of QOL by a single item questionnaire may also have reduced the generalisability of the results as this measure may lack validity and reliability.

2.3.2.4 Summary of evidence pertaining to optimism.

A number of studies have demonstrated that optimism exerts a protective effect on health outcomes. Multiple single studies have also demonstrated that optimism is associated with better physical function in those experiencing chronic pain. There is some evidence optimism is also associated with better QOL in those with chronic pain. Optimism appears to be associated with reduced levels of other risk factors such as catastrophising and NA and with increased levels of resistance factors such as pain acceptance. These relationships indicate that optimism may influence pain outcomes indirectly via such factors. There is some evidence that optimism may moderate the relationship between pain and physical function although the number of studies in the chronic pain literature is currently very limited.

2.3.3 Social-ecological factors – social support.

A large body of research has examined the influence of social support on health, with results generally suggesting positive associations between these two constructs (Penninx et al., 1997). Conceptually, social support is generally assessed by two types of measures - structural and functional. Structural measures assess the extent to which a network of people are available to provide support, while functional measures assess the extent to which social support is perceived to be available by the individual from existing social networks (Helgeson, 2003). Within the context of functional social support, three aspects are most commonly assessed; instrumental, emotional and informational support. Emotional support refers to the availability of people for reassurance, sympathy and to make a person feel valued and wanted. Instrumental support refers to the availability of practical assistance, for example with housework or shopping. Informational support refers to the provision of information or guidance (Helgeson, 2003).

In the general health literature, both structural and functional social support have been shown to be beneficial for adjustment (Stanton et al., 2006). Social support has been suggested to exert its protective effects on health and QOL directly, and indirectly, via moderating or buffering effects on other predictors (Cohen & Wills, 1985). It has been suggested that social support may exert a protective effect on health outcomes indirectly via a range of different mechanisms that include improved coping strategies, increased motivation to cope with stressors, support of positive health behaviours and reduced physiological reactivity to stress (Wills & Fegan, 2001).

Thoits (1995) summarized the findings of a number of earlier reviews examining the effects of social support on health and reported three major findings. Firstly, structural support is associated with direct positive effects on health but does not buffer the negative effects of stress on health. Secondly, emotional social support is linked to direct positive effects on health and buffers the negative effects of stress on health. The most common indirect effect examined in the literature in relation to social support has been its moderating effects on the relationship between stress and health and stress with QOL. Thirdly, a close loving relationship with another appears to reduce the negative effects of stress on health.

The literature examining links between social support and QOL in general health populations is extensive. However, research examining links between social support, QOL and physical function in the context of chronic pain is less extensive than that which has examined the influence of cognitive predictors. Blyth and colleagues (2007) suggested this was because pain adjustment research was focused for many years predominantly on the influence of intra-individual risk predictors on adjustment rather than the social context of pain. Of the research that has examined social influences on adjustment to chronic pain, the largest body of work examined the damaging effects of negative couples' interactions (Valente, Ribeiro & Jensen, 2009) or overly supportive spouses (Cano et al., 2006). Where research has investigated the influence of general

social support for those with chronic pain, the most common outcome measure has been indicators of mental health.

Considerable inconsistencies have been noted in relation to the direct and indirect effects of social support on physical function and QOL for those with chronic pain. This may partly be due to differences in the ways social support has been operationalised and measured. However, the noted inconsistencies also likely relate to differential effects of social support that are specific to the chronic pain context. For example, although social support is generally considered to act as a resource, overly supportive relationships may promote dependency and erode independence and unwanted support may reduce autonomy (Mavandadi, Rook & Newsom, 2007). This may be particularly true in the setting of chronic pain where pain behaviours may strongly socially reinforced (Fordyce et al., 1968).

A recent systematic review that explored mechanisms through which social experiences may modulate the perception of pain, offers some additional insights that may also help explain noted inconsistencies in the literature (Krahé, Springer, Weinman & Fotopoulou, 2013). This review included 26 laboratory based pain studies, conducted in healthy adults. Most studies manipulated the nature of the relations between the individuals in an interpersonal exchange, using a measure of pain or pain behaviour as an end-point. Thus, the nature of the research in this review differed substantially from the clinical research that forms the basis of this current review. Nonetheless, the results are pertinent because they offer insights into potential mechanisms that may influence how social experiences and pain may interact.

The results of Krahe and colleagues (2013) showed that, in general, positive interactions reduced pain, while negative, mixed valence or ambiguous interactions were associated with increased pain. However, these associations were moderated both by the type of relationship involved and by individual differences of the person in pain, such as their attachment style or level

of catastrophising. Specifically, pain was perceived to be reduced when a participant was open to support and recognised a positive orientation of the other person towards them. Placing their results within a theoretical neurocognitive framework that emphasizes the importance of the cognitive appraisal of threat and safety, Krahe and colleagues (2013) suggested that social experiences modulate the pain experience by either signalling the safety or threat of pain itself or by signalling the safety or threat of the environment in which pain occurs.

Krahe and colleagues' (2013) results reflect those of an earlier systematic review of socially modulated stress-responses in both humans and animals (Hennessy, Kaiser & Sachser, 2009). This review examined social buffering of the hypothalamic–pituitary–adrenocortical (HPA) axis, which is understood to be the body's primary stress-responsive system. It is activated in both aversive and arousing situations (Hennessy et al., 2009). This review found that while many studies demonstrated that the presence of a social companion moderates the response of the HPA to stressful situations, other studies of the same phenomenon failed to identify a significant effect. These authors concluded that the nature of the relationship between individuals appeared to be a critical factor in determining whether social support provided stress buffering effects on the HPA. Hennessy and colleagues (2009) also identified a number of other factors that appear to influence the degree to which stress buffering occurs as a result of social interactions. These include the broader social organization within which the person is embedded, and their developmental stage.

The above findings are also consistent with a related body of research that has demonstrated adult attachment style exerts an influence on social experiences within a pain context and on adjustment to chronic pain (Meredith et al., 2008). Such research is beyond the scope of this thesis to explore, but highlights the complexities involved in research regarding social influences on chronic pain and possible explanations for the inconsistencies in findings noted in the literature.

Social support has also been shown to interact with other variables that are associated with adjustment to chronic pain. For example, in 96 married individuals with chronic pain of mixed diagnoses, higher perceived support was associated with decreased catastrophising in those with longer pain durations (Cano, 2004). Links have also been consistently demonstrated between social support and indicators of mental health. In a four year, prospective analysis of 116 individuals with rheumatoid arthritis, Benka and colleagues (2012) found that emotional support, together with other predictors such as pain, disability, and depressive symptoms at baseline, significantly predicted depressive symptoms at four years. Social support was also a significant predictor of depression at one year post-injury in 274 Swiss individuals with work-related injuries. This sample had a mean age of 43 years. Social support was measured with not yet validated French and Italian versions of the social support scale of the Indication of the Rehabilitation Status questionnaire (IRES-3; Bührlen, Gerdes & Jäckel, 2005). This subscale assesses perceived support with four items, such as ‘‘If I need help there is hardly anyone who takes me off work’’. The subscale social support had to be translated into and is not validated yet. (Pjanic et al., 2014).

2.3.3.1 Empirical studies of social support and physical function: direct effects.

There is some evidence to show that chronic pain patients reporting high levels of social support are less disabled by pain. However, inconsistencies in the associations between social support and physical function have been noted. Specific protective effects have been shown to vary according to context, for example by the prevalence of negative interpersonal interactions (Okun & Keith, 1998; Nezlek & Allen, 2006) or by setting, for example the workplace compared to private lives (Hoogendoorn, van Poppel, Bongers, Koes, & Bouter, 2000). Spousal support has also been associated with depression and slower walk times in pain patients with high, but not low,

independence centrality (Martire et al., 2011). Results such as these illustrate the importance of clinical interventions being tailored to the individual.

Two relevant systematic reviews were identified that have explored links between social support and physical function for those with chronic pain. The first included 17 papers and examined the influence of social support on outcomes for individuals with chronic spinal pain (Campbell, Wynne-Jones & Dunn, 2011). Study outcomes included pain ratings, disability, recovery from injury and psychological variables such as levels of depression and catastrophising. The approach used to assess the quality of included papers was reported as based on the approaches used by a number of review articles they referenced. Standard quality criteria for research were applied, such as provision of adequate description of the recruitment procedure, inclusion and exclusion criteria, measures and use of appropriate analysis and sample size.

Of the included studies, only three explored the effect of social support on functional outcomes. Inconsistent effects were reported for both instrumental and emotional support. However, study quality was variable so no conclusions about the relationships between social support and physical function were able to be made. Based on included cross-sectional studies, results indicated that social support was more strongly associated with psychological factors than with pain perception or disability. These results suggest that social support may influence pain outcomes such as disability indirectly, by moderating psychological distress.

A further systematic review of psychosocial factors influencing adjustment to chronic pain in people with disabilities included 29 studies (Jensen et al., 2011). Six of these were prospective cohort studies, the remainder were cross-sectional in design. The authors did not report study quality ratings. Disabilities included in the review were spinal cord injury, amputation, cerebral palsy, multiple sclerosis and muscular dystrophy. Seven of the included studies examined social support (as opposed to pain contingent partner responses to pain) as a potential predictor of physical

functioning. Significant positive associations were reported by four of these studies. However, these results were noted predominantly in research relating to amputees, raising questions about the generalizability of the results as the adjustment issues faced by amputees are likely to differ substantially from those living with chronic pain.

In support of a protective effect of social support for function, Demange and colleagues (2004) found that in 542 European patients with rheumatoid arthritis, those with higher levels of social support experienced less disability. In this sample, perceived support exerted a stronger protective effect on function than network size. Similarly, Neugebauer and Katz (2004) found that instrumental support provided a weak protective influence on functional impairment resulting from rheumatoid arthritis in 404 primary care patients.

Evers, Kraaimaat, Geenen, Jacobs and Bijlsma (2003) also found that social support was associated with reduced disability. They assessed the relationship between emotional and instrumental support, and network size with disability levels at three and five years post diagnosis in 79 patients with newly diagnosed rheumatoid arthritis. Importantly, clinical status remained relatively stable over the five year period. Using regression analysis, they showed that less emotional and instrumental support, smaller network size and passive coping together predicted increases in functional disability at three years. At five years post diagnosis, coping was less important. At this time point, only lower support and smaller network predicted functional disability. This suggests that that for those with rheumatoid arthritis, as time extends out from diagnosis, available support is more important to function than coping style.

In contrast to these results, social support did not significantly predict disability in a similar study that prospectively examined social support as a moderator of changes in functional disability over a 13 year time period in 129 patients with rheumatoid arthritis (Strating, Van Schuur & Suurmeijer, 2007). Regressing disability at the time of final follow up, significant predictors

explaining 56% of the variance were gender, disability levels at prior time points and an interaction term between emotional distress and erythrocyte sediment rate (an inflammatory marker). It is possible that prior disability was so strongly associated with later disability that other predictors in the regression became non-significant, however it is noteworthy that the regression weight of social support in the final regression analysis was the smallest of all included predictors.

López-Martínez, Esteve-Zarazaga and Ramírez-Maestre (2008) also failed to establish a direct link between social support and physical function in a mixed group of 117 chronic pain patients recruited from a Spanish Clinical Pain Unit. These researchers investigated the relationships between social support, coping, pain severity, depressed mood and functional status and functional impairment using structural equations modelling. In their best fitting model, social support was significantly, negatively and directly associated with both depressed mood and pain severity, but not with functional status or impairment.

Reporting similar results, social support did not predict physical function in 407 older Dutch adults with chronic pain and co-morbid chronic health conditions and a mean age of 77 years (Hermsen, Leone, Smalbrugge, Dekker & van der Horst, 2014). Using regression analysis, this study examined the predictive capacity of a range of psychosocial predictors and biological measures for physical function and activity participation. Predictors included perceived social support, anxiety, depression, self-efficacy and coping, as well as previous falls, frailty and body-mass index. Social support was measured with the 12-item Social Support Scale (Feij et al., 1992). Bivariate associations were not reported. In the multivariate context, social support did not predict any of the functional outcomes, except participation restrictions in basic activities. The largest proportion of variance was accounted for by other psychosocial predictors, such as self-efficacy and coping style. The current model proposes that social support will be negatively associated with disability. In reality, this relationship is likely to be mutually reinforcing. This idea was explored

in a large sample of 1024 Swedish individuals with spinal pain. Those with longer durations of pain were characterized by lower levels of social support and higher levels of catastrophising, suggesting that as time with a pain condition extends, social support is eroded (Demmelmaier, Lindberg, Åsenlöf & Denison, 2008).

2.3.3.2 Empirical studies of social support and quality of life: direct effects.

Although a large volume of research exists that has explored links between social support and QOL in the general and health literature, a smaller number of studies appear to have examined these links in those with chronic pain. However, in general, strong positive relationships have been reported. These links may reflect, in part, the frequent inclusion of indicators of social functioning and satisfaction in QOL measures. Ethgen and colleagues (2004) investigated the relationship between social support and HRQOL in 108 rheumatology and rehabilitation patients with hip or knee osteoarthritis. Health-related QOL was assessed with the SF-36 (McHorney Ware, Lu & Sherbourne, 1994). Satisfaction with daily emotional and problem oriented social support, together with instrumental support and transactions of social companionship were significant predictors of physical health. Approximately 50% of variance in the physical domains of HRQOL was explained by these predictors.

Further exploring the relationship of social support to QOL in the multivariate context, Lee and colleagues (2008) investigated the predictive capacity of social support, combined with demographic information, pain severity, coping, depression and pain interference on QOL in 171 individuals with chronic pain of mixed aetiology. Quality of life was measured with the World Health Organization QOL– Bref Scale (WHOQOL Group, 1995) which provides four subscale scores (physical, psychological, social and environmental) and two items that assess overall satisfaction with QOL. Social support was measured with the Social Support Index (SSI;

McCubbin, Patterson & Glynn, 1982) which is composed of 17 items. A positive but weak correlation was reported between social support and overall QOL, and social support did not contribute significantly to the variance in the total QOL measure when examined together with other variables. Instead, pain severity, duration and pain-related interference explained physical QOL. Pain duration, depression and coping together predicted overall or total QOL. Franks, Cronan and Oliver (2004) provided further support for a protective effect of social support on QOL in a study of 568 women diagnosed with fibromyalgia. Regression analyses showed that perceived quality of social support was a significant positive predictor of perceived well-being.

2.3.3.3 Social support as a moderator.

Cohen and Wills (1895) proposed two main ways that social support may influence well-being. Firstly, they proposed a main effects model which suggested social support provided a direct positive influence on well-being. They also proposed that social resources buffer or moderate risk-well-being relationships. Consistent with this idea, the current model also proposed that social support, as a resistance factor, will moderate risk-adjustment relationships in the model. Although a moderating influence of social support on adjustment outcomes has been established in the broader stress and coping literature (Cobb, 1976, Cohen & Hoberman, 1983; Taylor et al., 2008), less research has explored these relationships in those with chronic pain. Stark-Taylor, Davis and Zautra (2013) investigated the effect of a satisfying relationship compared to a relationship characterized by low satisfaction or no relationship in a sample of 251 women with chronic pain due to osteoarthritis and/or fibromyalgia. They found that being in a happy marriage dampened the negative relationship between changes in pain and changes in disability.

The majority of other research examining moderating effects of social support has investigated NA as the outcome, with inconsistent results reported. For example, Benka and

colleagues (2014) investigated social support as a moderator of the relationship between functional impairment and depressive symptoms. This study involved a longitudinal analysis of 124 individuals with rheumatoid arthritis or fibromyalgia. Participants completed assessments annually for four years. Significant moderating effects were reported. Specifically, patients in this study reporting low social support and high levels of disability experienced more depressive feelings at all points of assessment.

Feldman, Downey and Schaffer-Neitz (1999) also found that perceived instrumental and emotional support significantly dampened or buffered the relationship between pain and negative mood in a sample of 109 people with complex regional pain syndrome. These authors did not report significance levels associated with the post-hoc testing of the moderation effects. However, visual inspection of the simple slopes graph suggested that the relationship between pain and negative mood was weaker under conditions of high social support.

The potential moderating effect of positive social support on the relationship between negative social experiences and depression was explored by Riemsma and colleagues (2000) in 229 Dutch patients with rheumatoid arthritis. Negative social experiences were associated with greater depressive symptoms and positive social experiences with less depression. A significant moderating effect was reported in which positive social support reduced the strength of the relationship between problematic support and depression. Specifically, the relationship between problematic support and depression was not significant in those with high levels of positive support but was significant in those reporting low positive support.

Contrasting with these results, Pjanic and colleagues (2014) tested social support as both a mediator and moderator of the relationship between pain and depression in 274 injured Swiss workers. Despite exerting a direct positive influence on depression, social support neither mediated nor moderated the relationship between pain and depression. Similarly, Doeglas and colleagues

(2004) examined the potential moderating effects of satisfaction with instrumental and emotional support on the relationship between disability and depression in 264 Dutch patients with rheumatoid arthritis. Potential moderating effects were assessed using an interaction term in regression analysis but failed to identify a significant effect.

2.3.3.4 Summary of evidence pertaining to social support.

Research investigating the direct relationship between social support and chronic pain outcomes has demonstrated inconsistent effects for the relationship between social support and pain-related physical function. However, a consistently positive association for the outcome of QOL has been reported. Inconsistent findings are also observed for the moderating effects of social support on the relationships between risk predictors with physical function and QOL. Some of these observed inconsistencies may be due to dual effects of social support in the chronic pain context, as a promoter of well-being and as a reinforcer of pain behaviours. From the broader related research, it is also likely that a range of other individual factors such as the characteristics of the person in receipt of support and the nature of the relationship between individuals may all influence the impact of social support on physical function and QOL for those with chronic pain.

2.3.4 Stress-processing factors – pain self-efficacy.

Pain self-efficacy is based on Bandura's (1977) general concept of self-efficacy and reflects confidence in completing a behaviour or task despite pain (Nicholas, 2007). According to Lazarus (1999), self-efficacy is seen as an appraisal process and, unlike dispositional traits such as optimism, is specific. For example, an individual may have high self-efficacy for managing fatigue, but low self-efficacy for pain (Lowe et al., 2008). There are a number of ways in which self-efficacy is suggested to impact pain-related disability and QOL. Higher pain self-efficacy is associated with task persistence in the face of adversity (Gatchel et al., 2007), active coping behaviours (Andrasik

et al., 2005, Jensen, Turner & Romano, 1991) and decreased anxiety and physiological arousal (Turk & Okifuji, 2002). Individuals with high self-efficacy may have higher motivation to engage in active coping behaviours because they have higher expectations for success (Gatchel et al., 2007). Pain self-efficacy has also been shown to activate opioid related pain inhibitory pathways, reducing pain severity (Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987). Conversely, low self-efficacy for managing pain may be associated with activity avoidance and increased use of medication (Jackson et al., 2014).

A large meta-analysis found that individuals with chronic pain report substantially lower pain-related self-efficacy compared to healthy controls (Burke et al., 2015). This meta-analysis, also referenced above, included 110 papers with a total sample size of 67,554 participants. The majority of included studies were based on treatment seeking samples with a smaller number of studies using community-based or primary care samples. Studies included measures of general self-efficacy as well as self-efficacy for managing pain. The pain-affected participants reported lower levels of self-efficacy compared to controls and their confidence for feeling in control of everyday life was substantially higher than their confidence to function despite pain. This suggests that lack of confidence for managing pain does not reflect a general trait based lack of confidence, but rather that poor pain self-efficacy may be specific to situations where pain is likely to be aggravated. The current adjustment model places pain self-efficacy as a resistance stress-processing factor. Thus, its potential role as both a mediator of relationships between other predictors and pain adjustment outcomes and a moderator effects of risk-adjustment relationships is reviewed together with evidence for its direct effects on pain-related disability and QOL.

2.3.4.1 Empirical studies of self-efficacy and physical function: direct effects.

A large body of research has established pain self-efficacy as a robust predictor associated with improved pain-related function. Much of this research was reviewed by Jackson and colleagues (2014) in a recent, large meta-analysis that included 86 studies and a total sample size of 15,616 pain-affected individuals. Included studies were cross-sectional or longitudinal in design and assessed self-efficacy for managing pain not general self-efficacy. Studies were based on heterogeneous samples of individuals with chronic pain as well as specific diagnostic groups such as those with low back pain, osteo and rheumatoid arthritis and fibromyalgia.

Physical outcomes included measures of pain-related disability and interference. More than 40 self-report measures of functional impairment were used and therefore operationalisations of function displayed a high degree of discrepancy. Outcomes included pain-related disability and interference with work, activities of daily living and social function. Varying measures were also used to assess pain self-efficacy. These assessed confidence to complete specific tasks or activities despite pain as well as confidence to manage pain or other related symptoms. Effect sizes and their heterogeneity were calculated for each outcome. Cohen's (1992) criteria for grading strength of effects sizes was adopted, that is effect sizes of $r = .10$, $r = .30$, and $r = .50$ were interpreted as small, medium, and large, respectively. When significant heterogeneity was identified, subgroup analyses and meta-regression were used to explore sources of variability. The moderating effects of key socio-demographic and pain variables such as age, proportion of females in sample and pain duration were assessed using the method of moments approach for meta-analysis (Higgins & Thompson, 2002).

Demonstrating that pain self-efficacy is a key positive influence on pain-related function, medium to large effect sizes were reported for the effect of pain self-efficacy on physical

impairment outcomes. However, high levels of heterogeneity of effect sizes were reported likely due to substantial variability in study design, measures and sample characteristics. Importantly, moderate effect sizes were evident not only for studies based on self-report measures of physical function, but also for those using performance-based measures. This suggests that the relationship between self-efficacy and pain-related impairment may be independent of self-report bias providing further evidence of a protective influence of pain self-efficacy on pain-related function.

Jackson and colleagues' (2014) meta-analysis also found that age and pain duration also influenced the relationship between pain self-efficacy and functional impairment. That is, the relationship between pain self-efficacy and pain-related function was stronger in older people and in those with longer pain durations. Thus, the relationship between self-efficacy and functional impairment appears to become more entrenched over time. The authors suggested that self-efficacy is likely to be self-reinforcing, such that people with high self-efficacy are more likely to undertake novel tasks or experiences and be positively reinforced by successes, where those with low self-efficacy are more likely to withdraw from functional tasks, leading to lowered confidence.

Other studies not included in the above meta-analysis have also reported similar results. For example, higher pain self-efficacy was associated with lower levels of disability and improved function in 209 youths with chronic headache (Kalapurakkel, Carpino, Lebel & Simons, 2014). Higher self-efficacy for physical activity was associated with higher physical activity levels in 309 older adults with arthritis (Sperber et al., 2014). Cross-cultural research has also demonstrated a significant association between pain self-efficacy and physical function. For example, in a sample of 324 Portuguese patients with chronic musculoskeletal pain, Ferreira-Valente, Pais-Ribeiro and Jensen (2014) found, using regression analysis, that pain self-efficacy was a significant predictor of pain-related interference. A New Zealand study of a heterogeneous group of 874 pain clinic patients found that higher pain self-efficacy was significantly associated with less disability

(Shipton, Ponnamperuma, Wells & Trewin, 2013). Self-efficacy was also moderately and significantly correlated with pain-related interference in 188 individuals with chronic pain and disabilities that included spinal cord injury, amputation and multiple sclerosis (Alschuler et al., 2016).

2.3.4.2 Empirical studies of pain self-efficacy and quality of life: direct effects.

Fewer studies have examined the relationship between pain self-efficacy and QOL, however, in those that have, statistically significant positive relationships have generally been reported. For example, Libby and Glenwick (2010) examined predictors of QOL in 57 children and adolescents with fibromyalgia, recruited from American paediatric rheumatology clinics. In a regression analysis, pain self-efficacy, together with catastrophising and daily hassles were significant predictors of child-rated QOL. However, of these predictors, both catastrophising and daily hassles were stronger predictors of QOL than self-efficacy.

In a study of 433 rehabilitation patients with whiplash and spinal cord injuries or fibromyalgia, Börsbo and colleagues (2010) found that pain self-efficacy and depression together predicted overall QOL, but the relationship with QOL was stronger for depression than pain self-efficacy. Quality of life was measured with the QOLS (Flanagan, 1978; Burckhardt & Anderson, 2003) which provides a single scale score incorporating satisfaction with life across a range of 16 different domains. Self-efficacy was measured with the Arthritis Self-Efficacy Scale (Lorig, Chastain, Ung, Shoor & Holman, 1989) which consists of three subscales that assess confidence for controlling pain, self-efficacy for function in daily living and confidence for controlling other symptoms related to chronic pain.

Yazdi-Ravandi and colleagues (2013) found that coping self-efficacy, not self-efficacy for pain, was a significant predictor of QOL in a heterogeneous group of 290 Iranian pain clinic

patients. Quality of life was assessed using the World Health Organization WHOQOL-BREF (EuroQol Group, 1990) which provides scores across four domains - physical, psychological, social and environmental. Coping self-efficacy was assessed with the Coping Self-Efficacy scale (Chesney, Neilands, Chambers, Taylor & Folkman, 2006) which measures confidence for certain behaviors when faced with life challenges. Coping self-efficacy was a significant predictor of all QOL subscales and the sole significant predictor in models predicting social and environmental QOL. In models predicting physical and psychological QOL, coping self-efficacy was a weaker predictor of QOL than pain intensity.

No studies were identified that examined the relationship between pain self-efficacy and QOL in community-based heterogeneous groups of adults with chronic pain. However, another recent study of 194 community-based individuals with osteoarthritis investigated predictors of daily variability in QOL using multilevel modeling analysis (Schneider et al., 2012). Lower self-efficacy was associated with greater variability in satisfaction with quality daily life. Taken together, these results demonstrate that pain self-efficacy is strongly associated with both pain-related physical function and QOL in those with chronic pain.

2.3.4.3 Pain self-efficacy as a mediator.

The current model proposes that pain self-efficacy as a stress-processing factor will mediate the relationships between condition parameters, intrapersonal and social-ecological factors and pain-related disability and QOL. Although few studies have examined mediation of relationships between intrapersonal or social-ecological factors with adjustment outcomes, multiple studies have established that pain self-efficacy mediates the pain-physical function relationship. Specifically, pain intensity has been found to be associated indirectly with pain-related disability via a negative association with pain self-efficacy.

For the main part, mediation in this literature has been tested using regression analysis and the guidelines provided by Baron and Kenny (1986). Using a longitudinal design, Costa and colleagues (2011) found that pain self-efficacy partially mediated the relationship between changes in pain severity and disability in 184 Australian primary care patients with low back pain. Specifically, regression analysis showed that the relationship between changes in pain severity was indirectly associated with changes in disability through a negative association with pain self-efficacy.

Arnstein (2000) also used regression to investigate whether pain self-efficacy mediated the pain-disability relationship in three different heterogeneous groups of pain clinic patients. Clinic settings varied and one sample included only pain patents without a prior history of depression. Pain self-efficacy partially mediated the relationship between pain severity and disability in all three groups, suggesting that even in patients with varying affective profiles, self-efficacy is an important mechanism through which the influence of pain on disability is expressed. These results were replicated in an earlier study by the same research group. Using cross-sectional data from 126 chronic pain patients recruited from pain clinics, pain self-efficacy was indirectly associated with disability via a negative association on pain self-efficacy (Arnstein, Caudill, Mandle, Norris & Beasley, 1999).

Pain self-efficacy has also been shown to mediate the relationship between pain severity and disability individuals with both fibromyalgia and whiplash injury. Using regression analysis, Puente and colleagues (2015) reported that the relationship between pain severity and disability was fully expressed via its negative association with pain self-efficacy in 144 women with fibromyalgia. Similarly, Peck and Smitherman (2015) used path analysis and bootstrapped estimates of indirect effects, to investigate perceived efficacy for headache self-management as a mediator of the relationship between headache severity and disability. The sample in this study was

907 undergraduate students with headache disorders. Again, the relationship between pain self-efficacy and disability was expressed partially indirectly, via a negative association with perceived efficacy.

A small number of studies have also established pain self-efficacy as a mediator of relationships between other predictors and physical function. For example, using regression analysis to test mediation, Perry and Francis (2013) also found that pain self-efficacy partially mediated the relationship between pain-related fear and disability in a heterogeneous group of 68 individuals with chronic pain. That is, pain-related fear was indirectly associated with disability via a negative association with pain self-efficacy. Shelby and colleagues (2008) also used regression analysis to investigate if different domains of self-efficacy mediated relationships between catastrophising and physical function, pain and emotional symptoms in 192 overweight individuals with knee osteoarthritis. The relationship between catastrophising and physical disability in this sample was fully mediated by self-efficacy for physical function. That is, the negative effects of catastrophising on disability were expressed entirely via a negative association with self-efficacy. Pain self-efficacy has also been shown to mediate the relationship between fear-avoidance and disability in 102 patients of a pain rehabilitation program with low back pain (Woby, Urmston & Watson, 2007). Similar to results reported by Shelby and colleagues (2008) the negative effects of fear-avoidance on disability were expressed entirely via a negative association with pain self-efficacy.

Self-efficacy has also been found to mediate the relationship between resistance predictors and chronic pain outcomes. For example, pain self-efficacy mediated the relationship between resilience (a latent factor that incorporated measures of extraversion, vitality and PA) and pain-related physical function in 275 patients with osteoarthritis of the knee (Wright et al., 2008). In a sample of 161 older, community based individuals with knee osteoarthritis, mastery beliefs

(defined as an internal sense of efficacy) mediated the relationship between optimism and well-being (Sherman & Cotter, 2013).

2.3.4.4 Pain self-efficacy as a moderator.

Rose and colleagues (2004) suggested that resistance factors can act as moderators that specify the conditions under which a risk factor exerts a negative influence. Only two studies were identified that have examined a moderating role of pain self-efficacy on relationships between risk predictors and pain-related physical function and QOL. Lowe and colleagues (2008) suggested that the level of self-efficacy belief may influence expression of coping strategy in those with rheumatoid arthritis. For example, these authors suggested that acceptance could be expressed either as surrender to pain or as patient tolerance of circumstances with anticipation of improved outcomes. They hypothesised that differences in coping expression may differentially influence pain-related affect. Lowe and colleagues (2008) thus examined whether pain self-efficacy moderated the relationship between coping and anxiety and depression in 127 individuals with rheumatoid arthritis. Moderation was tested using regression analysis and the guidelines of Baron and Kenny (1986). Significant interactions between coping and pain self-efficacy were found. Specifically, acceptance coping was associated with lowered depression only in individuals reporting high self-efficacy beliefs. Lowe and colleagues (2008) suggested that efficacy expectations may influence both the choice of coping strategy and its associated cognitions.

A much earlier study by Marlowe (1998) investigated general self-efficacy as a moderator of the relationship between stressful events and headache frequency in 114 community-dwelling headache sufferers. Moderation was tested via interaction terms in regression analyses. Variables were not mean centred, as has been suggested for moderation analyses in order to reduce effects of multicollinearity in regression models and to improve interpretability of co-efficients (Aiken &

West, 1991). Despite these issues, self-efficacy was a significant moderator of the stressful events-headache relationship. That is, stressful events was significantly associated with headache frequency in those with low self-efficacy but was not significant in those with high self-efficacy.

Research from non-chronic pain fields has also demonstrated a buffering effect of self-efficacy on stressor-adjustment relationships. For example, Prati, Pietrantoni and Cicognani (2010) demonstrated a buffering effect of self-efficacy on the relationship between stressor appraisal and QOL in 451 Italian rescue workers. Moderation was tested using multiple regression analyses using interaction terms. Significant interactions were probed using a statistical software macro called MODPROBE (Hayes & Matthes, 2009). The negative effect of stress appraisal on QOL was found to only be significant for rescue workers with low self-efficacy, demonstrating that high self-efficacy buffers the impact of perceived stressful encounters on professional quality of life.

2.3.4.5 Summary of evidence pertaining to pain self-efficacy.

In summary, multiple single studies and a large meta-analysis have demonstrated that strong direct links exist between self-efficacy, pain-related physical function and QOL. These effects have been consistently observed across a broad range of chronic pain diagnostic groups. Self-efficacy has also been shown in multiple studies to mediate the relationship between pain severity and pain-related function in varied groups of pain patients. Evidence also exists to show that the effects of other predictors such as catastrophising and optimism on pain-related physical function may also be expressed at least partially, via a negative association with pain self-efficacy. It appears likely that in addition to acting as a mechanism through which the effects of risk predictors on adjustment are expressed, pain self-efficacy may also buffer risk-adjustment relationships. This hypothesis is consistent with the current conceptual model, however, very little

research has so far examined it. Therefore, no conclusions about this potential effect can currently be made.

2.3.5 Stress-processing factors – pain acceptance.

Acceptance generally has been defined as 'willingness to experience difficult thoughts, feelings, body sensations, and memories without defense' (McCracken & Thompson, 2011). Pain acceptance is a pain coping strategy that is described as being comprised of two aspects; pain willingness and activities engagement (McCracken, Vowles & Eccleston, 2004). Pain willingness refers to the extent to which pain is allowed into internal experience without attempts to control it, while activities engagement refers to the extent to which life activities are maintained despite pain (Kranz, Bollinger & Nilges, 2010).

Over the past decade the concept of acceptance has become increasingly important in chronic pain research and treatment (Kranz et al., 2011). Acceptance may be important for adjustment because a lack of willingness to accept pain may be associated with continued seeking of interventions aimed at cure, which may in turn result in missed opportunities for a satisfying life (McCracken, 1998). Prior to reaching a stage of acceptance, cure-seeking is often reported by those living with chronic pain (Osborn & Smith, 1998). However, it has been suggested that when living with chronic pain, better QOL might be achieved if a complete cure for chronic pain was acknowledged to be unachievable, and resources were directed instead towards the pursuit of more satisfying life activities despite pain (McCracken et al., 2004).

Acceptance has been strongly linked to both pain-related physical function (McCracken & Eccleston, 2003, 2006) and to pain-related affective experiences. It has been suggested that pain willingness is associated with reduced NA whilst continued engagement in life despite pain has been linked to increased PA (Kranz et al., 2011). Acceptance is a key tenet of the psychological

flexibility model of chronic pain (McCracken & Morley, 2014). This model is outlined in Section 1.5.1 in Chapter One. It proposes that acceptance is linked with other psychological processes such as mindfulness and values awareness and together, these factors influence pain-related function (Vowles, Sowden & Ashworth, 2014). Psychological flexibility has been defined as the ability to experience thoughts and emotions in the present moment and to adapt behaviour according to personal values (Vowles & McCracken, 2010). Empirical studies evaluating the Psychological Flexibility model of chronic pain as a whole are reported below, followed by research that has addressed the association between acceptance as a predictor of pain-related function and QOL.

2.3.5.1 Empirical studies of acceptance and physical function: direct effects.

A recent study that evaluated the overall capacity of the psychological flexibility model of chronic pain to explain adjustment outcomes offered insight into ways in which acceptance may confer adjustment benefits for those with chronic pain. Vowles and colleagues (2014) used structural equation modelling to explore the utility of the psychological flexibility model for explaining pain-related function in a heterogeneous group of 274 pain clinic patients. Outcomes were pain-related disability, affective distress and pain severity. Providing support for the model as a whole, acceptance, mindfulness and values-based action were moderately correlated and together all predicted disability, although the strength of the association between values and mindfulness with disability was stronger than that of acceptance.

A large amount of research has explored acceptance as a predictor of pain-related physical function. McCracken (1998) published one of the first studies that introduced the concept of acceptance to the chronic pain literature. This study of 160 treatment seeking individuals with chronic pain used a preliminary 34 item version of the Chronic Pain Acceptance Questionnaire to explore the predictive capacity of acceptance for a range of chronic pain outcomes that included

physical disability (McCracken, 1998). Acceptance significantly predicted pain-related disability in a regression analysis, even after adjusting for demographic factors and pain severity.

Several studies have investigated the differential effects of acceptance compared to other pain coping strategies. McCracken and Eccleston (2003) compared the influence of acceptance to distraction, reinterpreting pain sensations, coping self-statements, ignoring pain, praying and hoping and increasing behavioural activity in a heterogeneous sample of 230 pain patients recruited from a pain management centre. Acceptance was a significant predictor of pain-related disability together with praying and hoping, coping self-statements and reinterpreting pain. A later study by the same authors with the same sample but different measures of acceptance and coping, reported similar results (McCracken & Eccleston, 2006).

Esteve, Ramírez-Maestre and López-Martínez (2007) compared the influence of acceptance to catastrophising, coping self-statements and active and passive coping approaches on pain-related physical impairment in 117 pain clinic patients with mixed pain diagnoses. Structural equations modelling was used to explore a model in which acceptance, coping strategies and pain severity predicted functional status, functional impairment, anxiety and depression. Of the coping variables, only acceptance contributed to the variance in functional status. Together, pain severity and acceptance explained 18% of the variance in a measure of functional impairment.

The same research group (Esteve & Ramirez-Maestre, 2013) found the relationship between acceptance and physical function varied according to diagnostic and adjustment profiles of individuals with chronic pain. Using structural equations modelling, they examined a model in which acceptance and fear-avoidance were tested as predictors of function and negative mood in three groups of pain patients - 128 gastro-intestinal patients with irritable bowel disease, 141 primary care patients with chronic back pain and heterogeneous group of 137 pain patients

recruited from a pain management unit. Pain acceptance was significantly and positively associated with daily functioning only in the patients with back pain, although was significantly and negatively associated with negative mood in patients with back pain and irritable bowel disease. Negative links between acceptance and adjustment were absent in the group of pain clinic patients who reported the worst physical and emotional adjustment to pain. These results suggest that the protective effects of acceptance may be lessened in patients who are experiencing high levels of pain-related disability and distress although may be an important resistance factor for physical function and mood in less disabled groups of patients.

2.3.5.2 Empirical studies of acceptance and quality of life: direct effects.

Acceptance based interventions have also been shown to confer benefits for QOL for those with chronic pain. These relationships were demonstrated in a study that evaluated the overall predictive capacity of the Psychological Flexibility Model of chronic pain for explaining variance in a measure of HRQOL (McCracken & Velleman, 2010). Health-related QOL was measured with the SF-36 (Ware et al., 1993). 'Psychological flexibility' was represented by measures of acceptance, values-based action and mindfulness. The sample consisted of a heterogeneous sample of 239 pain-affected primary care patients. Regression analyses demonstrated that all three of these facets of psychological flexibility, together with pain severity, predicted 28% of the variance in physical component score of the SF-36. After pain severity, pain acceptance demonstrated the strongest relationship with physical function.

Evidence for a protective effect of acceptance on QOL can also be found in a meta-analysis of research investigating the effect of acceptance-based intervention on adjustment outcomes (Veehof et al., 2011). This review included 22 studies with a total sample size of 1235 patients. Studies were included if they investigated the effectiveness of an acceptance or mindfulness based

treatment program for patients with chronic pain of any diagnosis. Both controlled and non-controlled studies were included. Excluded studies were those that consisted only of a single treatment session, lacked an abstract, or provided insufficient data to calculate standardized mean differences. Outcomes included pain severity, depression and anxiety levels, QOL and physical wellbeing. Study quality in this meta-analysis was assessed according to criteria provided by the Cochrane Collaboration (Higgins & Altman, 2008) and the Jadad scale (Jadad, 1996). Included studies were graded as high, medium and low. Two studies scored high, eight scored medium, and 12 scored low on the quality criteria. Of these, only five studies specifically examined the effect of the intervention on QOL. It was also not possible to separate effects specific to acceptance, as many interventions also involved mindfulness components. Despite these shortcomings, a small and significant positive effect was identified of the interventions on QOL.

Only a small number of other studies not included in the above meta-analysis were identified that investigated the direct relationship between acceptance and QOL using cross-sectional data in those with chronic pain. Mason, Mathias and Skevington (2008) examined this relationship in 86 pain clinic patients with chronic low back pain with a mean age of 54.3 years. Quality of life was measured with the World Health Organization Quality Of Life Assessment (WHOQOL) – Pain. This scale is composed of the generic WHOQOL-100 profile, which consists of 25 facets of QOL assessed in six domains. In addition to these standard items, four additional facets assess pain and its impact on QOL. A moderate strength positive correlation was reported between measures of acceptance and overall QOL. Regression analyses were used to assess the contribution of pain willingness and activities engagement after controlling for the effects of pain severity. In regression analyses, both acceptance and pain severity were significant predictors of

the physical WHOQOL domain. However, of the two acceptance subscales, only pain willingness but not activity engagement predicted physical QOL.

Qualitative research has identified acceptance as an important process for promoting adjustment to chronic pain. LaChappelle, Lavoie and Boudreau (2008) explored the process of acceptance in a group of 45 women recruited from the general community. Acceptance was identified as helpful for coping, and was reported to incorporate diagnosis, realisation that no cure was available and then, redefinition of a 'new normal'. Education, diagnosis and social support were all reported to promote acceptance.

2.3.5.3 Acceptance as a mediator.

Acceptance has been examined as a both a mediator and moderator of relationships between risk predictors and physical function in chronic pain. For example, Fish, Hogan, Morrison, Stewart and McGuire (2013) investigated acceptance as a mediator of the relationship between pain severity and pain-related interference in a heterogeneous community-based group of 550 individuals with chronic pain. Similar to the current research, participants were recruited online from pain support groups and forums. Structural equation modeling was used to test the study hypotheses. The model that best represented the data was one in which pain severity was both directly and indirectly associated with interference. The indirect association of pain severity with interference occurred via a negative association with pain acceptance. When pain severity predicted anxiety and depression, the relationships between these two factors was expressed entirely by their negative association with pain acceptance. These results indicate that pain acceptance represents a mechanism through which the relationship between pain and disability, anxiety and depression is expressed and highlights the importance of addressing it in treatment.

Elander, Robinson, Mitchell and Morris (2009) tested the two subscales of a measure of acceptance, pain willingness and activities engagement, as both mediators and moderators of the relationships between pain severity, pain coping, negative pain thinking and HRQOL in a sample of 209 individuals with haemophilia related joint pain. Health-related QOL was measured by the SF 36 (Ware et al., 1993); which provides subscale scores for physical and mental aspects of quality of life (SF-36 PCS and MCS scales). Moderation and mediation were tested via regression analyses. In terms of direct effects, pain willingness explained a small amount of variance in the measure of physical HRQOL over that contributed by pain severity. A weak mediating effect of pain willingness was noted in the relationship between pain severity and physical HRQOL. That is, pain severity was directly associated with lower HRQOL but was also indirectly associated with this outcome via a negative association with pain willingness.

Vowles, McCracken and Eccleston (2008) also showed, in a sample of 334 pain clinic patients, that pain acceptance mediated the relationships between catastrophising and a range of outcomes that included anxiety, depression, pain-related disability and psychosocial disability. Few other studies were identified in non-experimental research that examined acceptance as a mediator of paths between other predictors and chronic pain adjustment outcomes. Most commonly, mediation effects of acceptance have been demonstrated in relation to treatment related change processes. In a study of 342 female and 67 male participants in a Swedish multidisciplinary pain rehabilitation program, acceptance, together with life control, affective distress and social support were tested as simultaneous mediators of treatment related change in pain-related interference, pain severity and depression. Pain interference, pain severity, and depression (outcome measures) over time were mediated by changes in pain-related acceptance, life control, affective distress, and social support. However, the strongest indirect effect was through acceptance (Akerblom, Perrin, Fischer & McCracken, 2015). Although not directly comparable to the hypotheses of the current research,

these results suggest that acceptance is an important mechanism through which therapeutic interventions achieve adjustment gains.

Vowles, Witkiewitz, Sowden and Ashworth (2014) used multilevel structural equation modeling to assess treatment-related change in 117 patients who completed an English multidisciplinary chronic pain rehabilitation program. Changes in measures related to treatment interventions were also examined as potential mediators of treatment outcomes over a three month time period. A range of different outcomes were examined including physical disability and pain acceptance was identified as a mediator of pre to post treatment changes in disability.

Using the cross-product of the coefficients approach with bootstrap resampling, Wicksell, Olsson and Hayes (2010) also showed that psychological inflexibility (conceptually, the opposite of acceptance) mediated treatment related change in a small sample of 21 participants with whiplash related chronic pain. Using the same approach in a later study by the same research group (Wicksell et al., 2013) psychological inflexibility mediated treatment related changes in another small sample of 40 women with fibromyalgia. These results together indicate that acceptance represents an important mechanism through which treatment changes in outcomes such as physical function are achieved and highlight the importance of acceptance as a target of clinical intervention.

2.3.5.4 Acceptance as a moderator.

Elander and colleagues (2009) also identified significant moderating effects of pain acceptance on the relationship between pain severity and HRQOL in a group of men with joint-related pain due to haemophilia. Mean age of the sample was 41 years. Two subscales of a measure of acceptance, pain willingness and activities engagement, were examined as moderators. A significant interaction was noted between pain willingness and pain intensity. Specifically, the relationship between pain severity and physical HRQOL was significant at each of the three levels

of the moderator (high, medium and low) but the strength of the relationship diminished as pain willingness increased, suggesting that pain willingness weakened the negative relationship between pain severity and physical HRQOL.

A moderating role of pain acceptance on the relationship between pain and NA was demonstrated in a study of 110 community-based women with osteoarthritis and fibromyalgia (Kratz, Davis & Zautra, 2007). Multi-level modelling was used to assess whether pain acceptance interacted with changes in pain to influence NA. Pain acceptance was demonstrated to moderate the relation between pain and NA, that is the association between weekly peaks in pain intensity and concomitant increases in NA were attenuated at higher levels pain acceptance.

Laboratory-based research has also demonstrated that high levels of pain acceptance buffer (dampen) the relationship between catastrophising and physical task performance. Richardson and colleagues (2010) examined pain willingness (a subscale of Chronic Pain Acceptance Questionnaire, CPAQ; McCracken et al., 2004) as a moderator of the relationship between catastrophising and both task performance and self-reported pain-related interference. The latter relationship was tested based on self-reported pain-related interference over the past week. The moderating effect of acceptance on the relationship between catstrophising and task performance was tested during an experimentally induced pain procedure. The sample comprised of 67 adults with chronic low back pain. A significant interaction was identified between pain willingness and catastrophising in the model predicting task performance but not the model predicting self-reported pain-related interference. However, when simple slopes of this significant interaction were plotted at three levels of pain willingness (low, medium and high) the strength of this relationship increased as acceptance decreased but none of the slopes differed significantly from zero. The authors suggested that the significant interaction noted in the invoked pain condition might be explained by a loosening effect of pain-willingness on negative pain-related cognitions and attention to pain

associated with high levels of catastrophising. That different results were noted for task performance during an experimental pain condition compared to self-report measures raises questions about the generalisability of self-report measures to real-life settings. The non-significant simples slopes indicates that further research is required in order to explore the nature of the moderating effect of acceptance on the relationship between catastrophising and task performance.

2.3.5.5 Relationship between pain acceptance and pain self-efficacy.

Acceptance is most commonly measured in research and clinical practice by various forms of the Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004). This questionnaire consists of two subscales; activities engagement and pain willingness. As is evidenced by the above research, pain acceptance appears to be an important aspect of chronic pain adjustment processes. However, as Nicholas (2006) highlighted, the extent to which acceptance processes overlap with other adjustment-related processes is unclear. In particular, pain self-efficacy is likely to be related to acceptance as both processes involve engagement in normal life activities despite pain. For example, items on the CPAQ (McCracken et al., 2004) include “I am getting on with the business of living no matter what my level of pain is” and “I lead a full life even though I have chronic pain”. Similar items on the PSEQ (Nicholas, 2007) include ‘I can live a normal lifestyle, despite the pain” and “I can enjoy things, despite the pain”.

However, Nicholas (2006) points out that the philosophical underpinnings of the two constructs differ substantially. Pain self-efficacy is a cognitive-behavioural concept that focuses on having confidence to engage in life activities despite pain (Nicholas, 2006) while acceptance is associated with an experiential component aimed at ‘allowing’ the internal experience of pain as opposed to resisting it (Block-Lerner, Wulfert & Moses, 2009). Thus, with acceptance it may be possible to engage in normal life activities despite pain even when confidence to do so is lacking.

The integration of constructs from varying paradigms offers pain research the opportunity to broaden understanding of adjustment processes. However, such integration relies on explicit understanding of the underlying epistemological assumptions (McCracken, 2013). It has been suggested that such research is best undertaken within the context of a unified model that guides it.

A number of studies have examined pain self-efficacy and acceptance as simultaneous predictors of adjustment. For example in a mixed pain clinic sample of 252 patients with a mean age of 50 years and pain duration of 94 months, Nicholas and Asghari (2006) used regression to investigate whether pain acceptance explained variance in pain-related disability after adjusting for the influence of pain self-efficacy. Predictors included catastrophising, fear of movement, pain self-efficacy, and the two aspects of pain acceptance - pain willingness and activities engagement. Pain self-efficacy was measured by the Pain Self-Efficacy Questionnaire (PSEQ, Nicolas, 2007). Acceptance was measured by the Chronic Pain Acceptance Questionnaire (CPAQ, McCracken et al., 2004). A strong positive correlation was noted between the PSEQ and CPAQ scores. Similar relationships have been reported by other authors, ranging between .60 (Nicholas & Asghari, 2006) to .73 (Fish et al., 2013).

In this study (Nicholas & Asghari, 2006), significant predictors of disability in the multi-variate context were pain self-efficacy, fear-avoidance and the activities engagement subscale of the CPAQ, but not the pain willingness subscale. The same study explored the capacity of pain self-efficacy and activities engagement and pain willingness aspects of pain acceptance for explaining variance in pain-related disability. Three separate regression equations were specified that controlled for age, pain severity, fear-avoidance, catastrophising and depression. In these analyses, only pain self-efficacy explained additional variance over that explained by the other predictors. Together, these results suggest that the activities engagement aspect of acceptance

explains a small amount of variation in disability scores over that explained by pain self-efficacy. However, when a range of other risk predictors related to disability are included in the analysis, pain acceptance may not explain additional unique variance in disability scores.

The same research group completed an international comparison of predictors of disability in two heterogeneous samples of 311 individuals all recruited from pain clinics (Sardá, Nicholas, Asghari & Pimenta, 2009). One sample was from Australia and the other from Brazil. Predictors in each sample included depression symptoms, catastrophizing, pain self-efficacy, pain acceptance and pain severity. In the Brazilian sample, predictors were measured by Portuguese translations of the same self-report scales. All scores fell within the middle range in both samples in comparison to normative data and the overall demographic and score profile of the samples were similar to each other. However, mean scores for depression and self-efficacy were significantly higher in the Brazilian sample, and scores for catastrophising were significantly lower. Hierarchical regression analyses in both samples showed that only pain variables (number of pain sites and pain severity) and pain self-efficacy made significant contributions to explaining variance in the measure of disability.

Similar results were reported by Kalapurakkel and colleagues (2014) in a study that examined pain self-efficacy and pain acceptance as resilience processes predicting headache. Predictors of headache-related disability and depression were examined in a sample of 207 individuals aged 17 years or less recruited from a multidisciplinary headache clinic. In the analysis predicting disability, pain was a significant predictor together with pain self-efficacy. This latter predictor explained an additional 29% of the variance in disability over that explained by pain. When pain acceptance was added to the analysis, it explained a significant but small amount (2%) of additional variance. By contrast, the regression equation predicting depression showed the opposite pattern. Pain, pain self-efficacy and pain acceptance were all significant predictors of

depression but pain acceptance demonstrated a stronger association with depression symptoms than did pain self-efficacy.

Together, these results suggest that in pain clinic and rehabilitation samples, pain self-efficacy is a stronger predictor of disability than acceptance, and that acceptance is a stronger predictor of depression. The above research have two implications for the current research. Firstly, pain self-efficacy and pain acceptance represent statistically related but theoretically distinct constructs. When examined together in multivariate models that also include predictors such as pain severity, depression, fear of movement or catastrophising, pain self-efficacy appears to be a stronger predictor of pain-related disability than pain acceptance. However, due to the lack of similar research in non-pain clinic samples, it remains unclear whether these findings can be generalized to these settings.

2.3.5.6 Summary of evidence pertaining to pain acceptance.

Acceptance is linked to improved physical function and QOL in chronic pain. These associations remain significant even after adjusting for demographics and pain severity. Few studies have examined the mediating or moderating capacity of acceptance on the relationship between risk predictors such as pain severity, NA or negative social experiences. However, a small amount of evidence exists to demonstrate that acceptance may both mediate and moderate the relationship between pain severity and adjustment. Acceptance has also been found to mediate treatment related change. There is limited evidence to show that acceptance may also moderate the relationship between catastrophising and pain-related function.

2.4 Demographic Covariates

2.4.1 Gender.

The prevalence of many chronic pain conditions varies by gender (Racine et al., 2012, Keefe et al., 2000). For example, women experience chronic headache, complex regional pain syndrome and fibromyalgia more often than men (Greenspan et al., 2007). Women also tend to report pain more intensely, frequently, and in more body regions (Blyth, March & Cousins, 2003; Breivik, Collett, Ventafridda, Cohen & Gallacher, 2006).

A systematic review exploring gender-based differences in pain-related function included six relevant studies that were all graded high quality according to the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines (El-Shormilisy, Strong & Meredith, 2015). In three high-quality studies, women were found to be more functionally impaired by pain compared to men. However, in the other three high quality studies no significant gender-based differences in function were noted.

However, other studies not included in the above review have found that female gender was associated with significantly increased risk for poor functional outcome following onset of sciatica in 283 participants, approximately one third of whom were female (Peul, Brand, Thomeer & Koes, 2008). Being female was also associated with slightly increased risk of poorer function post traumatic whiplash injury in 3704 adults (Berglund, Bodin, Jensen, Wiklund & Alfredsson, 2006) and with long term disability after a diagnosis of low back pain in a sample of 148 participants recruited from primary care settings (Enthoven, Skargren, Carstensen & Oberg, 2006). In a study of factors associated with disability in 396 American adults with arthritis recruited from primary care settings, male gender was predictive of lower disability levels, together with otherwise good health and a diagnosis of fibromyalgia (Baruth, Wilcox, Schoffman & Becofsky, 2013).

A small number of studies have explored gender effects on QOL, with reported findings somewhat different to those reported for disability or pain-related function. Van Velzen and colleagues (2014) investigated HRQOL in 975 patients with complex regional pain syndrome and found no effect of gender on this outcome. Similarly, in a study of 160 patients with injury-related chronic pain that examined relationships between pain severity, post-traumatic stress, depression, anxiety, disability and life satisfaction, no significant gender differences were reported for the outcome of life satisfaction (Stålnacke, 2011).

Despite a large body of research, there is currently limited evidence to support causal mechanisms that may drive these gender-based differences. A systematic review that examined potential causes of gender-based differences in pain outcomes included 129 experimental studies conducted in healthy participants and those with pain (Racine et al., 2012). Although hormonal, physiological and central nervous system processing factors were all identified as potentially explanatory mechanisms, the evidence was either inconsistent, insufficient or of very small effect (Racine et al., 2012). The same review considered a potential mediating role for cognitive and affective factors such as anxiety, depression, catastrophising and self-efficacy. Results in relation to anxiety were mixed, and thus no conclusions were made.

Evidence from a small number of studies in this review suggested that catastrophising may mediate gender differences in pain perception (Racine et al., 2012). Similar findings were reported in another small review by Sullivan and colleagues (2001). However, a more recent systematic review examining gender based differences in pain-related function identified that included seven relevant studies (El-Shormilisy, Strong & Meredith, 2015) found a trend towards stronger positive associations between catastrophising and disability for women, but these differences did not reach statistical significance.

It is also possible social factors may play a role in gender-based differences in pain because pain beliefs and coping are acquired, at least in part, by social learning which is influenced by gender (Racine et al., 2012). For example, men are traditionally expected to behave more stoically in relation to pain than women (Fillingim, 2000). Gender differences have also been noted to exist in spousal responding to pain. Wives, for example, have been shown to be significantly more likely to show facilitative behaviour in response to patient pain behaviour than husbands (Smith, Keefe, Caldwell, Romano & Baucom, 2004).

2.4.1.1 Summary of evidence pertaining to gender.

Differences in the reporting of pain severity and frequency for men and compared to women. Mixed results have been reported in reported in relation to gender-based functional differences related to pain. Precise causal mechanisms to explain these differences remain unclear although there is also a small amount of evidence to support the idea that catastrophising may mediate some of gender-based differences in pain reporting.

2.4.2 Age.

Although prevalence data (Blyth et al., 2001; Nahin, 2015) demonstrate that pain prevalence peaks in those aged more than 65 years, there is debate in the literature regarding the extent to which age impacts function and QOL in those with chronic pain. Some studies have suggested that pain-related interference increases with age (Thomas, Peat, Harris, Wilkie & Croft, 2004; Turk, Okifuji & Scharff, 1995), while other studies have demonstrated the opposite (Lachapelle & Hadjistavropoulos, 2005). Hunter (2001) completed a systematic review of demographic factors predictive of chronic pain and pain-related disability that included 13 studies and concluded that inconsistent evidence existed to support a relationship between increasing age and higher pain-related disability. It is possible these mixed results reflect either that pain may

constitute an additional risk factor for poorer function in older adults already at risk due to other age-related factors. However, it is also possible that reduced physical demands of older people produce less pain-related distress compared to younger people whose life goals may be more thwarted by pain and this may translate into functional gains. In summary, although some differences in pain processing and pain coping strategies have been noted in older compared to younger adults, there is contradictory evidence regarding the influence of age on pain-related disability or QOL.

2.4.3 Education.

Education attainment has also been suggested to influence pain adjustment outcomes, possibly because pain coping may be enhanced by cognitive skills strengthened by education (Cano, Mayo & Ventimiglia, 2006). Roth and Geisser (2002) found that education was inversely related to pain-related disability in a sample of 209 individuals with chronic spinal pain. Further, catastrophising mediated the relationship between educational attainment and pain-related disability. Specifically, individuals with lower levels of education were more likely to develop maladaptive pain cognitions (Roth & Geisser, 2002). These findings reflected those of Day and Thorn (2010) who found that a primary school literacy level in 115 Americans with chronic pain was associated with higher catastrophising.

Education was also found to be a predictor of clinical status (represented by a range of measures such as grip strength and walking time) in 385 patients with rheumatoid arthritis (Callahan & Pincus, 1988). The worst physical outcomes were noted for those individuals with only a primary school education but additional education over a standard undergraduate university degree conferred no outcome advantage. Lower education has also been linked to reduced HRQOL

in a sample of 359 individuals with early stage inflammatory joint disease (Geuskens, Burdorf, Evers, & Hazes, 2008) and rheumatoid arthritis (Linde, Sørensen, Østergaard & Hetland, 2008).

To summarise, lower levels of education appear to be linked to worsened pain adjustment outcomes. One possible mechanism for this influence might be that some clinical interventions rely on cognitive skills that might be enhanced by education (Cano, Mayo & Ventimiglia, 2006). Poorly educated individuals may also be more prone to using negative cognitive coping strategies.

2.4.4 Compensability.

Compensation and insurance systems have been suggested to negatively influence outcomes for injured individuals for several reasons including the possibility of financial gain, aggravation of pain by the stress of the claim process and a focus on winning the claim as vindication of injury, rather than on recovery (Teasell, 2001). It has also been suggested that it is not compensation per se that contributes to higher disability rates for injured workers, but rather a link between compensation and unemployment that explains the observed worsened outcomes (Dworkin, Handlin, Richlin, Brand & Vannucci, 1985). Other demographic factors related to compensation may also be influential because individuals seeking workers' compensation tend to have more physically demanding jobs, be younger, less educated and of lower SES (Teasell, 2001).

A large meta-analysis of post-surgical outcomes included 129 studies mostly in the areas of orthopaedic, plastic, and spinal surgery (Harris, Mulford, Solomon, van Gelder & Young, 2005). Outcomes were mainly functional, and included general functional and general health outcome scores. Ninety-five per cent of included studies reported a significant negative influence of compensation on outcome. These findings were consistent across differing procedures, countries, length of follow-up and type of compensation.

An earlier meta-analysis of compensation and perceived pain severity that included 32 studies found that compensation was reliably associated with increased pain and decreased treatment efficacy (Rohling, Binder & Langhinrichsen-Rohling, 1995). Study quality was reported as ranging between poor and excellent according to the internal and external validity, reliability of measurement and appropriateness of statistical analysis. Total sample size was 7651 participants. Effect sizes for compensation on pain ratings ranged between .50 and .60. Although this meta-analysis did not directly address the relationship between compensation and functional impairment or QOL, the well-established links between pain severity and these variables suggests potential relevance of compensation to these outcomes.

A number of single studies not included in the above reviews have also reported significant negative associations between compensation and chronic pain outcomes. For example, in an investigation of 158 pain clinic patients, Turk and Okifuji (1996) compared demographic and functional status for compensated and non-compensated individuals. Significant demographic differences between groups were identified; the compensated group had significantly more men, was younger and had shorter pain duration. Compensation was linked to higher levels of pain-related interference and disability.

Similarly, Atlas and colleagues (2006) examined longer term outcomes in 440 patients with low back and leg pain due to lumbar disc injury and found that those who received workers' compensation early in their injury trajectory reported worse disability and QOL than those who did not. Another prospective study of 192 individuals with chronic low back pain, compared pain and disability in those who received compensation to those who did not (Rainville, Sobel, Hartigan & Wright, 1997) and found that compensated individuals reported significantly more disability even after adjusting for baseline demographic differences between groups. In summary, compensation appears to be associated with more disability and reduced QOL in those with accident related

injuries although it is possible some of these observed associations may reflect links between compensation and other demographic variables such as employment status and education level.

2.5 Risk and Resistance Factors: Summary, Limitations, Future Research and Conclusions

In summary, the literature generally supports the direct pathways hypothesised by the current model. In particular, strong, direct and positive relationships have been demonstrated in the literature for most risk factors with pain-related disability. That is, as levels of most identified risk factors increase, pain-related disability also increases. For a smaller number of risk predictors, such as partner responses to pain, less evidence was available to support direct negative relationships with pain-related disability.

There was also less consistent evidence identified to support the hypothesised direct relationships between risk factors and QOL. Variations in results can likely be attributable to substantial variations in how QOL is operationalised and measured. Differences in sample characteristics introduce a further source of variability. Despite these issues, a direct negative relationship has generally been demonstrated to exist between both pain severity and catastrophising with QOL. However, there is currently little research to support relationships between other risk factors and QOL.

Considering direct effects of resistance predictors, a large body of evidence supports that a strong, direct relationship exists in the expected directions between pain self-efficacy and acceptance with both pain-related physical function and QOL. However, research in chronic pain populations supporting direct relationships between other resistance predictors, such as optimism and PA particularly with QOL is limited. This is despite the fact that the protective effects of these factors have been demonstrated for health outcomes more generally (Fredrikson, 2004; Rasmussen, Scheier & Greenhouse, 2009; Steptoe, Dockray & Wardle, 2009). Notably, there was no research

identified that has examined these relationships in a community based heterogeneous sample of adults with chronic pain. Thus, the current research will address some important deficits in the available literature.

In relation to indirect effects, the current model hypothesises that pain appraisal and coping factors, both risk and resistance, will mediate the relationships between other predictors and outcomes. A small amount of evidence exists to support a potential mediating role for the three appraisal processes, catastrophising, fear-avoidance and self-efficacy that is consistent with the hypotheses of the current model. That is, that pain appraisal and coping factors will mediate the relationships between other risk and resistance predictors and disability. However, the research examining the mediating role of catastrophising and fear-avoidance hypothesised in the current model is quite limited. To date this research has been undertaken in specific samples, not in mixed groups of pain patients. For example, only two studies were identified that examined a mediating role of catastrophising between the risk predictors in the model and physical function or QOL (Guite, McCue, Sherker, Sherry, & Rose, 2011; Mun, Okun & Karoly, 2014). A mediating role of pain self-efficacy has been well established in the literature. There is also evidence in a small number of studies to show that pain acceptance may function as both a mediator and moderator of the relationships between other predictors and pain adjustment outcomes.

The current model also hypothesises that resistance factors moderate the -relationships that exist between risk predictors and outcomes. There is currently very limited research that has examined these hypotheses. A small amount of evidence exists to demonstrate a moderating effect of optimism on the relationship between pain severity and pain-related interference (Cannella, Lobel, Glass, Lokshina & Graham, 2007), although at present there is as much evidence for a mediating role of optimism in this relationship (Ferreira and Sherman, 2007; Wong & Fielding,

2007). No research was identified that has examined a potential moderating effect of PA on the relationships between risk predictors and outcomes.

The current model hypothesises two effects that relate to the relationship between social support and adjustment outcomes. Firstly, that social support exerts a direct protective influence on pain-related function and QOL. Secondly, that social supports moderates the relationship between risk predictors and outcomes. The literature currently provides only partial support for these hypotheses. The influence of social factors on physical function and QOL for those with chronic pain appears complex. Research investigating the influence of social support on chronic pain outcomes has demonstrated consistent positive associations for QOL, but inconsistent effects have been reported for measures of physical function.

Inconsistent findings were also noted in the existing literature for the moderating effects of social support on the relationships between risk predictors and physical function and QOL. Inherent complexities operating within the construct of social support, such as attachment influences and appraisals of the pain and nature of the support, appear to be important in determining the influence of social support on pain-related disability and QOL. Further exploration of all these hypothesised mediator and moderator effects, both conceptually and empirically, appears critical to furthering understanding of variations in adjustment to chronic pain, and to further development of appropriate clinical interventions.

A number of demographic factors have been suggested to be associated with physical function and QOL for those living with chronic pain. Although there is inconsistent evidence to demonstrate that older age is associated with poorer function or QOL, a moderating effect of gender on pain outcomes has been consistently demonstrated. There is also evidence to suggest that both educational attainment and compensability may positively and negatively, respectively, directly influence chronic pain outcomes.

In summary, this modified version of Wallander's risk-resistance model of adjustment (Wallander et al., 1989; Wallander & Varni, 1998) provides a useful framework through which a large body of literature could be reviewed, analysed and integrated. The model offers the opportunity to bring together a range of different predictors from varying theoretical paradigms and ecological levels to examine their combined predictive effects on outcomes using a theory driven approach. Despite the heterogeneous nature of the research reviewed in terms of diagnoses, sample sizes and statistical approaches, trends in results were able to be recognised and considered in light of the hypotheses offered by the model.

Consequently, both the conceptual model, as well as the findings of the above literature review, drive the questions that underpin the current research. Specifically, using a cross-sectional approach, this research aims to explore the extent to which psychosocial risk and resistance factors combine directly, indirectly and interactively to explain variation in function and QOL in Australian adults with chronic pain.

2.6 Overall Research Questions

In summary, the overall questions arising from this review are:

Which psychosocial risk and resistance factors relate to variations in adjustment for those living with chronic pain in -

a) an Australian pain clinic sample and;

b) in an Australian community-based sample and;

c) how do these factors inter-relate in both samples to explain variations in adjustment?

In order to place the current results within the context of the existing literature outlined in Chapter Two, an additional aim of the research is to compare the reported disability levels of both groups with normative pain clinic data. The research questions pertaining to these comparisons are:

a) Do the reported disability levels of the two samples in this research differ from each other?

b) Do the reported disability levels of the two samples differ from published normative pain clinic data?

The next chapter, Chapter Three, describes the aims, hypotheses and methodology of Study One.

Chapter Three – Study One Rationale, Design and Method

3.1 Rationale

Study One aimed to extend the research in this field by conducting a preliminary examination of an adapted version of Wallander's risk-resistance adjustment framework (Wallander et al., 1989; Wallander & Varni, 1998) to explore direct and indirect effects of factors related to adjustment to chronic pain. This framework, which can be seen below in Figure 3.1 as it applies to Study One, was used to organise the literature reviewed in Chapter Two. The model offers a theoretically driven and inclusive approach through which direct, indirect and interactive effects related to chronic pain adjustment can be explored. It was used to guide the Study One hypotheses. A unique aspect of this research is that it considers a complex interaction of psychosocial factors.

An important aim of Study One was to identify mechanisms through which factors in the model may negatively or positively influence pain-related disability (mediation), as well as potential conditions under which negative effects of risk variables may be expressed on disability (moderation). These effects are currently under-researched. More specific understanding of the direct and indirect relationships between predictor variables and chronic pain outcomes may contribute to improved specificity of clinical interventions.

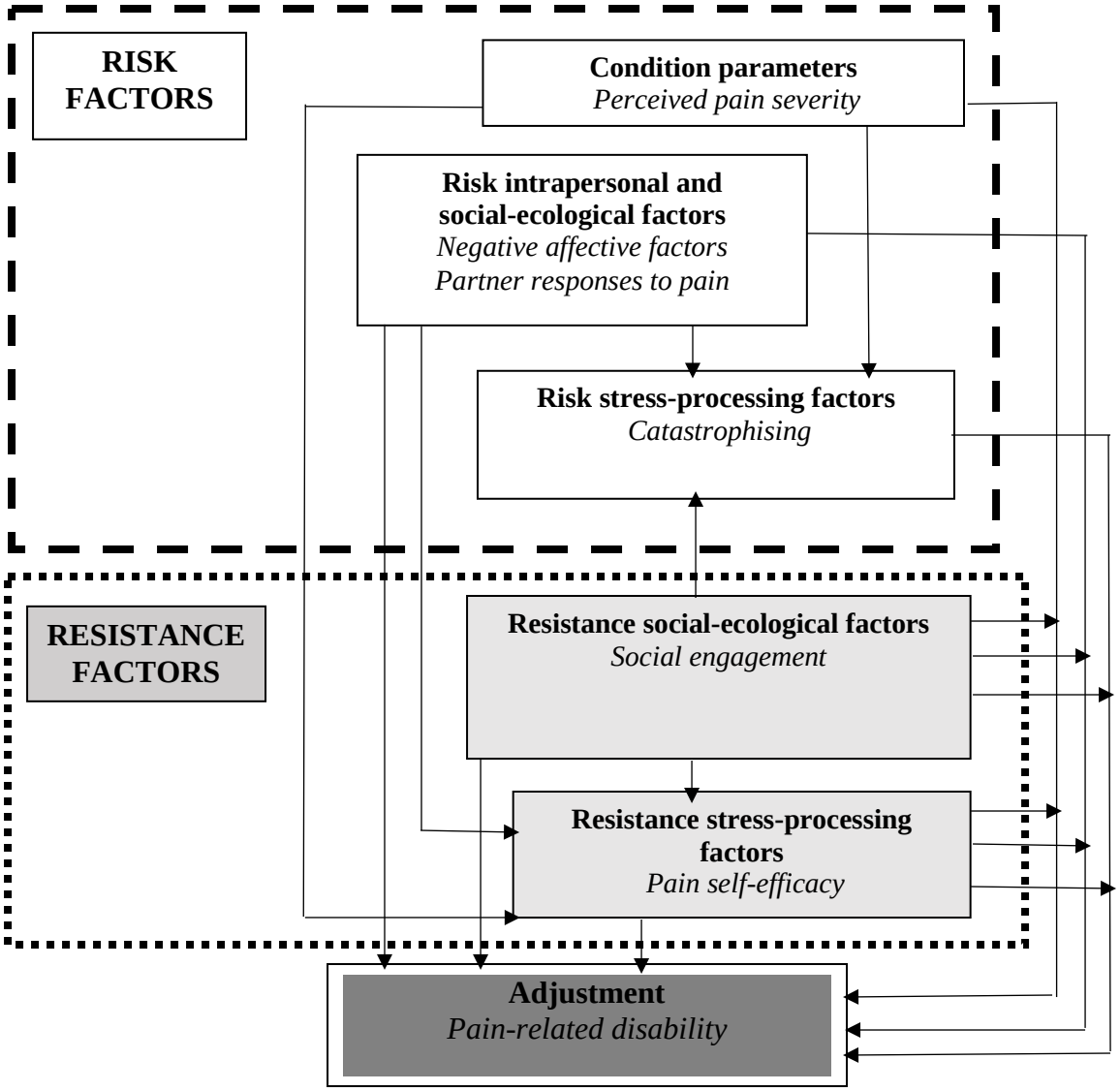

Figure 3.1 Proposed model of adjustment to chronic pain (adapted with permission from Wallander et al., 1989; Wallander & Varni, 1998).

Note 1: Study hypotheses are indicated by arrows.
Note 2: Existing literature was used to guide placement of variables within the model, hence stress-processing and social-ecological variables are placed as both risk and resistance factors.

3.2 Aims

Guided by the adapted risk and resistance conceptual model (Wallander et al., 1989; Wallander & Varni, 1998) depicted in Figure 3.1, Study One aimed to add to the chronic pain

literature by statistically investigating, using cross-sectional data, the direct, moderating and mediating effects of the identified risk and resistance factors on the pain-related disability of those living with chronic pain. Consistent with the theoretical model, three statistical effects of psychosocial factors on pain-related disability are hypothesised:

a. Direct effects
b. Mediated effects
c. Moderated effects

The research aims of Study One therefore are:

1. To assess the strength of the relationships between risk and resistance factors with pain-related disability.
2. To assess the extent to which the risk factors (perceived pain severity, catastrophising, anxiety, depression, solicitous and punishing partner responses to pain) together explain variation in pain-related disability.
3. To assess if the resistance factors (pain self-efficacy and social engagement) contribute unique variance to pain-related disability over and above that accounted for by the risk factors.
4. To explore whether the stress-processing factors (catastrophising and pain self-efficacy) mediate the effects of condition parameters, intrapersonal and social-ecological factors on pain-related disability.
5. To investigate whether the resistance factors (pain self-efficacy and social engagement) moderate the relationships between risk factors (perceived pain severity, catastrophising, anxiety, depression and solicitous and punishing partner responses) with pain-related disability.

6. A final aim was to compare the psychosocial and functional profile of the current sample to normative pain clinic data in order to place the current results in the context of the existing literature.

The remaining section of Chapter Three outlines Study One design, hypotheses and method.

3.3 Design

A within-group correlational design using cross-sectional data was used to investigate factors associated with pain-related disability. Hierarchical multiple regression was used to test the direct effects of predictors on pain-related disability. Mediating and moderating effects of risk and resistance predictors associated with pain-related disability were investigated using PROCESS (Hayes, 2013), a macro for SPSS that uses an ordinary least squares regression-based path analytic approach to estimate direct and indirect effects.

Risk factors were pain severity, catastrophising, anxiety, depression and punishing and solicitous partner responses to pain. Resistance factors were pain self-efficacy and social engagement. Several methodological issues arose in relation to the measures that needed consideration. Firstly, the social engagement factor was used as a proxy measure for social support. This measure assessed the extent to which individuals were engaged in social activities but not perceived support available through such interactions. It may have also assessed the functional capacity to socialise. Although this measure was not ideal, it was the only measure in the pain clinic database that could be used to broadly explore the construct of positive social support. This limitation was addressed in Study Three, where measures were specifically selected.

Secondly, risk and resistance factors associated with a measure of positive pain adjustment were unable to be explored in Study One as an appropriate measure was not available. However, because Study One aimed to explore a subset of effects proposed by the conceptual model before

testing a more comprehensive version in Study Three, one outcome measure was deemed acceptable. Quality of life was included as a measure of adjustment in Study Three.

3.4 Hypotheses

Study One hypotheses were guided by the conceptual model seen in Figure 3.1 and by the empirical literature reviewed in Chapter Two. The literature review demonstrated that negative relationships exist between perceived pain severity (Arnow et al., 2011; Raftery et al., 2011), anxiety (Lerman et al., 2015; Ocanez et al; 2010), depression (Meyer et al., 2007; Pincus et al., 2002), catastrophising (Jensen et al., 2011) and pain adjustment outcomes. There is evidence to suggest that solicitous and punishing partner responses to pain may also exert a negative influence on physical adjustment (Leonard et al., 2006). The literature review also demonstrated that a robust positive relationship exists between pain self-efficacy and pain-related disability (Burke et al., 2015). The evidence for a direct influence of social support on chronic pain adjustment outcomes is inconsistent (Campbell et al., 2011), however, social support may be associated with improved pain adjustment outcomes in some settings (Demange et al., 2004; Evers et al., 2003) and has been shown to exert protective effects on adjustment outcomes in broader health settings (Penninx et al., 1997). Therefore, in line with previous literature, the following hypotheses were proposed:

1. Higher levels of risk factors (pain severity, catastrophising, anxiety, depression, and punishing and solicitous partner responses to pain) will be associated with greater pain-related disability.
2. Higher levels of resistance factors (pain self-efficacy and social engagement) will be associated with lower pain-related disability.

Consistent with research suggesting that adjustment to chronic conditions is better predicted by a combination of risk and resistance factors than by single factors alone (Greenberg, Speltz, DeKlyen, & Jones, 2001; Wallander et al., 1989; Wallander & Varni, 1998), the third hypothesis is:

3. Resistance factors (pain self-efficacy and social engagement) will account for additional variance, over and above that explained by the risk factors (pain severity, catastrophising, anxiety, depression, and punishing and solicitous partner responses to pain), in explaining pain-related disability.

The current model proposes two types of indirect effects: a) mediator effects when some or all of the direct relationship between pain intensity, intrapersonal and social-ecological factors and disability is attributed to appraisal processes, and (b) moderator effects where the relationship between the risk factors and adjustment is moderated by levels of the resistance factors. Therefore the following indirect effects are hypothesised:

4. Self-efficacy and catastrophising will mediate the relationships between pain severity, anxiety, depression, partner responses to pain and social engagement with pain-related disability. Refer to Figure 3.2 below.

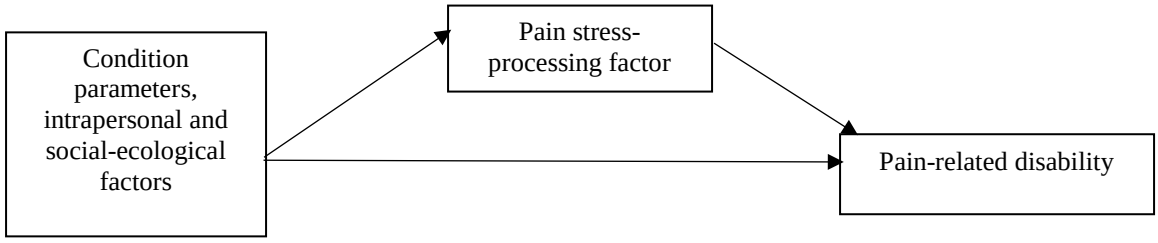

Figure 3.2 Mediator model.

5. Social engagement and pain self-efficacy, as resistance factors, will moderate, or lessen, the strength of the negative relationships that exist between pain severity, catastrophising, anxiety, depression, and partner responses to pain with pain-related disability. Refer to Figure 3.3 below.

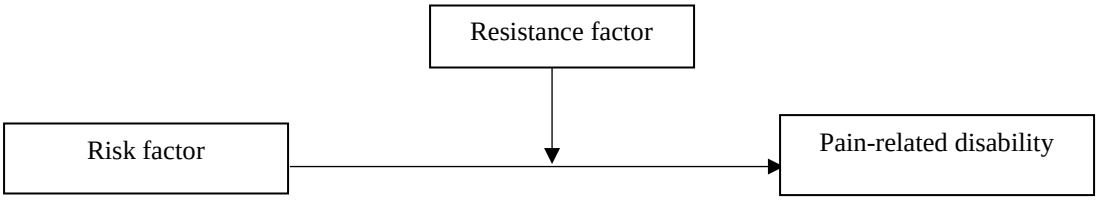

Figure 3.3 Moderator model

6. A final hypothesis proposed that no significant differences will exist between measures of risk and resistance factors and disability in the current sample compared to normative data.

The hypothesised models are driven by the literature reviewed in Chapter Two and depict unidirectional relationships between predictors and outcomes. Although it is acknowledged these relationships may be better represented as bi-directional, unidirectional relationships allow a complex model such as the one guiding this research to be statistically explored.

3.5 Method

3.5.1 Participants

Participants in Study One were adults referred to a multi-disciplinary pain clinic for assessment of suitability to complete an intensive pain management program (PMP) between the years of 2000 to 2009. The PMP runs five days per week over three weeks duration, on an outpatient basis. The pain clinic is multidisciplinary and PMPs are aimed at improving physical and psychological

functioning. Individuals aged over 18 years, with chronic, non-malignant pain conditions of more than three months duration were eligible for assessment. Thus, the Study One analyses were not based on original data but instead used pre-collected clinical data obtained from the Barwon Health Pain Management Unit. The Study One sample comprised 352 participants, 207 women and 145 men. Mean age of the sample was 51.61 (SD = 13.66) years. The majority of participants were born in Australia (82.5%). A small proportion of participants were from Europe (8.7%), the United Kingdom (5.0%), with the remaining participants reporting their country of birth was in Asia, Africa, North America or the Middle East. These data are largely consistent with 2011 Australian census data (Australian Bureau of Statistics, 2011) which showed 24.6% of the population was born overseas, with the four largest overseas birthplace groups being the United Kingdom, New Zealand, China and India. More than half the participants (59.4 %) were divorced, separated or widowed, whilst 29% were married or in a defacto relationship. These data differ from 2011 Australian census data (Australian Bureau of Statistics, 2011) which showed that of all people in Australia aged 15 years and over, 48.7% were married and 11.5% were either divorced or separated. Table 3.1 shows demographic details of the Study One sample.

Specific diagnostic information was not recorded but the patients referred for assessment for a PMP include those with disease and injury-related pain as well as non-specific pain conditions. Unfortunately, the proportion of participants with pain conditions compensable under either the Victorian Traffic Accident Commission or the Victorian Worksafe Authority was not reliably recorded within the clinic database and were therefore not able to be included in the analyses. Additionally, pain duration was not recorded in the pain clinic database and was also unable to be included as a variable in the analyses. These issues both represent limitations of Study One, as compensability has previously been found to be associated with poorer function in those with chronic pain (Harris et al., 2005; Rohling et al., 1995) and pain duration is also likely to

influence adjustment processes (Cano, 2004; Lee et al., 2008). These limitations were addressed in Study Three where both compensability and pain duration were recorded and their association with pain adjustment outcomes analysed.

Table 3.1

Demographic Details

Demographic		Number or mean (%)
Gender		
	Male	145 (41%)
	Female	207 (59%)
Age		
	Mean (SD)	51.61 years (13.66)
	Range	20 – 87 years
Marital status		
	Married / defacto	101 (28.7%)
	Single	26 (7.4%)
	Divorced / separated/ widowed	209 (59.4%)
	Missing	16 (4.5%)

3.5.2 Procedure.

Data were obtained from patients who completed a standard series of self-report questionnaires, in the same order, as part of their assessment process at the pain clinic. Thus, the original intention of the data was primarily for clinical purposes. These pre-collected data formed the basis of this study.

3.5.2.1 Ethics approval and intellectual property.

Ethics approval was obtained from both Barwon Health and Monash University Human Research Ethics Committees and an intellectual property agreement was signed (see Appendix A).

3.5.3 Measures.

3.5.3.1 Demographic factors.

Demographic factors reliably recorded in the pain clinic database were age and gender. Other demographic details that may have potentially influenced adjustment, such as education and compensability, were not reliably recorded and were therefore unavailable for analysis.

3.5.3.2 Measures of adjustment.

3.5.3.2.1 Pain-related disability.

Adjustment was operationalised as pain-related disability which was measured by a modified version of the Roland and Morris Disability Questionnaire (RMDQ; Roland & Morris, 1983). The original RMDQ was developed and validated by Roland and Morris (1983) for assessing the functional impact of back pain. The generic version of the questionnaire used in this study asks respondents to relate the items to their pain, regardless of its site. This modified RMDQ consists of 24 items, which are answered by checking if the item applies as true on that day. Example items include ‘Because of my pain, I use a handrail to get upstairs’ and ‘I get dressed more slowly than usual because of my pain’. The questionnaire is scored by summing the number of items checked as true to calculate one total scale score. Scores range from zero to 24, with higher scores indicative of greater pain-related disability. The RMDQ is reported to have acceptable concurrent validity with other measures of pain-related function, has high reported levels of test-retest reliability and has been demonstrated to be sensitive to change (Johansson & Lindberg, 1998; Roland & Morris, 1983). Cronbach’s alpha is reported as 0.92 (Roland & Morris, 1983), in this study it was comparable, calculated as .87.

3.5.3.3 Risk factors.

3.5.3.3.1 Condition parameters - perceived pain intensity.

Perceived pain intensity was measured by the three item Pain Intensity subscale of the West-Haven-Yale Multidimensional Pain Inventory (MPI; Kerns et al., 1985). Scores range from zero to six, with higher scores reflecting more severe pain over the preceding week. The subscale is scored by calculating the response mean of the three items. Example items include 'Rate the level of your pain at the present moment' and 'On the average, how severe has your pain been during the last week?' The MPI has established acceptable levels of reliability and concurrent validity (Kerns et al., 1985). Cronbach's alpha co-efficient for this subscale is reported as .72 (Kerns et al., 1985), which is consistent with the current study where it was also calculated as .72. Pallant (2010) points out that Cronbach's alpha values are sensitive to the number of items in a scale, with low Cronbach's alpha values not uncommon in scales with fewer than 10 items. For scales with a small number of items, the mean inter-item correlation represent a more appropriate measure of internal consistency (Pallant, 2010). The minimum recommended threshold for mean inter-item correlation is .30 (Nunnally & Bernstein, 1994), although Clark and Watson (1995) suggest a range of .15 to .50 is acceptable. Mean inter-item correlation for the pain severity subscale of the MPI (Kerns et al., 1985) was .47 which indicates acceptable reliability of this subscale.

3.5.3.3.2 Intrapersonal risk factors - anxiety and depression.

Anxiety and depression were measured by the anxiety and depression subscales of the Depression Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995a). The original 42 item DASS has been demonstrated to have good reliability and validity (Lovibond & Lovibond, 1995a). The shortened 21 item version of the DASS, was used in this study, with items divided evenly into three subscales assessing anxiety, depression and stress. Each item described a symptom and

respondents are asked to indicate the frequency with which they have experienced that symptom in the past week. Items are scored between zero (*never*) and three (*almost always*). The subscales are scored by summing the item responses; higher scores therefore, reflect higher levels of anxiety, stress or depression. Anxiety subscale items include ‘I was worried about situations in which I might panic and make a fool of myself’ and ‘I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)’. Depression subscale items include ‘I felt I had nothing to look forward to’ and ‘I felt downhearted and blue’. The DASS 21 has good reported reliability, with Cronbach's alphas reported as .94 for the depression subscale and .87 for the anxiety subscale (Lovibond & Lovibond, 1995a). Acceptable convergent, concurrent and discriminant validity of the scale has been demonstrated (Antony et al., 1998; Brown, Chorpita, Korotitsch & Barlow, 1997). Cronbach’s alphas were calculated in the current data as .93 (depression) and .85 (anxiety).

3.5.3.3.3 Social-ecological risk factors – punishing and solicitous partner responses to pain.

Punishing partner responses to pain were measured by the punishing responses subscale in Section Two of the MPI (Kerns et al., 1985). Respondents are asked to score the frequency with which their partner engaged in negative or critical behaviours when the respondent was in pain. Scores range from zero to six, with higher scores reflecting greater frequency of that behaviour. The subscale is scored by calculating the response mean of the three items. Example items include (when I am in pain) ‘my partner expresses irritation at me’ and ‘my partner ignores me’. The MPI has established acceptable levels of reliability and concurrent validity (Kerns et al., 1985). Cronbach’s alpha for the punishing responses subscale of the MPI is reported as .84 (Kerns et al., 1985), in this study it was a comparable .85 indicating adequate scale reliability.

Solicitous partner responses to pain were measured by the solicitous responses subscale in Section Two of the MPI (Kerns et al., 1985). Respondents are asked to score the frequency with which their partner engaged in solicitous behaviours when the respondent was in pain. Scores range from zero to six, with higher scores reflecting greater frequency of that behaviour. The subscale is scored by calculating the response mean of the three items. Example items include (when I am in pain) ‘my partner asks me what he/she can do to help’ and ‘my partner takes over my jobs or duties’. Cronbach’s alpha for the solicitous support subscale of the MPI is reported as .78 (Kerns et al., 1985), in this study it was calculated as .86 indicating adequate scale reliability.

3.5.3.3.4 Stress-processing risk factor – catastrophising.

Catastrophising was measured by the nine-item subscale of the Pain Response Self-Statements Scale (PRSS; Flor, Behle, & Birbaumer, 1993) which assesses situation-specific cognitions related to pain coping efforts. All items are scored on a zero to five scale, according to how often respondents experience a particular thought when their pain is severe. Higher scores indicate more frequent catastrophising. Each subscale is scored by calculating the mean of the seven items. Examples of items on the catastrophising subscale include ‘I cannot stand this pain any longer’ and ‘I am a hopeless case’. The PRSS has demonstrated construct validity (Flor et al., 1993). Cronbach’s alpha of the catastrophising subscale has been reported as .92 (Flor et al., 1993). The Cronbach’s alpha value in this study was comparable .86 indicating acceptable reliability of this subscale.

3.5.3.4 Resistance factors.

3.5.3.4.1 Social-ecological resistance factor - social engagement.

Participants’ engagement in social activity was measured with the social activities subscale in Section Three of the MPI (Kerns et al., 1985). Four items measure the frequency with which

participants engage in social activities. Scores range from zero to six, where zero indicates respondents never engage in that activity and six indicates they do it very often. The subscale is scored by calculating the response mean of the four items. The four constituent items in the social engagement subscale of the MPI (Kerns et al., 1985) ask how often participants 'play cards or other games', 'visit friends', 'visit relatives' and 'visit a park or a beach'. Cronbach's alpha for this subscale is reported as .74 (Kerns et al., 1985), in this study when all four items were included it was .66. An acceptable lower threshold for Cronbach's alpha is suggested to be .70 (Nunally, 1967). However, due to the small number of items in this subscale, inter-item correlations were also assessed. Mean inter-item correlation for the social engagement subscale of the MPI (Kerns et al., 1985) was .32, suggesting adequate internal consistency of this subscale. Copies of all Study One questionnaires can be found in Appendix B.

3.5.3.4.2 Stress-processing resistance factor - pain self-efficacy.

Pain self-efficacy was measured by the Pain Self-Efficacy Questionnaire (PSEQ; Nicholas, 2007). This 10-item inventory assesses beliefs about the confidence to accomplish activities despite pain. The PSEQ is scored by summing the item responses on a scale of zero to six, where zero indicates not at all confident and six represents complete confidence. Possible scale scores range from 0 to 60, with higher scores indicating stronger self-efficacy beliefs (Nicholas, 2007). Items include 'I can enjoy things, despite the pain' and 'I can cope with my pain in most situations'. The PSEQ has good concurrent validity, reliability and internal consistency (Nicholas, 2007). Cronbach's alpha for the PSEQ is reported as high, .92 (Nicholas, 2007), in this study it was also high, .94.

3.5.4 Statistical analyses.

All data were analysed using the Statistical Package for the Social Sciences, Versions 20.0 and 24.0 (IBM Corp., 2010 & 2015). The risk only and risk-resistance models were explored using hierarchical multiple regression. Mediating and moderating effects were investigated using PROCESS (Hayes, 2013), a macro for SPSS that uses an ordinary least squares regression-based path analytic approach to estimate direct and indirect effects. Data preparation processes are described including the replacement of missing values. This is followed by a description of the approach used to investigate the study hypotheses. The following steps were used to address the research hypotheses:

1. Data were screened for missing values, these were replaced using the Expectation-Maximisation algorithm. All analyses were tested in the imputed as well as the case deleted datasets (Section 3.5.4.1).
2. Variable distributions were assessed. Required assumptions for correlational and multiple regression analyses were checked (Sections 3.5.4.2).
3. Power calculations were completed to ensure adequate sample size (Section 3.5.4.3).
4. Scale reliability was assessed (Sections 3.5.3.2, 3.5.3.3 and 3.5.3.4).
5. Single sample t-tests were used to compare the profile of the current sample to normative pain clinic data. (Section 3.5.4.4).
6. Pearson correlation co-efficients were calculated to assess direct bivariate relationships between predictor variables and disability (Section 3.5.4.5).
7. Hierarchical multiple regression was used to assess the proportion of variance explained by the risk-resistance model that was over and above that explained by the risk only model (Section 3.5.4.6).

8. Mediating and moderating effects were investigated using PROCESS (Hayes, 2013) (Section 3.5.4.7).

3.5.4.1 Data screening – missing data and univariate distributions.

Prior to assessing missing data, the data were screened for errors and negatively worded items were reversed scored. Missing data patterns were assessed using frequency tables, graphs and Little's Missing Completely at Random (MCAR) test (Little, 1988). The initial data set contained 376 cases. Nine cases were recorded in the database twice and subsequently deleted. Thirteen further cases were deleted because one or more of the entire scale items were missing. One case was deleted as the subject was under 18 years of age, leaving 352 cases for analysis.

The proportion of missing data for most variables was generally low and less than 5%. The anxiety and depression subscales of the DASS (Lovibond & Lovibond, 1995) were missing 7% and 10% of values respectively. A high proportion of missing data (approximately 25%) was also noted in the solicitous and punishing partner responses subscales of the MPI (Kerns et al., 1985). Little's test (Little, 1988) of these subscales indicated that the data on these subscales were not missing completely at random (NMCAR). The high rate of missing data on these subscales may have been because the version of the MPI (Kerns et al., 1985) administered to pain clinic patients, unlike the original version of the scale, did not instruct participants to reference the person with whom they were closest, regardless of whether they were partnered. Thus, people who were not partnered at the time of assessment may have left these responses blank thinking that they needed to be partnered. This issue was addressed in Study 3 as a statement instructing participants to reference the person with whom they were closest was included.

All missing data were replaced using the Expectation-Maximisation algorithm (EM). The EM algorithm uses the means, variances, and co-variances of the available data to calculate

replacement values and is an accurate and robust procedure for replacing values that are missing at random (MCAR, Graham, 2009). In support of the use of imputation to replace NMCAR data, Graham (2009) argues that imputation may produce comparatively less biased results than case deletion. To assess effects of the imputation on results, two data sets were created; a fully imputed one and a second one where cases with missing partner data were deleted. Analyses were then completed in both databases. With one exception, no significant statistical differences were found between the two datasets, therefore, for all but one of the analyses, only results from the imputed dataset are reported. One of the mediation effects changed from being marginally significant, and of small magnitude, to statistically non-significant when calculated using the case deleted dataset. Although the clinical implications of both sets of results are the same, both results for this analysis were reported. See Appendix C for full details regarding missing data analysis and imputation.

Univariate distributions of all variables were examined in both datasets, imputed and case deleted, to ensure assumptions were not violated in either set. Skewness and kurtosis values, together with Shapiro-Wilk and Kolmogorov Smirnoff univariate normality statistics were assessed. Plots were also inspected to ensure normality. These included histograms with normality plots, box plots and detrended normal quantile-quantile (Q-Q) plots. A small number of univariate outliers were detected in the anxiety subscale of the DASS (1.42%) and the pain severity (1.14%) and social engagement subscales of the MPI (.85%). These were assessed for their influence on results by creation of an additional dataset that replaced the outliers values with the series mean as is suggested by Tabachnik and Fidell (2013). When the analyses were repeated with these outliers removed, results were unchanged, therefore these cases were retained in the analyses.

3.5.4.2 Assumption tests – univariate and multivariate.

Tests of normality indicated that most variables demonstrated substantively normal distributions. Kurtosis and skewness values all fell within the recommended range of +/- 1.5 (Tabachnik & Fidell, 2013, Appendix C). Inspection of frequency histograms with normality plots revealed approximately normal distributions of most variables, however a rectangular distribution of the depression subscale of the DASS (Lovibond & Lovibond, 1995) was noted. However, because skewness and kurtosis values of this variable were both below 1.0 (.43 and -.96 respectively), a decision was made not to transform this variable. The Kolmogorov-Smirnov and the Shapiro Wilk statistics for age were non-significant, indicating a normal distribution. These statistics for the remainder of the variables were statistically significant. This is suggestive of non-normality, although significant values of these statistics are not uncommon in larger sample sizes of greater than 200 and inspection of plots has been recommended as a better assessment of distribution (Pallant, 2010). Details of Study One normality screening can be found in Appendix D.

The assumption of equality of variance was met for *t*-tests. For all correlations, bivariate scatterplots of the relationships were inspected to ensure linearity and homoscedasticity. These assumptions were met. Examination of the normal probability plots and scatterplots of the standardised residuals in the hierarchical regression analyses indicated that the assumptions of linearity, homoscedasticity and independence of residuals were not violated. The assumptions of multicollinearity and singularity were satisfied. These were tested by checking correlation coefficients to ensure that they did not exceed the recommended levels of r = .90 (Tabachnik & Fidell, 2013). Variance inflation factors and tolerance statistics were also checked and were all well below the recommended threshold of 10 (Hair, Black, Babin & Anderson, 2010). Standard errors

of the regression co-efficients were also checked to ensure they were not excessively large, as these can become inflated when issues with multicollinearity occur (Tabachnik & Fidell, 2013).

The significance level of statistical tests, that is, the alpha level, was set at .05 unless otherwise specified. Given multiple tests were used a Bonferroni adjustment of the alpha level could have been conducted however this was not deemed necessary because all analyses were specified a priori. It has also been suggested that Bonferroni adjustments should not be used even in the setting of multiple tests because the Type 1 error rate cannot be decreased without increasing the Type II error rate, which is equally as incorrect (Perneger, 1998). As a way of increasing confidence in the reliability of estimates of indirect effect sizes, bootstrapped confidence intervals were used in place of alpha levels.

It is important in multiple regression to ensure that any multivariate outliers in the dataset are not exerting undue influence on results (Tabachnik & Fidell, 2013). Mahalanobis distances were calculated to detect multivariate outliers. The critical value for Mahalanobis distance for the variables in Study One was 26.13 as indicated by the Chi square critical values table for 8 predictors at the alpha level of .001 (Tabachnik & Fidell, 2013). Only four cases were noted to be above the critical value (27.04, 29.15, 30.36 and 38.86). A decision was made to retain these multivariate outliers in the data set because these values were clustered together and did not greatly exceed critical values. To ensure the outliers on Mahalanobis distances did not influence results, the regression and indirect effects analyses were examined in a dataset that had these values deleted. Results were unchanged. The decision to retain the multivariate outliers was also supported by an analysis of Cook's distances. Tabachnik and Fidell (2013) suggest that cases with values greater than one are problematic. No values were noted greater than .06.

3.5.4.3 Power calculations.

Power calculations are used to estimate the minimum sample size required to provide a reasonable likelihood of detecting a given effect size (Tabachnik & Fidell, 2013). Power calculations in the current research were made using GPOWER (Faul, Erdfelder, Lang & Buchner, 2007). The hierarchical multiple risk-resistance multiple regression model contained the largest number of predictors, which was 8. The ability to detect moderate to large effect sizes (between .15 and .35) in a multiple regression analysis with 8 predictors at the level of .95 power was estimated as requiring a sample size of between 74 and 160 participants. Therefore the current sample size of 352 participants, which well exceeded the estimated required sample size, was assumed to provide sufficient power for multiple regression analyses.

3.5.4.4 Comparison of the current sample to normative data.

In order to place the current results in the context of the existing literature, Study One data were compared to normative pain clinic data using single sample t-tests.

3.5.4.5 Bivariate relationships between predictors and pain-related disability.

Hypothesis One proposed that higher levels of risk factors (pain severity, catastrophising, anxiety and depression and punishing and solicitous partner responses to pain), would be associated with greater disability. Hypothesis Two proposed that higher levels of resistance factors (pain self-efficacy and social engagement), would be associated with lower levels of disability. These two hypotheses were examined by using Pearson's product-moment correlations using total scale scores. Varying recommendations have been made in relation to interpretation of the strength of correlations between variables (Pallant, 2010). The guidelines adopted in this thesis were those provided by Cohen (1988), which suggest that small correlations are those ranging between .10 to 29, medium correlations range between .30 and .49 and large correlations are those above .50.

3.5.4.6 Risk only and risk-resistance models.

Hypothesis Three proposed that the resistance factors, pain self-efficacy and social engagement, would account for additional variance, over and above that explained by the risk factors (pain severity, catastrophising, anxiety and depression and punishing and solicitous partner responses to pain), in pain-related disability. This hypothesis was examined using hierarchical multiple regression. A hierarchical model was analysed such that demographic predictors were entered in Step One, followed by risk predictors in Step Two and resistance predictors in Step Three. The amount of variance explained by both the risk only and the risk-resistance model was assessed and the statistical significance of individual predictors was examined.

3.5.4.7 Testing indirect effects.

Analysis of indirect effects, mediation and moderation, depicted in the adapted model (see above Figure 3.1) were conducted with PROCESS (Hayes, 2013), a macro for SPSS. PROCESS uses an ordinary least squares path analytical framework to test both direct and indirect effects (Hayes, 2013). PROCESS was an ideal statistical tool for the current research because it facilitates exploration of models with more than one mediator, as well as moderation effects and allows adjustment for covariates.

A particular strength of using PROCESS to estimate indirect effects is that bootstrap confidence intervals are used. While the Sobel test is still widely used to assess mediation effects in regression, Preacher and Hayes (2008) state that bootstrap confidence intervals are preferable because, unlike the Sobel test, they do not impose distributional assumptions on the indirect effects. The bootstrap technique creates multiple subsamples from an original database and enables the examination of parameter disturbances relative to each of these spawned samples (Byrne, 2010). This effectively acts as a proxy re-sampling technique. In this research, mediation and moderation

effects were tested using 10,000 bootstrap iterations and 95% bias-corrected confidence intervals. The number of bootstrap iterations used in the current research is substantially larger than the 1000 recommended by Efron and Tibshirani (1986) as the minimum number of iterations required to construct reliable bias-corrected confidence intervals. The large number of bootstrap iterations used in the current research therefore increases reliability of estimates of indirect effects. Results were interpreted as statistically significant if the bias-corrected confidence intervals did not include zero (Hayes, 2013). As stated in Chapter One, an important caveat with all analyses presented in this thesis applies to the condition of causality. Because of the cross-sectional nature of the data used in this research, causality between a predictor variable and an outcome cannot be inferred. Therefore, any comments regarding an influence of a predictor on an outcome in this thesis refer to statistical relationships only and not cause and effect relationships. As Hayes (2013) points out, the types of analyses contained in this research facilitate exploration of relationships between variables, which remains worthwhile even when definitive cause-effect relationships are unable to be established.

3.5.4.7.1 Mediation effects.

Of interest in Study One were mediation effects. A mediator is an explanatory link in the relationship between one variable and another. It can be conceptualized as a mechanism through which a predictor influences an outcome (Rose et al., 2004). Hypothesis Four proposed that pain self-efficacy and catastrophising, would mediate the relationships between condition parameters, intrapersonal and social-ecological factors (pain severity, anxiety, depression, solicitous and punishing partner responses to pain and social engagement) and pain-related disability. Mediator and moderator effects were only assessed for predictor-outcome relationships that were correlated at moderate or greater strength ($r \geq .30$) as this indicated that a relationship existed to be mediated

or moderated (Holmbeck, 2004). Specifically, the current study used PROCESS (v2.13) Model Four (Hayes, 2013) to test mediation effects in single and then parallel mediator models.

Simple mediation analyses were conducted first. Separate models were specified for each risk and resistance factor acting as a predictor of pain-related disability, examining first catastrophising and then pain self-efficacy as mediators. These simple three variable models assessed the extent to which catastrophising and pain self-efficacy acted as a mechanism through which the effect of each predictor was expressed indirectly on pain-related disability.

Where significant indirect effects of both mediators were observed, these effects were subsequently explored in parallel mediator models. In these latter models the predictor variable is hypothesised to exert its effect on the outcome partly via at least two mediators which are often correlated (Hayes, 2013). A condition of the analysis is that no mediator causally influences the other but mediators may be correlated (Hayes, 2013). Parallel mediator models reflect a situation where more than one mediating factor may be operating simultaneously. The direct effect of the predictor on the outcome is assessed when the effects of the mediators are controlled (Hayes, 2013). Estimation of simultaneous mediation effects allows for more specific targeting of clinical resources for rehabilitation interventions as these models identify which of multiple possible influences are the mechanisms through which the predictor in the model is influencing the outcome.

PROCESS (Hayes, 2013) also permits formal testing of differences between specific indirect effects. Indirect effects in PROCESS (Hayes, 2013) are scaled in terms of the metrics of the predictor and outcome variables in the equation (Hayes, 2013), therefore mediation effects with the same predictor and outcome measures can be compared, even if the mediators are measured on different scales. PROCESS (Hayes, 2013) provides confidence intervals for pairwise comparisons of specific indirect effects, indicating if one indirect effect is statistically significantly different from another. A confidence interval that does not contain zero provides evidence of a statistically

significant difference between indirect effects (Hayes, 2013). Importantly, a statistically significant difference between effects does not necessarily imply that one effect is larger than the other. Hayes (2013) points out that such comparisons are only possible if the point estimates for the two indirect effects are in the same direction. A pictorial representation of a parallel mediator model is depicted below in Figure 3.4.

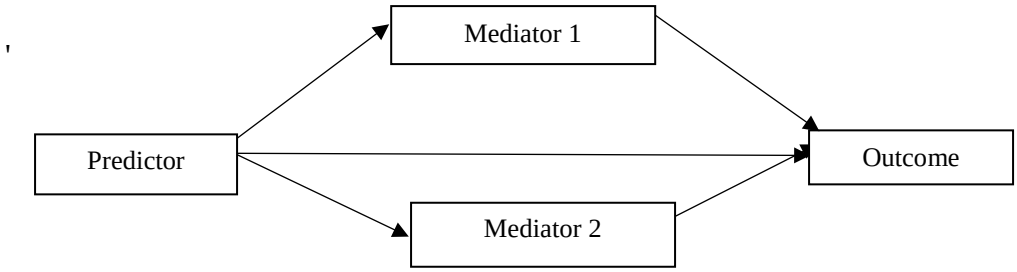

Figure 3.4 Parallel mediator model.
Note. Model adapted from Hayes (2013)

To test Hypothesis Four, separate parallel mediator models were specified for each predictor, with pain-related disability as the outcome, and the two pain stress-processing factors, pain self-efficacy and catastrophising as mediators. PROCESS (Hayes, 2013) calculates a number of different effects within mediator models. These include the direct effect (c') which refers to the relationship between the predictor and outcome after controlling for the mediators in the model. Specific indirect effects (for example a_1b_1) are also calculated which refer to the role of a specific mediator in the relationship between the predictor and outcome.

Effect sizes in mediation analyses provide an indication of how much of the effect of a predictor on an outcome is occurring through the mediator. Cohen (1988) defined an effect size as the extent to which the phenomenon is present in the population. Other authors have defined as effect size as a number that conveys the practical significance of an effect (Preacher & Kelley,

2011). Varying recommendations have been made regarding the interpretation of the magnitude of effects sizes, but one commonly adopted approach is that of Cohen (1988), in which effect sizes of .01, .09, and .25 are defined as small, medium, and large respectively. Therefore, these definitions were adopted in this study.

Both Hayes (2013) and Preacher and Kelley (2011) point out that a number of different approaches exist to calculate and express effect sizes with ongoing debate in the literature as to the most appropriate method for reporting mediation analyses. Common approaches include the unstandardised and completely standardised indirect effects. The unstandardised indirect effect reflects the decrease in the effect of the predictor on the outcome when the mediator is added to the model (Preacher & Kelley, 2011). It is independent of sample size and is calculated and interpreted according to the original scales used to measure the variables in the model. The completely standardised effect is also independent of sample size but removes the scaling of both the predictor and outcome. That is, the resulting effect size no longer reflects the underlying scales of the variables in the model and is therefore comparable across different metrics (Preacher & Kelley, 2011). Hayes (2013) argues that it is preferable to report indirect effects in unstandardised form because standardised effects are scaled in terms of the variability of the sample and are therefore not comparable across different studies. Therefore, only the unstandardised effects, with their associated confidence intervals, are reported in the current research.

3.5.4.7.2 Moderation effects.

Addressing the fifth hypothesis, the potential moderating effects of pain self-efficacy and social engagement on the relationships between risk factors and pain-related disability were also tested using PROCESS (Hayes, 2013). PROCESS calculates two way interactions in moderation models. Specifically, PROCESS Model 1 (moderation model) was used to test moderation effects.

Bootstrapping is also used within PROCESS to calculate bias-corrected confidence intervals for moderation effects. As described above in Section 3.7.4.6.1, 10,000 bootstrap iterations were used to calculate moderation effects, using 95% bias-corrected confidence intervals.

Significant interactions were probed, post hoc, to identify the level of the moderator that interacts with risk factor to affect its relationship with the outcome. PROCESS conducts post-hoc probing of significant interactions in two ways. Firstly, simple slopes were plotted at three levels of the moderator, high, medium and low (Aitken & West, 1991). Secondly, a regions of significance analysis was conducted using the Johnson-Neyman technique (Johnson & Neyman, 1936). This technique calculates the specific values of the moderator at which the relationship between the predictor and outcome becomes statistically significant.

3.6 Threats to validity

Various potential validity threats were considered. These included internal validity threats such as effects of missing data and biased effects estimates. Threats to external validity were also considered, such as common method bias and adequacy of sample size. Methods for dealing with these potential threats is described below.

3.6.1 Effects of missing data.

As the data were pre-collected, there was no ability to implement measures to reduce the risk of missing data. Where entire scales or several scales for a single participant were missing this case was deleted as the number of cases affected was small and there were concerns about the reliability of the remaining data for that participant (as described in Section 3.7.4.1). Relatively high rates of missing data were observed for the partner responses to pain subscales of the MPI (Kerns et al., 1995) and these data were replaced using the EM algorithm. As all the analyses were repeated in two datasets, one with partner responses imputed and one with these cases deleted and

only minor differences were noted between the two sets of results, missing data did not appear to pose a significant threat to the validity of this study.

3.6.2 Biased effects estimates.

In order to improve reliability of estimates of indirect effects, bootstrapped confidence intervals were calculated for all indirect effects.

3.6.3 Common method bias.

Common method bias (CMB) can bias results of research that is conducted using a single method of assessment, for example a paper and pencil questionnaire. Common method bias can introduce systematic response bias that may either increase or decrease response values. It has been suggested that where significant CMB exists, a large proportion of variance can be explained by a single factor (Podsakoff, MacKenzie, Lee & Podsakoff, 2003). Several methods have been proposed in the literature to assess and correct for CMB, including the correlational marker technique, the confirmatory factor analysis marker technique and the unmeasured latent method construct technique all within structural equations modelling (Podsakoff et al., 2003).

The current study used exclusively paper and pencil questionnaires and therefore CBM was a potential issue. However, considerable debate exists in the literature regarding the extent to which CMB actually exists and, if it does, how much it biases estimates (Spector, 2006). Further, Richardson, Simmering and Sturman (2009) highlight that debate also exists regarding the adequacy of the above techniques for both identifying and correcting CMB. These authors used simulated data sets to test all three correction techniques and concluded that no post hoc technique can be recommended to correct for CMB's potential effects, nor is any technique recommended as a way to detect CMB bias. Therefore, the possibility that CMB may have biased estimates in the

current research is acknowledged but due to existing controversy about appropriate assessment and correction techniques, it was not assessed. Results are presented in Chapter Four.

Chapter Four – Study One Results

4.1 Overview

Study One examined the utility of the adapted risk-resistance model to explore factors associated with physical adjustment to chronic pain in a pain clinic sample. Risk and resistance factors were restricted to those available in the pain clinic database. Chapter Four presents Study One results. Descriptive statistics are reported first, followed by results of each of the hypotheses.

4.2 Descriptive Statistics

Scores for pain-related disability, anxiety, depression, catastrophising and solicitous and punishing partner responses to pain all ranged from zero to the maximum scores possible. Scores for pain self-efficacy and social engagement ranged from zero to just below the maximum scores. Only one significant gender difference was noted; men reported more punishing responses to pain from their partners compared to women ($t = 2.33$, $p < .05$). Descriptive statistics of study measures according to gender can be seen in Table 4.1.

Table 4.1

Means, Standard Deviations of Study Variables Comparing Males to Females

	% missing	Mean (SD)		*t* (p)
		Male (N=145)	Female (N=207)	
Age	.00	52.02 (12.87)	51.33 (13.93)	.47 (.64)
Pain-Related Disability	2.00	14.72 (5.29)	14.34 (5.62)	.63 (.53)
Pain Severity	2.82	4.47 (.97)	4.48 (.96)	-.14 (.89)
Anxiety	10.20	5.13 (5.34)	5.78 (4.94)	-1.18 (.24)
Depression	7 .40	9.10 (6.69)	8.91 (6.05)	.27 (.77)
Catastrophising	.09	3.13 (1.10)	3.16 (1.11)	-.25 (.81)
Solicitous partner responses	24.70**	3.35 (1.53)	3.42 (1.49)	-.46 (.65)
Punishing partner responses	24.70**	2.24 (1.46)	1.87 (1.50)	2.32 (.02)
Pain Self-Efficacy	2.30	20.30 (11.86)	22.62 (11.34)	-1.52 (.13)
Social Engagement	1.40**	1.73 (1.05)	1.83 (1.23)	-.78 (.44)

Note 1. ***** Missing data is reported as the proportion of values missing across all items for that scale.
Note 2. ****** Indicates a statistically significant value of Little's Missing Completely at Random test suggesting data were not missing at random.

In order to place the current results in the context of the literature, the current sample was compared to normative Australian pain clinic data (Nicholas, Asghari & Blyth, 2008). Many measures used in the current study were the same as those in the normative dataset, therefore direct comparisons were possible. Results can be seen in Table 4.2.

Table 4.2

Comparison of Study One Measures to Normative Pain Clinic Data

	M (SD) normative data (n=6124)	*M* (SD) Study 1 (n=352)	*t* (p)
Age	48.4 (16.2)	52.02 (12.87)	4.47 (.00)
Pain-Related Disability	12.3 (5.7)	14.53 (5.46)	7.52 (.00)
Pain Severity	4.2 (1.1)	4.48 (.97)	5.38 (.00)
Catastrophising	2.7 (1.2)	3.13 (1.11)	7.63 (.00)
Pain Self-Efficacy	25.5 (13.8)	21.46 (11.60)	-5.11 (.00)

Note. Normative data obtained from Nicholas, Asghari & Blyth (2008)

4.3 Covariates of Pain-related Disability

Age and gender were explored as potential covariates in the regression analyses as these were the only demographic variables reliably recorded in the pain clinic database. The relationships of gender and age to pain-related disability were examined using independent sample *t*-tests and Pearson's product moment correlations respectively. A *t*-test examining differences in pain-related disability by gender can be seen in Table 4.1 and revealed no significant group differences t (350) = .63, p = .53. Age was not associated with pain-related disability (r [352] = -.06). Therefore, neither age nor gender were entered into the regression analysis as covariates.

4.4 Study Hypotheses

4.4.1 Hypothesis one – relationships between risk factors and pain-related disability.

The first hypothesis that higher levels of risk factors would be associated with greater pain-related disability was supported. Moderate strength positive correlations were observed between all risk factors and pain-related disability except for solicitous and punishing partner responses to pain which were also positively associated with disability but at weak strength. Results can be seen in Table 4.3.

4.4.2 Hypothesis two – relationships between resistance factors and pain-related disability.

The second hypothesis that higher levels of resistance factors would be associated with less pain-related disability was also supported. Pain self-efficacy and social engagement were both negatively correlated with pain-related disability at strong and moderate strength, respectively. Results can be seen in Table 4.3.

Table 4.3

Correlations Between Variables

	1	2	3	4	5	6	7	8	9
1. Disability	1								
2. Pain Intensity	.47**	1							
3. Depression	.43**	.31**	1						
4. Anxiety	.41**	.33**	.66**	1					
5. Catastrophising	.36**	.44**	.52**	.39**	1				
6. Solicitous responses	.24**	.22**	-.01	.12**	.14**	1			
7. Punishing responses	.15**	.06	.24**	.20**	.14**	-.30**	1		
8. Self-Efficacy	-.61**	-.37**	-.52**	-.39**	-.42**	-.12*	-.13*	1	
9. Social Engagement	-.32**	-.18**	-.43**	-.26**	-.23	-.02	.07	.48**	1

Note. ** Correlation is significant at the .01 level, * Correlation is significant at the .05 level

4.4.3 Hypothesis three – risk-resistance model compared to a risk only model.

The third hypothesis proposed that resistance factors would account for additional variance in pain-related disability, over and above that explained by the risk factors. This hypothesis was tested using hierarchical multiple regression. Risk factors were entered in Step One (Model One). Resistance factors were entered in Step Two (Model Two). As shown in Table 4.4, the combined risk and resistance factors accounted for a significant 49% of the variance in pain-related disability (F (8, 343) = 40.36, $p < .001$). The addition of the resistance factors accounted for a significant 13% of the variance in pain-related disability over and above that accounted for by the risk factors, $\Delta R^2 = .13$, ΔF (2, 341) = 41.27, $p < .001$. Perceived pain severity, solicitous and punishing partner responses to pain and pain self-efficacy all made significant unique contributions to pain-related disability in the final equation. The direction of the association between catastrophising and pain-related disability in the final risk-resistance model was in the opposite direction to the observed bivariate correlation, although the magnitude of this standardised regression weight was very small.

Table 4.4

Hierarchical Multiple Regression of Pain-related Disability on Risk and Resistance Factors

	β	R_2	△ R_2	F	△ F
Step 1		.36	.36	32.48***	32.48***
Pain severity	.23***				
Anxiety	.10				
Depression	.06				
Catastrophising	-.03				
Solicitous Responses	.17***				
Punishing Responses	.11*				
Step 2		.49	.13	40.36***	41.27***
Pain self-efficacy	-.42***				
Social engagement	-.03				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The regression weights shown in this table are those from Model 2, the risk-resistance model.

Variance inflation factors (VIFs) were carefully checked in order to explore potential effects of multicollinearity. The highest VIF was that of depression (2.36), possibly due to its close links with anxiety. Although this VIF was well below Tabachnik & Fidells' (2013) recommended threshold of 10, the effect of averaging the two DASS subscale scores of anxiety and depression to produce a combined score was explored. This predictor was labelled in the subsequent model as negative affect (NA).

The analysis was repeated, entering risk factors in Step One and resistance factors in Step Two. Results can be seen in Table 4.5. In this second model, the combined risk and resistance factors also accounted for a significant 49% of the variance in pain-related disability (F (7, 342) =

47.54, $p < .001$). The addition of the resistance factors accounted for a significant 14% of the variance in pain-related disability over and above that accounted for by the risk factors, $\Delta R^2 = .14$, ΔF (2, 342) = 47.58, $p < .001$. Perceived pain severity, NA, solicitous and punishing partner responses to pain and pain self-efficacy all made significant unique contributions to pain-related disability in this second model. However, the direction of the association between catastrophising and pain-related disability in this model remained small and negative.

Table 4.5

Hierarchical Multiple Regression of Pain-related Disability on Risk and Resistance Factors, using Negative Affect as a Risk Factor

	β	R_2	ΔR_2	F	ΔF
Step 1		.35	.35	37.40***	37.40***
Pain severity	.24***				
Negative Affect	.13**				
Catastrophising	-.06				
Solicitous Responses	.17***				
Punishing Responses	.11*				
Step 2		.49	.14	47.54***	47.54***
Pain self-efficacy	-.45***				
Social engagement	-.01				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The regression weights shown in this table are those from Model 2, the risk-resistance model.

Further model simplification was undertaken to investigate additional potential collinearity effects in the model. Firstly, social engagement was removed because it did not contribute unique variance to pain-related disability. This did not change the negative regression weight of catastrophising. The remaining predictors were removed one by one and replaced iteratively in the models. The regression weight for catastrophising was only positive in models where either pain

severity or pain self-efficacy were removed. It remained a non-significant predictor of disability in all models.

4.4.4 Hypothesis four – mediation models.

The fourth hypothesis proposed that stress-processing factors, catastrophising and pain self-efficacy, would mediate the relationships between condition parameters, intrapersonal and social-ecological factors that were associated with pain-related disability at moderate or greater strength. This is consistent with guidelines provided by Rose and colleagues (2004) who suggest that mediation effects are generally only tested once a predictor–outcome relationship is established. Therefore, only the relationships between pain severity, anxiety, depression and social engagement were tested in mediator models.

As outlined in Section 3.7.4.7.1 in Chapter Three, mediation effects were assessed first in single mediation models, then where significant effects were identified, in parallel mediator models. The strength of the reported effect sizes for the indirect effects in in parallel mediator models is adjusted for the mediating role of the other variable (Hayes, 2013). Refer to Figure 3.4 in Chapter Three. Mediation effects were assessed with ordinary least squares regression using the PROCESS macro for SPSS (Hayes, 2013), using 10,000 bootstrap iterations and 95% confidence limits.

4.4.4.1 Hypothesis four – catastrophising as a sole mediator.

In single mediator models, pain severity, anxiety and depression all influenced pain-related disability indirectly through a statistically significant positive association with catastrophising. Social engagement influenced pain-related disability indirectly through a negative association with catastrophising. Indirect effects ranged from small (anxiety, depression) to moderate (pain severity, social engagement) in magnitude. The direct paths between predictors and pain-related disability

remained statistically significant in the mediated models, indicating that the effects of risk predictors on pain-related disability were expressed directly as well as indirectly, via catastrophising. Results can be seen in Table 4.5.

4.4.4.2 Hypothesis four – pain self-efficacy as a sole mediator.

In single mediator models, pain severity, anxiety and depression all influenced pain-related disability indirectly through a statistically significant negative association with pain self-efficacy. Social engagement also influenced pain-related disability indirectly through a statistically significant positive association with pain self-efficacy. Indirect effects ranged from small (anxiety, depression) to large (pain severity, social engagement) in magnitude.

The direct paths between the predictors and disability remained statistically significant with pain self-efficacy in the model indicating that pain severity, anxiety and depression all influenced pain-related disability directly as well as indirectly. When pain self-efficacy mediated the relationship between social engagement and disability, the direct relationship between social engagement and pain-related disability was not statistically significant. Thus, influence of social engagement on pain-related disability was expressed entirely via a positive association with pain self-efficacy. Unstandardised regression co-efficients, associated confidence intervals and unstandardised effect sizes can be seen below in Table 4.6.

Table 4.6

Summary of Simple Mediation Analyses Predicting Disability (10,000 Bootstrap Samples)

Predictor (X)	Coefficient (SE) Predictor to M (Path a)	Coefficient (SE) M to Disability (Path b)	Indirect Effect of X on Disability (CIs) (Path *ab*)	Coefficient (SE) X to Disability (Path *c'*)	Model summary R^2
Mediator: Catastrophising					
Pain Severity	5.33 (.06)***	.94 (.26)***	.50 (.16) [.22; .84]#	2.31 (.31)***	.25, *F*(2, 347) = 59.20***
Anxiety	.09 (.01)***	1.31 (.26)***	.12 (.03) [.07; .19]#	.31 (.06)***	.19, *F*(2, 347) = 41.28***
Depression	.09 (.01)***	.94 (.28)***	.09 (.03) [.03; .15]#	.29 (.05)***	.21, *F*(2, 347) = 46.41***
Social Engagement	-.23 (.05)***	1.53 (.25)***	-.35 (.10) [-.58; -.18]#	-1.24 (.24)***	.19, *F*(2, 347) = 41.66***
Mediator: Pain Self-Efficacy					
Pain Severity	-5.65 (.75)***	-.20 (.02)***	1.15 (.18) [.83; 1.52]#	1.65 (.25)***	.46, *F*(2, 347) = 101.89***
Anxiety	-1.10 (.15)***	-.22 (.02)***	.24 (.04) [.18; .32]#	.19 (.05)***	.41, *F*(2, 347) = 120.88***
Depression	-1.16 (.10)***	-.22 (.02)***	.25 (.03) [.20; .31]#	.13 (.04)**	.40, *F*(2, 347) = 117.48***
Social Engagement	6.18 (.58)***	-.24 (.02)***	-1.49 (.17) [-1.84; -1.18]#	-.10 (.24)	.39, *F*(2, 347) = 110.43***

Note. ***p<.001, **p<.01, *p<.05, # = statistically significant indirect effect

4.4.4.3 Hypothesis four – parallel mediation models – risk factors.

When tested in parallel mediation models, the positive associations between pain severity, anxiety and depression and pain-related disability were mediated by statistically significant indirect effects via pain self-efficacy but not via catastrophising. Thus, higher levels of pain severity, anxiety and depression were associated with lower perceived efficacy to manage pain, which in turn was negatively associated with pain-related disability. The magnitude of the indirect effects via pain self-efficacy were moderate (anxiety, depression) to large (pain severity). The direct paths between all risk predictors and pain-related disability remained statistically significant with both mediators in the model. This indicates that pain severity, anxiety and depression were all directly associated with pain-related disability, as well as indirectly. Pairwise comparison tests were significant indicating a statistically significant difference between the two indirect effects. Unstandardised regression coefficients, significance levels, standard errors, unstandardised effect sizes and associated confidence limits and results of pairwise comparison tests can be seen in Figures 4.1 to 4.3.

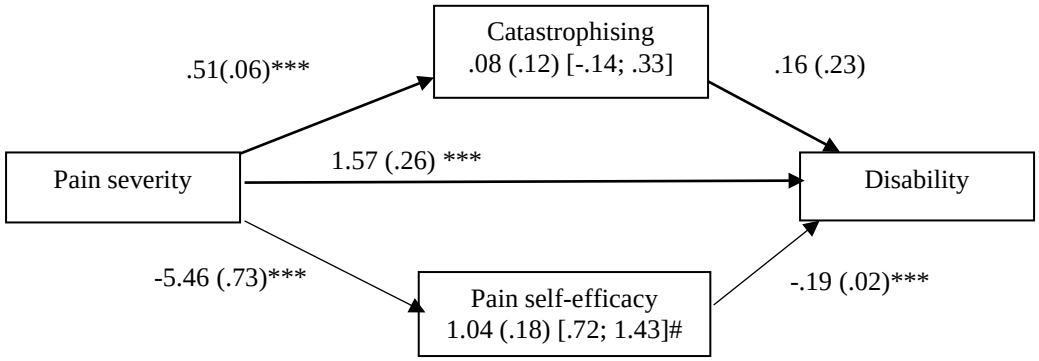

Figure 4.1 Pain self-efficacy but not catastrophising mediates the relationship between pain severity and pain-related disability.

Note 1. Total effect of pain severity on disability = 2.70 (.27) ***, model summary: R_2: .46, $F(3, 346) = 96.85$***

Note 2. Total effect = unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the relevant boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Note 3. ***p<.001, **p<.01, *p<.05, # = statistically significant indirect effect.

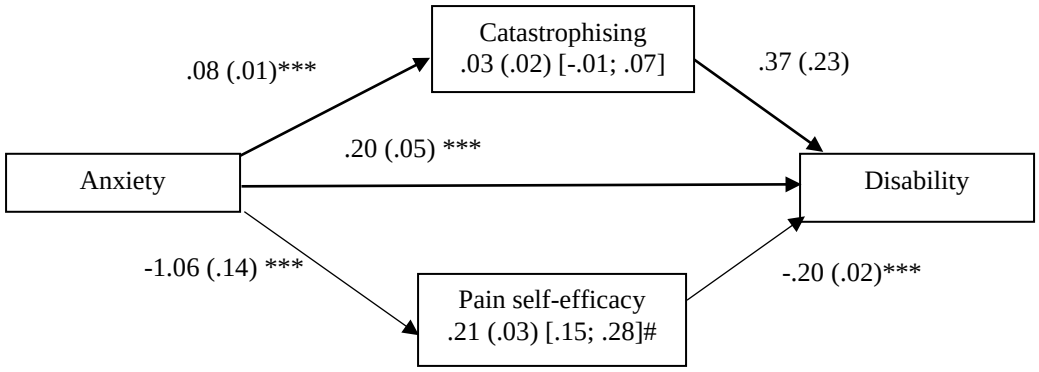

Figure 4.2 Pain self-efficacy but not catastrophising mediates the relationship between anxiety and pain-related disability.

Note 1. Total effect of anxiety on disability = .44 (.05) ***, model summary: R_2: .41, $F(3, 346) = 81.64$***

Note 2. Total effect = unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the relevant boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Note 3. ***p<.001, **p<.01, *p<.05, # = statistically significant indirect effect.

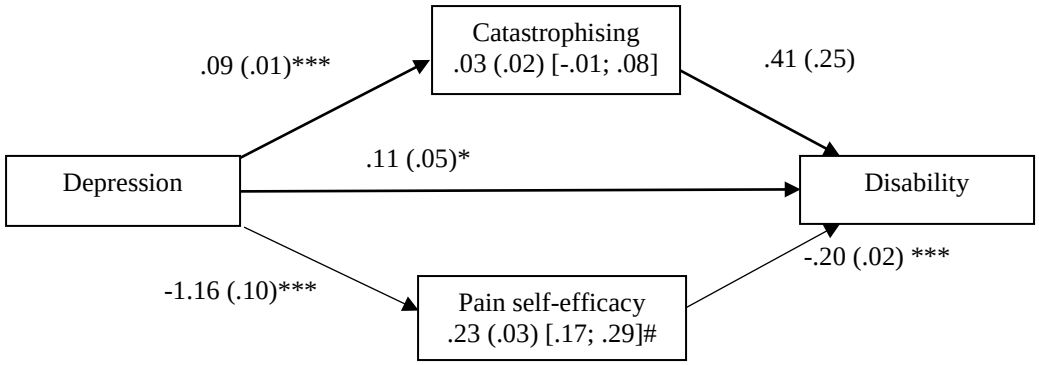

Figure 4.3 *Pain self-efficacy but not catastrophising mediates the relationship between depression and pain-related disability.*

Note 1. Total effect of depression on disability = .37 (.04) ***, model summary: R_2: .41, $F(3, 346) = 79.09$***

Note 2. Total effect = unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the relevant boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Note 3. ***p<.001, **p<.01, *p<.05, # = statistically significant indirect effect.

4.4.4.4 Hypothesis four – parallel mediation models – resistance factors.

The negative association between social engagement and pain-related disability was simultaneously mediated by statistically significant indirect effects via both pain self-efficacy and catastrophising. Higher levels of social engagement were associated with higher pain self-efficacy, which in turn was negatively associated with pain-related disability. At the same time, higher levels of social engagement were associated with lower levels of catastrophising, which in turn was positively associated with pain-related disability. Pairwise comparison of indirect effects in this model indicated a significant difference. As the effect size for the indirect effect of social engagement via pain self-efficacy was larger than that via catastrophising and both were in a negative direction, this indicates social engagement decreases pain-related disability more by increasing pain self-efficacy than by decreasing catastrophising. See Figure 4.5.

Hypothesis Four was therefore only partly supported as only the relationships between pain severity, anxiety and depression with pain-related disability were mediated by only one of the pain appraisal processes in the model. The direct relationship between this predictor and pain-related disability became non-statistically significant when pain self-efficacy and catastrophising were in the model. This indicates that in this sample, with both mediators in the model, social engagement was associated with less pain-related disability entirely by its association with pain self-efficacy and to a lesser degree, by its association with catastrophising.

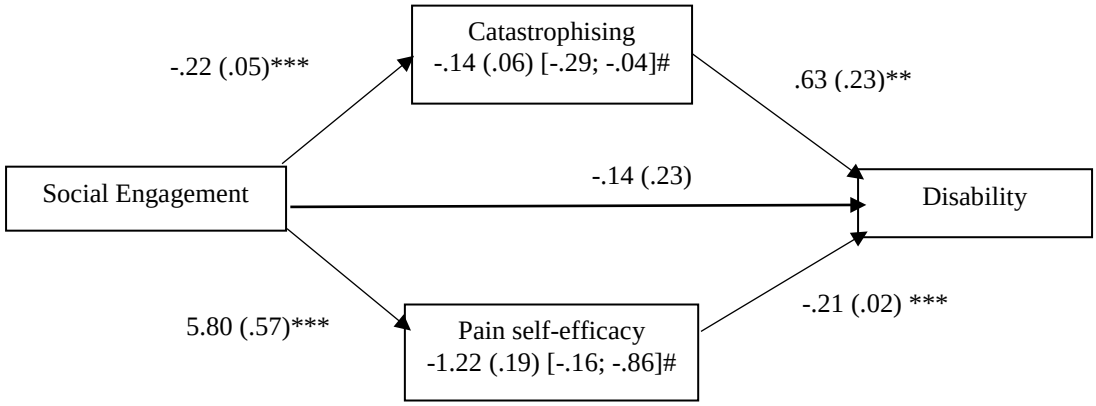

Figure 4.4 Pain self-efficacy and catastrophising mediate the relationship between social engagement and pain-related disability.

Note 1. Total effect of social engagement on disability = - 1.60 (.25) ***, model summary: R_2: .40, $F(3, 346)$ = 76.53***, comparison of indirect effects = 1.11 (.23) [.68; 1.57]

Note 2. Total effect = unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the relevant boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Note 3. ***p<.001, **p<.01, *p<.05, # = statistically significant indirect effect.

4.4.5 Hypothesis five – moderation models.

Hypothesis Five proposed that resistance factors, pain self-efficacy and social engagement, would moderate the relationships between the risk predictors and pain-related disability. Consistent with Rose and colleagues (2004) who suggested that moderation effects are generally only tested

once a predictor–outcome relationship is established, relationships between risk factors and disability were only tested for moderation when predictor variables were correlated with pain-related disability at moderate strength or greater ($r \geq .30$).

Moderation effects were calculated with PROCESS (Hayes, 2013) using 10,000 bootstrapped iterations and 95% bias-corrected confidence intervals. Post-hoc probing of significant interactions was planned. Hypothesis 5 was not supported as neither pain self-efficacy nor social engagement moderated the relationships between pain severity, catastrophising, anxiety and depression with pain-related disability. All the confidence intervals of the interaction terms in the linear regression models crossed zero indicating non-statistically significant interaction terms, thus no moderating effects. See Tables 4.7 and 4.8.

Table 4.7

Regression Models Estimating Pain Self-Efficacy as a Moderator of the Relationships between Risk Predictors and Pain-Related Disability

		Co-efficient	*SE*	*t*	*BC CIs*
Model 1					
R^2 = .44 *MSE = 16.95*					
	Pain Severity	1.30	.44	2.93	-.41; -.11
	Pain Self-Efficacy	-.26	.07	-3.50	.43; 2.17
	Interaction	.01	.02	.90	-.02; .05
Model 2					
R^2 = .41 *MSE* = 17.84					
	Anxiety	.13	.07	1.80	-.01; .27
	Pain Self-Efficacy	-.23	.02	-10.26	-.27; -.19
	Interaction	.01	.00	1.73	.00; .01
Model 3					
R^2 = .39 *MSE* = 18.41					
	Depression	.09	.06	1.46	-.03; .21
	Pain Self-Efficacy	-.22	.03	-8.60	-.27; -.17
	Interaction	.00	.00	1.04	.00; .01
Model 4					
R^2 = .38 *MSE* = 18.71					
	Catastrophising	.73	.38	1.91	-.02; 1.48
	Pain Self-Efficacy	-.20	.04	-4.81	-.29; -.12
	Interaction	.00	.01	-.33	-.03; .02

Note. MSE = Mean standard error; SE = standard error; BC CIs = bias corrected confidence intervals

Table 4.8

Regression Models Estimating Social Engagement as a Moderator of the Relationships between Risk Predictors and Pain-Related Disability

		Co-efficient	*SE*	*t*	*BC CIs*
Model 1					
R^2 = .28 *MSE = 21.82*					
	Pain Severity	2.21	.47	4.68	1.28; 3.14
	Social Engagement	-1.78	1.06	-1.67	-3.87; .32
	Interaction	.14	.23	.62	-.32; .60
Model 2					
R^2 = .22 *MSE* = 23.62					
	Anxiety	.29	.09	3.32	.11; .46
	Social Engagement	-1.37	.33	-4.20	-2.01; -.73
	Interaction	.06	.05	1.36	-.03; .16
Model 3					
R^2 = .21 *MSE* = 24.01					
	Depression	.30	.07	4.18	.16; .44
	Social Engagement	-.84	.37	-2.26	-1.57; -.11
	Interaction	.01	.04	.26	-.06; .08
Model 4					
R^2 = .19 *MSE* = 24.63					
	Catastrophising	1.46	.44	3.29	.59; 2.34
	Social Engagement	-1.23	.65	-1.90	-2.51; -.04
	Interaction	.02	.20	-.11	-.37; .41

Note. MSE = Mean standard error; SE = standard error; BC Cis = bias corrected confidence intervals

4.5 Summary and Implications

The adapted risk-resistance model tested in the current study provided a useful tool through which factors related directly and indirectly to pain-related disability could be explored. This section provides a summary of results and discusses implications of these for Study Three. Full discussion of the results can be found in Chapter Nine.

4.5.1 Results summary.

The first two study hypotheses were largely supported. All risk and resistance predictors, except solicitous and punishing partner responses to pain, demonstrated at least moderate strength negative and positive correlations respectively with pain-related disability. Hypothesis Three proposed that resistance factors would account for additional variance in pain-related disability, over and above that explained by the risk factors. This hypothesis was supported. The risk-resistance model explained 13% more variance in the measure of pain-related disability compared to the risk only model. This demonstrates the utility of including not only risk, but also resistance factors, when investigating factors associated with adjustment to chronic pain.

Hypothesis Four proposed that that catastrophising and pain self-efficacy would mediate the relationships in the model between other variables in the model that were associated with pain-related disability at moderate or greater strength. This hypothesis was largely supported. In single mediator models, both catastrophising and pain self-efficacy mediated the relationships between pain severity, anxiety, depression and social engagement with pain-related disability. However, when these indirect effects were assessed simultaneously in parallel mediator models, only pain self-efficacy, but not catastrophising, mediated the relationships between risk factors and pain-related disability. The relationship between social engagement and disability was mediated by both catastrophising and pain self-efficacy, although the indirect effect via pain self-efficacy was larger.

Thus, pain self-efficacy represented an important mechanism through which the effects of the other predictors on pain-related disability were expressed. The final hypotheses regarding the moderating effects of pain self-efficacy and social engagement on risk-outcome relationships in the model were not supported.

4.5.2 Measurement issues identified in Study One.

Study One served an exploratory purpose as it provided the opportunity to examine the utility of the adapted risk-resistance model for exploring factors associated with physical adjustment to chronic pain in a pain clinic sample. A number of issues were identified that had implications for Study Three. These are detailed below.

4.5.2.1 Measurement of anxiety and depression.

Pearson correlations between DASS (Lovibond & Lovibond, 1995) subscales that measured anxiety and depression were strong, indicating a degree of shared variance. Although this was expected because of the relatedness of these constructs, in order to reduce potential problems with multi-collinearity in Study Three a decision was made to assess NA as an intrapersonal risk factor in place of anxiety and depression. Evidence for NA as a predictor of pain-related physical function and QOL is presented below.

4.5.2.2 Negative affect as a predictor of pain-related function and quality of life.

Negative affect has been suggested to be comprised of three core negative emotional experiences; anxiety, depression and anger (Watson & Clark, 1984; Watson & Pennebaker, 1989). Even NA that occurs within a non-pathological range has been suggested to influence the trajectory of pain conditions (Nicholson, Houle, Rhudy & Norton, 2007). Negative and positive affect (PA) are generally seen as theoretically independent constructs, however statistically their relationship

can vary between being fully independent to being at the opposite ends of the same spectrum (Lumley et al; 2011).

Substantially less research has examined links between NA and chronic pain outcomes compared to research investigating anxiety and depression as predictors of pain-related function. However, in the existing research, a significant positive relationship has been demonstrated. For example, in a prospective evaluation of the relationship between NA, PA and chronic pain outcomes following spinal surgery in 141 individuals, higher NA was predictive of higher pain-related disability three months after surgery (Seebach et al., 2012).

Negative affect was also a significant predictor of disability in two studies using a regression-based approach. The first (Karsdorp & Vlaeyen, 2011) was in a sample of 301 participants with chronic pain in the arms, neck, or low back. Together, gender, age, pain duration, catastrophising, NA and pain-avoidance explained 25% of the variance in a measure of pain-related disability. In the second, Agar-Wilson and Jackson (2012) also established NA as a significant predictor of both disability and QOL in a heterogeneous sample of 128 Australian adults with chronic pain. Diagnoses of those in the sample included back pain, fibromyalgia and osteoarthritis. Together, pain intensity, NA, pain self-efficacy, older age and lack of involvement in pain management rehabilitation predicted 48% of variance in a measure of pain-related disability. Negative affect, disability, self-efficacy for pain, self-efficacy for emotion regulation and helplessness coping together explained 42% of the variance in QOL.

Similar to observed links between depression and pain, higher NA is also associated with higher pain severity. For example, a review that examined factors influencing the clinical course of fibromyalgia, found that NA was a key predictor of pain intensity (Staud, 2004). As highlighted in Chapter Two, interactive effects between NA and PA have also been reported in those with chronic pain (Zautra et al., 2001; Zautra et al., 2004) which has been suggested to reflect an

increasingly inversely relationship of these two constructs in stressful situations. In summary, NA appears to be a risk factor for pain-related adjustment outcomes.

4.5.2.3 Social engagement.

Social engagement was used in Study One as a proxy measure for social support, as a measure for social support was not available. Social engagement was moderately negatively correlated with pain-related disability (See Section 4.4.1 Hypothesis One). However, it was unclear whether this measure indicated the degree of support that participants perceived when visiting friends and relatives or reflected their functional capacity, that is, their ability to get out of the house, or both. These issues meant that the specific moderating effects of social support on the relationships between other risk predictors in the model with pain-related disability required further exploration. Despite that social support has been demonstrated to moderate or buffer the effects of risk factors on adjustment outcomes in the general health literature (Cohen & Wills, 1985), very few studies have explored whether social support moderates or buffers the relationship between risk factors and chronic pain adjustment outcomes. Use of a validated measure of emotional and instrumental support was therefore important in Study Three in order to better assess such effects.

4.5.2.4 Additional outcome measure for Study Three.

A major strength of Study Three is that it aimed to explore positive as well as negative pain adjustment processes. It was therefore important to include a measure of positive adjustment, such as QOL. It was intended that improved understanding of factors associated with well-being despite pain may enhance strength based approaches to chronic pain rehabilitation.

4.5.3 Additional predictors for Study Three.

Several other protective factors were identified in Chapter Two that were unable to be included in Study One but may be associated with improved adjustment outcomes or may moderate risk-outcome relationships or both. These factors included pain acceptance (McCracken & Eccleston, 2003, Wright et al, 2011), optimism (Ferreira & Sherman, 2007, Hanssen et al., 2012) and PA (Villanueva, Cornett, Yocum and Castro, 1999). It was planned that these constructs would be included in Study Three.

As an exhaustive review in Chapter Two of every potential protective factor for pain adjustment was not possible. It was thought that resistance factors in addition to those identified in Chapter Two may improve the explanatory capacity of Study Three models and provide new insights into indirect effects associated with pain adjustment processes. As qualitative research has been shown to be useful for determining undiscovered variables for later use in quantitative research (Creswell, 2009) a second qualitative study was planned. Study Two sought a sample of well-adjusted individuals because it was thought that these individuals may offer the best insights into resistance factors associated with positive adjustment outcomes. No research to date appears to have explored resistance factors in a sample of people demonstrating good function despite chronic pain. Chapter Five briefly reviews some of the related qualitative literature and introduces the methodology for Study Two. Chapter Six presents results of Study Two.

Chapter Five – Study Two

5.1 Rationale

Historically, chronic pain adjustment research has focused on identification of risk factors predisposing to poorer adjustment. More recently, it has become evident that positive cognitive, affective and social factors may provide a protective effect against the adverse effects of adjustment risk factors and may promote improved adjustment outcomes (Smith & Zautra, 2008). Improved understanding of protective factors promoting better adjustment outcomes will inform a strengths based approach to treatment (West et al., 2012).

Of the relatively small amount of research that has specifically examined the influence of protective factors on chronic pain adjustment, much of this work has been undertaken either in pain clinic samples (Hyun-Park & Sonty; 2010; Ramırez-Maestre, Esteve & Lopez, 2012; Vowles, McCracken & Eccleston, 2007; Wright et al., 2011) or in community dwelling individuals reporting varying adjustment to pain (Ong, Zautra & Reid, 2010; Woby et al., 2004; Wright et al., 2008). Few, if any, studies have explored potentially protective factors for chronic pain adjustment in those who appear to be coping well with the condition. Therefore, a sample of individuals who were well-adjusted to chronic pain were sought for Study Two, the main aim of which was to investigate novel resistance factors that may promote better adjustment to chronic pain. As qualitative research can be used to determine new variables for quantitative research (Creswell, 2009), Study Two utilised a qualitative approach. In order to consider the results of the current research in the context of the existing literature, a brief review of the related qualitative research is provided below.

5.2 Brief review of qualitative chronic pain adjustment research

One of the earliest qualitative investigations of the chronic pain experience involved interviews with nine women recruited from a hospital pain clinic (Osborn & Smith, 1998). Four themes were identified: the search for an explanation, effects of pain on the self, invalidation and social withdrawal. These results have been replicated in the multitude of qualitative studies that have since been published investigating the subjective experience of chronic pain. Summarising much of this research, two recent metasyntheses reviewed qualitative research examining the impact of low back pain (Bunzli et al., 2013; Snelgrove & Liossi, 2013).

The first of these reviews (Bunzli et al., 2013) included 25 studies, six of which were completed in community samples, with the remainder using pain clinics or pain treatment program samples. Three main themes were identified; the social construction of chronic low back pain, its psychosocial impact and coping with pain. Almost all included studies identified themes of stigmatization and invalidation. Profound negative impacts on sense of self as a result of pain were reported. Biomedical diagnoses were considered important because they provided legitimacy worthy of social support. Reported coping strategies included acceptance, social withdrawal and strategies to gain credibility, such as attributing pain to a physical cause (Bunzli et al., 2013).

The second meta-synthesis (Snelgrove & Liossi, 2013) included 28 studies; all but two recruited participants from pain management clinics or rehabilitation programs. Three main themes were identified; the impact of chronic pain on the self, on relationships and on coping. Negative effects of pain on sense of self were reported together with positive and negative pain related social interactions. Lack of diagnosis and unresponsiveness to treatment appeared to produce feelings of invalidation and distress. Reported coping strategies included rest, medication and cognitive approaches such as distraction and stoicism (Snelgrove & Liossi, 2013).

A small number of qualitative studies have specifically explored protective factors influencing chronic pain adjustment. Sofaer-Bennett, Moore, Lamberty and O'Dwyer (2007) interviewed 63 older adults recruited from pain clinics and found that perseverance despite pain and maintenance of social connections appeared to enhance function. West and colleagues (2012) interviewed four men and six women recruited from the community. Themes included recognition of individual strength, pain acceptance, accepting help and looking for the positives in life. Positive adjustment processes were also examined in 15 artists recruited from a pain management unit (Lynch, Sloane, Sinclair & Bassett, 2013). Artists were chosen because participation in art despite pain was considered to represent a form of resilience. Participants reported that art fuelled a sense of growth and represented a means to connect with others. Both these factors were felt to foster resilient adjustment. Few, if any, studies to date have investigated the chronic pain adjustment process specifically in individuals who appear to be well adapted to the condition.

5.3 Design

Study Two employed a sequential mixed methods design in which qualitative and quantitative data were used to address different aspects of the research aims (Creswell, 2009). The main aim was to investigate factors that promoted the capacity to cope with pain in a group of individuals who appeared to be well-adjusted to life with pain. This population was chosen as it was thought these individuals may offer unique insights into the capacity to cope well with pain. Purposive sampling was therefore used. General practitioners and allied health staff at a medical practice in regional Victoria invited patients whom they considered to be well-adjusted to chronic pain to participate in the research.

Semi-structured interviews were used to facilitate an in-depth understanding of the experience of living with chronic pain and to explore factors associated with adjustment. As a

purposeful sample was sought, quantification of the impact of pain on participants' lives at the time of interview was needed. Therefore, two questionnaires measured participants' levels of anxiety, depression, stress and physical disability.

5.4 Aims

Study Two had three aims. The first was to undertake an in depth analysis of the process of adjustment in people living with chronic pain who were perceived to be managing their pain condition well. A second aim was to explore resistance factors that may facilitate better adjustment or may have contributed to coping well with the demands of the condition. A third aim was to investigate whether risk factors were identified in addition to those reviewed in Chapter Two that may confer additional explanatory capacity to the risk-resistance model to be tested in Study Three or that may offer new insights into indirect effects of predictors on pain adjustment processes.

5.5 Method

5.5.1 Research epistemology.

The epistemological approach adopted for Study Two was pragmatism. Pragmatism sees reality as socially constructed by the beliefs and perceptions of the participants and rejects the idea of a single reality or truth. Pragmatist researchers are simultaneously involved in an interactive process with participants and are contextually and historically influenced (Mertens, 2014). Pragmatism recognizes the complexity of social phenomena by enabling a role for values and interpretive meaning while at the same time accepting explanation as a legitimate goal of social research (Mertens, 2014). In this framework, the researcher is free to study what is of interest, using the most appropriate methods with the aim of bringing about positive change (Mertens, 2014). Using this approach, data was analysed thematically. Participants' comments and insights were

accepted on face value but were also interpreted in light of participants' social and historical context, their values and the deeper meaning that might lie behind their experiences.

5.5.2 Procedure.

5.5.2.1 Ethical approval.

Ethics approval for the research was granted by the Monash University Human Research Ethics Committee. A copy of this approval can be found in Appendix A.

5.5.2.2 Sampling.

Participants were recruited through a multidisciplinary general medical practice that employed general medical practitioners (GPs), nurses, physiotherapists, exercise physiologists, dieticians and podiatrists. Purposive sampling was used because the research aimed to investigate adjustment processes in individuals who appeared to be coping well. Inclusion criteria included a diagnosis of non-malignant chronic pain and age over 18. Following ethics approval, the Clinical Director of the practice completed a clinic database search, using the terms 'chronic pain' or 'fibromyalgia' which identified 90 patients.

Ninety patients with either one or both of these diagnoses were identified. GPs and allied health staff were then sent a list of these patients and asked to identify those whom they considered to be well adapted to their pain condition. 'Well adapted' individuals were operationalised to staff as those people who were functioning well physically, not experiencing high levels of anxiety and depression and who had never required referral for specialist pain medicine intervention. Letters of invitation (Appendix E) to participate in the research were then posted by the clinic to 50 of these selected individuals. The letter invited potential participants to contact the student researcher by telephone, email or post. Upon receipt of contact, the student researcher telephoned the person, explained the study, and obtained verbal consent to interview at a mutually convenient time.

Written consent was obtained at the time of interview. All interviews were conducted at the health clinic and were audio recorded. Interviews lasted between 45 and 70 minutes. Interviews were transcribed verbatim and analysis was conducted iteratively, where the content of one interview was allowed to inform the next.

Because the aim of the research was to explore factors associated with an ability to cope with chronic pain, participants were further screened at the time of the interview for pain-related disability and psychological distress. The measures used for this screening are outlined below. Of the ten individuals who were interviewed, only data from who reported a 'well-adjusted' profile were analysed. The definition of 'well-adjusted' according to the score profiles of participants is detailed below.

5.5.2.3 Participants.

Ten individuals were screened for participation in the research. Of these, six individuals (one male, five females) aged between 33 and 65 years old (M_{age} = 47 years) met eligibility criteria and participated in the study. Recruitment ceased after six participants as small sample sizes can be considered adequate for smaller sized thematic analysis projects (Braun & Clarke, 2013) and because meta-themes have been reported to be present after six interviews (Guest, Bunce & Johnson, 2006).

Four participants were married or living with a partner. Two participants had a bachelor degree, two had completed a diploma level qualification and two had completed education up to Year 11. Only two participants were working, either part or full time. The remainder were either unemployed, retired or were full time parents. At the time of the interview, none of the participants' pain conditions were compensable although one participant had previously had a claim under the Victorian WorkSafe Authority. All six participants had been diagnosed with fibromyalgia (FM).

Three participants reported additional diagnoses of low back pain (LBP) and osteoarthritis (OA). Duration of the participants' pain ranged between 1.5 and 10 years. Pain medications included paracetamol, low dose anti-depressants, anti-inflammatories, codeine and opioids. All participants lived in regional Victoria and were Australian born with English as a first language. Participant demographics can be seen in Table 5.1.

Table 5.1

Participant Demographic Details

Demographic		Number or mean (SD)
Gender		
	Male	1
	Female	5
Age		
	Mean (SD)	47.00 years (10.56)
	Range	33 – 65 years
Education level		
	Year 11	2
	Diploma	2
	Bachelor Degree	2
Marital status		
	Married / defacto	4
	Single	2
Employment status		
	Unemployed	2
	Working part-time	1
	Working full time	1
	Parent	1
	Retired	1
Pain duration		
	Mean (SD)	5.58 years (3.01)
	Range	(1.50 – 10 years)
Diagnoses		
	Fibromyalgia	3
	Low back pain and fibromyalgia	2
	Osteoarthritis and fibromyalgia	1

5.5.2.4 Measures.

Participants' levels of pain-related disability and psychological distress were quantified at the time of the interview with two validated measures. Psychometrics of these measures are outlined below. Demographic and diagnostic information was also obtained from participants at the time of the interview. Copies of study questionnaires can be found in Appendix B as they are the same as the measures used for these constructs in Study One.

To quantify pain-related disability, participants completed the Roland Morris Disability Questionnaire (RMDQ; Roland & Morris, 1983). The original RMDQ was developed by Roland and Morris (1983) for assessing the functional impact of back pain. The generic version of the

questionnaire used in this study asks respondents to relate the items to their pain, regardless of its site. This modified RMDQ consists of 24 items, which are answered by checking if the item applies as true on that day. Example items include ‘I stay at home most of the time because of my pain’ and ‘Because of my pain, I lie down to rest more often’. The questionnaire is scored by summing the number of items checked as true. Higher scores indicate greater disability. The RMDQ is reported to have acceptable concurrent validity with other measures of pain-related function, has high reported levels of test-retest reliability and has been demonstrated to be sensitive to change (Johansson & Lindberg, 1998; Roland & Morris, 1983). Cronbach’s alpha is reported as 0.92 (Roland & Morris, 1983).

To quantify symptoms of anxiety, depression and stress, the shortened 21 item version of the original 42 item Depression, Anxiety and Stress Scale (DASS 21; Lovibond & Lovibond, 1995b) was used. The DASS 21 item divides the 21 items evenly into three subscales assessing anxiety, depression and stress symptoms. Respondents are asked to indicate the frequency with which they have experienced that symptom in the past week. Items are scored between zero (*never*) and three (*almost always*). The subscales are scored by summing the item responses; higher scores reflect higher levels of anxiety, stress or depression. The DASS 21 has good reported reliability, with Cronbach's alphas reported as .94 for the depression subscale, .87 for the anxiety subscale and .91 for the stress subscale (Lovibond & Lovibond, 1995a). Acceptable convergent, concurrent and discriminant validity of the scale has been demonstrated (Antony et al., 1998; Brown et al., 1997). Clinical criteria for defining normal, moderately elevated, severely elevated and extremely severely elevated levels of anxiety and depression are provided by the scale developers in the DASS User Manual (Lovibond & Lovibond, 1995b) and can be seen below in Table 5.2.

Table 5.2

Score Profiles for the Depression, Anxiety and Stress Scale (Lovibond & Lovibond, 1995b)

	Depression	Anxiety
Normal	0 - 9	0 - 7
Mild elevation	10 - 13	8 - 9
Moderate elevation	14 - 20	10 - 14
Severe elevation	21 - 27	15 - 19
Extremely severe elevation	28+	20+

In line with the DASS 21 suggested clinical thresholds for psychopathology (Lovibond & Lovibond, 1995b) and the median disability score reported by the authors of the RMDQ (Roland & Morris, 1983), participants were defined as ‘well adjusted’ if they did not scores any DASS 21 subscales as ‘severe elevation’ or above and did not endorse more than half of the disability items on the RMDQ. Only data from these “well adjusted” participants were included in the analyses. Participants with scores on the DASS in the severely or extremely severely elevated range in any domain, or above 18 items on the RMDQ were referred back to their GP for review with a letter of explanation for the referral. Written consent for this referral was obtained at the time from participants. Screening results reported by excluded participants can be found in Table 6.2

5.5.2.5 Semi structured interview.

Semi-structured interviews were used to explore participants’ experiences of living with chronic pain. The interview schedule was based on the research aims and literature review and was developed by the researcher in consultation with three academic staff in the Faculty of Education at Monash University all of whom had extensive experience in qualitative research. In total the interview schedule contained nine questions that addressed the following areas of interest; the impact of chronic pain, coping with pain, the process of adjusting to living with pain and factors

that enhanced resilience (See Appendix E). Examples of questions include: ‘Tell me, in your own words, what it is like to live with chronic pain?’ and ‘Can you describe the process you went through in order to adapt to the living with pain? ’ Questions were deliberately open ended and the student researcher adopted a conversational style aimed at creating a two way dialogue to facilitate thematic exploration.

5.5.3 Data analysis.

5.5.3.1 Quantitative analyses.

Descriptive statistics of questionnaire data were analysed with the Statistical Package for Social Sciences Version 20.0 (SPSS., IBM Corp).

5.5.3.2 Qualitative analyses.

Thematic analysis was used to analyse the interview data (Braun & Clark, 2006; Guest, MacQueen & Namey, 2012). Thematic analysis is a qualitative method that can be applied across a range of theoretical and epistemological approaches to provide a complex analysis of the data (Braun & Clark, 2006). The analysis process adhered to the six stage process outlined by Braun and Clarke (2006). Interviews were initially recorded and listened to again to allow familiarisation. Half of the interviews were professionally transcribed to facilitate analysis of the data prior to the next interview. When possible, initial coding was commenced prior to the next interview. An inductive approach was used to identify themes (Patton, 1990). That is, the data were analysed without trying to fit them into a pre-existing coding frame. Initially data were coded descriptively by content and were then categorised into possible themes. Themes were identified when they were described by three or more participants or when they captured an aspect of experience considered by the researcher to be important to the overall research question.

5.5.4 Validity checks.

A copy of each completed transcript was sent to each participant to again check consent to use the entire content of the interview and to ensure that each participant was satisfied the transcript accurately reflected the interview. This strategy is consistent with suggested methods for ensuring validity of results of qualitative research (Morse, Barrett, Mayan, Olson & Spiers, 2008). Themes were cross-checked with data by the main university supervisor. It is acknowledged that this analysis may not represent the only possible account of the data, however these checks were performed in an attempt to ensure validity of the reported results.

5.5.5 Organisation of themes.

Consistent with Braun and Clark's (2006) methodology, themes were first identified and then categorised into 'meta-themes'. Finally, conceptual mapping of the themes was undertaken. Consideration was given to the broader meanings in the data and was referenced within the context of the qualitative chronic pain literature summarised above. Results are presented in Chapter Six.

Chapter Six – Study Two Results

6.1 Overview

This chapter presents results of Study Two. Participants' adjustment profiles are presented as well as positive and negative factors that participants described were related to their ability to live with chronic pain. Implications for Study Three are presented at the end of this chapter. Study Two was published in 2016 in the Journal of Advances in Mental Health. A copy of the manuscript is included in Appendix H. All participant names are pseudonyms.

6.1.1 Participants' adjustment profiles.

Participants' adjustment profiles varied. Most commonly, participants reported mild to moderate elevations in at least one subscale of the Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995a), and endorsed half or less of the disability items on the Roland Morris Disability Questionnaire (RMDQ, Roland & Morris, 1983). Most participants reported disability scores that were well below the threshold set for inclusion of 12. Only one participant, Michael, 65 years, reported anxiety, depression and stress symptoms within the 'normal' range as well as a low disability score. The majority of other participants reported moderately elevation of at least one DASS subscale. All participants reported regular use of pain medications but only Michael reported regular use of the stronger opioid-based analgesia. Pseudonyms for participants, DASS scores and medication use can be seen in Table 6.1.

Table 6.1

Medication, Measures of Depression, Anxiety, Stress and Disability – included participants

Pseudonym	Age	Medication	Depression	Anxiety	Stress	Disability
Sally	44 years	Paracetamol, AD	Normal	Mild elevation	Normal	2
Bettina	44 years	Low dose AD	Normal	Moderate elevation	Mild elevation	2
Jane	51 years	-	Normal	Moderate elevation	Normal	6
Jacqui	45 years	Low dose AD	Moderate elevation	Mild elevation	Normal	4
Kim	33 years	Paracetamol, AD, AI	Moderate elevation	Moderate elevation	Normal	12
Michael	65 years	Oral opioid	Normal	Normal	Normal	4

Note 1. AD = anti-depressant, AI = anti-inflammatory
Note 2. Disability scores are from the RMDQ (Roland & Morris, 1983). Scores range from zero to a maximum of 24.
Note 3. Higher scores indicate greater pain-related disability. Depression, anxiety and stress score categories are from the DASS (Lovibond & Lovibond, 1995b). Cut-off scores for each of the severity categories are reported in *Section 5.5.2.4* in Chapter 5.

Table 6.2

Medication, Measures of Depression, Anxiety, Stress and Disability – participants excluded after initial interview.

Pseudonym	Age	Medication	Depression	Anxiety	Stress	Disability
Joan	69 years	Paracetamol	Normal	Normal	Normal	**17**
Jim	55 years	Codeine, anti-inflammatory	Moderate elevation	Normal	Moderate elevation	**18**
Eileen	63 years	Slow release opioid, anti-depressant, paracetamol	**Extremely severe elevation**	**Extremely severe elevation**	**Extremely severe elevation**	**18**
John	77 years	Slow release opioid, anti-depressant, paracetamol	Mild elevation	**Severe elevation**	Mild elevation	10/24

Note 1. AD = anti-depressant, AI = anti-inflammatory
Note 2. Disability scores are from the RMDQ (Roland & Morris, 1983). Scores range from zero to a maximum of 24.
Note 3. Higher scores indicate greater pain-related disability. Depression, anxiety and stress score categories are from the DASS (Lovibond & Lovibond, 1995b). Cut-off scores for each of the severity categories are reported in *Section 5.5.2.4* in Chapter 5.

6.1.2 Theme identification.

Analysis of the data derived from the interviews identified two main themes - resistance processes and negative pain related experiences. Resistance processes refers to positive factors that

appeared to promote an ability to live with pain. These can be seen in Table 6.2. Resistance processes were further coded into nine subcategories. These were stoicism, confidence and motivation to manage pain, having a management plan, physical activity, support from health professionals, getting a diagnosis, cognitive strategies, and positive social experiences. Cognitive strategies and positive social experiences were further coded into four and three categories respectively.

Negative pain related experiences were those experiences that appeared to make life with chronic pain more difficult. These factors were coded into four subcategories which were pain-related losses, negative social experiences and negative impacts on mood and negative impacts on sense of self. These factors can be seen in Table 6.3. Both pain-related losses and negative social experiences were then further coded into four and two categories respectively.

Table 6.3

Identified Themes: Resistance Processes

1.	Stoicism.	
2.	Confidence.	
3.	Motivation to manage pain.	
4.	Having a management plan.	
5.	Physical activity.	
6.	Support from health professionals.	
7.	Getting a diagnosis.	
8.	Cognitive strategies.	
		Acceptance
		Comparisons to those worse off
		Positive outlook
		Distraction
9.	Positive social experiences.	
		Instrumental support
		Emotional support
		Feeling understood and validated

Table 6.4

Identified Themes: Negative Pain-Related Experiences

1. Pain-related losses.	
	Physical losses
	Social losses
	Workplace losses and problems
	Sleep disturbance
2. Negative social experiences.	
	Invalidation
	Negative couples' communication
3. Negative impacts on sense of self.	
4. Negative impacts on mood.	

6.1.3 Resistance processes.

6.1.3.1 Stoicism.

All six participants discussed the concept of personal strength and stoicism in relation to their pain condition. This largely referred to a determination not to give in to pain. Most participants felt this was a part of their personality that had pre-dated their pain. Jane said she didn't want the pain to dictate her life situation.

> *I don't know if it's determination or just not willing to give in…. it's almost like … something's trying to stop me doing stuff so I'm trying to fight against that. It's not like I'm super strong. It's just I just don't want this to get me. I'm determined to, if I want to work I'm going to work.*
>
> (Jane, aged 51)

Several participants described their stoicism as arising from their families or from earlier life experiences. Jacqui said growing up in a single parent family had taught her to keep drawing on her inner strength. Sally, described a resilient attitude that existed through her extended family, some of whom were affected by chronic pain themselves: "*I think it keeps away the 'woe is me' attitude. Because we don't let one another get low. And if someone did go there, we would be told to 'Pull our head in, get on with it'*. Michael also recognised that his capacity to cope came partly

from a learned attitude from his family. *"I think, I think so, it's just part of the family. It doesn't matter how bad the circumstances you just never give up"*. Jacqui considered self-reliance a strength in her pain management and said her earlier life experiences had helped her cope with having chronic pain.

> *When I was younger I was a distance runner and I would do anything from a 1500 metre race to the Sun Super Run and things like that. So you're constantly looking inside a bit more and just push a bit more, a bit more.* (Jacqui, aged 45)

6.1.3.2 Confidence to manage pain.

Michael, Jacqui and Sally said they had an innate sense of confidence that they could cope despite their pain and thought this may have come at least partly from their families. Jacqui said "*Yeah, I mean we've always been that sort of a family where you pick yourself up and get on with it 'what are you whinging about?' … and that's…what keeps me going*" Kim, Bettina and Sally recognised that recovery was up to them. Kim said *"I mean essentially everything is up to you at the end of the day, how you steer your life after something has happened."*

6.1.3.3 Motivation to manage pain - working and caring for others.

Sally and Jane both reported they were motivated to cope with pain because they needed to work. Sally also needed to take care of her elderly parents.

> *If I don't do what's right (for me) I can't … fulfil my role in my family unit. Because I still have to earn an income and I have a father with dementia. And a mother that is caring for him so I have to be able to function to support them.* (Sally, aged 44)

Caring responsibilities for others appeared to foster a sense of resilience. Kim recognised that playing a supportive role in her young niece's life motivated her to keep active. She described this relationship as playful and free of adult judgements and expectations:

And she doesn't care, she's got aunties in her life up in Melbourne that have great jobs, and earn a lot of money, and they'll give her Wiggles Concert tickets or whatever. I bring her Playdoh, and stickers and stuff that cost me four dollars at the Reject Shop. And at the moment because she's four, it's fine. (Kim, aged 33)

Jacqui also said that doing practical helpful things for others helped her cope with her pain. She had noticed that thinking of others helped her feel better. She also noticed that her pain coping was partly driven by trying to reduce the impact of her condition on others. Jacqui said that despite pain, she wanted to preserve her relationship with her family.

So that was probably the kick up the backside that I needed. I wasn't going to let pain… ruin a relationship with my Mum and my kids. They deserved better, so that was probably the incentive I suppose, to get up and have a go. (Jacqui, aged 45)

6.1.3.4 Having a management plan.

All participants said a specific management plan was important. For many, this meant keeping physically active, accessing professional treatment, getting adequate sleep and optimising medications. There was emphasis on discovering an effective approach and then committing to adherence. Sally reported she had spent time refining a management plan that worked for her and then committed to sticking to it.

You know I suppose, I could be miserable and make everyone else miserable around me, or I could get on with my life…. I just had to get off my butt and put the effort in and do the work myself so…you have to find what works for you because not everyone's pain is the same and I have found what works for me. (Sally, aged 44)

Jane identified that sticking to her management plan provided her with a sense of hope that her pain might improve. *"I've still got my eyes open for, this might not be forever if I do my stretches and, and I've been doing like, relaxation tapes at night…."*.

6.1.3.4 Physical activity.

Most participants believed that exercise was essential to health, even when in pain, and reported that working with GPs, physiotherapists and exercise physiologists helped them slowly become more active. Jane stated that recommencing exercise represented a turning point in her recovery. "*It was just focusing on my body and moving it. I don't know, just makes you feel like you're doing something. (I realized) I'm going to be in pain whether or not I do this, or not do this*". Michael, aged 65, said that by working with physiotherapists he gradually re-taught his body that not every movement was going to be associated with pain. Bettina described that her GP had helped allay her anxiety about becoming more active.

> *Being more confident to take on physical exercise (helped me)….. my GP did help. She encouraged me, "Don't be scared of that. If you do it and it's too hard, don't push yourself, but if you try and you get some benefit from it, then that … would be better for you." Again, it was just hearing it from someone that I trusted that I thought, of course, I should be doing that sort of thing.* (Bettina, aged 44)

Sally also said keeping physically active took personal discipline.

> *Exercise is so important, there's some mornings you are lying in bed and you are so sore from being in bed and you don't feel like doing it but you know that by the time you get down the end of the road, you are going to be feeling so much better so you just have to drag yourself out of bed and make yourself you do it.* (Sally, aged 44)

Kim identified links between physical activity, positive mood and engaging socially with others: "*So I leave the pool feeling like my old self, you know, like my old self, really good… just feel really upbeat and positive, you know, and I might go to the supermarket, and shopping, and I might go home and ring a friend.*"

6.1.3.5 Support from health professionals.

Several participants reported that health professionals who took their symptoms seriously significantly fostered their ability to cope. Professional treatment from GPs, physiotherapists and massage therapists was generally reported to promote coping. Michael talked at length about how good professional treatment had helped him cope:

> *I think that's been … a good part of the coping mechanism, is that you've got someone you can rely on, they know exactly what's wrong, and he didn't need x-rays or anything like that, he could tell me exactly what was wrong.* (Michael, aged 65)

For some, feeling supported seemed to arise from a sense that they felt heard and were understood by health professionals. For some, it took 'doctor shopping' and seeing several GPs before they met someone who seemed to understand their symptoms. Others reported the main benefits they derived from health professionals were related to physical and psychological treatment. Four participants reported that provision of information related to their condition was critical to being able to cope and promoted acceptance. Bettina commented:

> *For me (the knowledge) is more about putting the symptoms into perspective to some extent, accepting that they're there but accepting that just because you've got some of those things that hold you back doesn't mean that you can't still participate in a more full way.*
>
> (Bettina, aged 44)

6.1.3.6 Getting a diagnosis.

Five participants said getting a diagnosis legitimised treatment, helped plan management and promoted acceptance. For some, a diagnosis represented a turning point in recovery.

> *It wasn't until they finally diagnosed fibromyalgia and I think it was then more the fact that they could say to me 'This is what you have got' and then you go 'Right I am not an idiot, I am not a hypochondriac, there is something wrong with me, I can get on and I can deal with this.* (Sally, aged 44)

By contrast, all participants reported feeling frustrated, anxious and invalidated as a result of not having a diagnosis. This led some to question whether their pain was 'in their head'. Michael described that early after onset of his pain he found it particularly difficult to navigate a system where he felt there wasn't anyone who was helping him to find a diagnosis.

6.1.3.7 Cognitive strategies.

All participants reported a range of cognitive strategies they used in order to cope with chronic pain. These included acceptance, comparing to those worse off, maintaining a positive attitude and distraction. Pain acceptance was frequently mentioned. Michael described his pain-coping approach as one that incorporated acceptance, patience and stoicism. Sally reported that acceptance of pain involved an initial grief reaction and then acceptance helped her re-engage with life.

> *Well you can be a victim …. but it's not going to change it, it's still going to be there… So you can wallow in self-pity or you can accept the fact that this is the way it's going to be. So I suppose it's almost like going through a grieving period, and you can go 'Right, I can sit around the house and not get on with my life' or 'I can get out there and enjoy myself*

and get on with it and to do that, well this is what I need to do. (Sally, aged 44)

Bettina reported that acceptance was linked to getting a diagnosis. She was also clear that acceptance did not mean giving up and was in fact linked to optimism.

I'll try and accept that I've got this banner now and I sit under it but I can … move beyond it … I'm just going to have to make positive, informed choices about how I will treat the condition and in so doing be able to take on more normal things again.

(Bettina, aged 44)

Other participants reported that making mental comparisons to those worse off than them aided coping. Sally reported she had several friends with life threatening health conditions and this reminded her that what she was dealing with was not so bad. Bettina found solace that her pain condition was not life threatening. Michael made historical references to his family, reflecting that what he was dealing with was nothing compared to what they faced.

You think about what your grandmother went through in the Depression …and all of that, your family actually coped with that. They're your relatives and your forebears and all the rest of that, they coped with all of that, I've got nothing to cope with compared to them.

(Michael, aged 65)

Other reported cognitive strategies included positive thinking. For some participants this incorporated an attitude of optimism and hope. Kim described making an effort to 'be in the moment'. Bettina described making a conscious effort to keep her thinking positive.

It would be rather than get bogged down in all the negative sides of my condition...it would just be put good thoughts in my head or think about the fact that you'll be able to pick up the

kids at the end of the day, you'll be able to do that. It'll be okay. It is a state of trying to stay always on the positive side of the ledger" (Bettina, aged 44)

Jane thought developing a more positive attitude in her workplace helped her make more social connections. Michael reported that he tried to keep a positive attitude despite struggles with chores.

Sometimes you get in the grumps and you don't want to do any of it, but you've got to, at a certain point, get into it and do it. So I think everything that comes up I try and turn it into a positive. (Michael, aged 65)

Two participants reported that distraction was helpful. Jane found that it was possible to tune out to the pain and felt this was important. Jacqui also found she could distract from the pain:

The vibration of the mower will within minutes give me a severe headache …but if I ignore that and I think "I really want this mown because it's going to look good mown"… the pain is still there, but you just concentrate on the job that you're doing, rather than constantly thinking about the pain that you're in. (Jacqui, aged 45)

6.1.3.8 Positive social experiences.

Participants reported experiencing a range of positive social experiences that appeared to enhance their capacity to cope with chronic pain. These included experiencing instrumental and emotional support and feeling understood and validated. Four participants said that both instrumental and emotional support fostered coping. Four participants identified that instrumental support fostered their coping efforts. For Jane this support was at times a two-way street. *"If he wasn't supportive he wouldn't be doing what he does. And if I'm exhausted and hurting and, he'll go and cook tea or, but then we do that for each other"*. Jacqui said practical support was helpful, but not if it was motivated by pity.

> *I don't want pity. That's one thing I don't want, you know. If someone says 'I'll give you a hand,' because they genuinely want to give me a hand, that's fine, but if they want to do something for me because they think I can't then I'm probably more likely to get my back up*. (Jacqui, aged 45)

Both emotional and instrumental support was experienced within couple relationships and within families. Jacqui, aged 45, felt supported when her partner simply noticed her pain: "*I don't talk about it with him. I said to him 'one of us living with it is enough.' So I don't moan and groan to him….but if I'm a bit quiet or something he'll say 'Sore today?' 'Yep.'*"

Discussing instrumental support, Jane noted it was helpful when her partner accepted her physical limitations or negative moods without additional negotiation about daily chores. Sally remarked that it was her mother, rather than her partner, who provided the most emotional support. Several other participants also described support obtained from outside their partner relationship. Jacqui recognised that simply having stability in her family life helped her cope with her condition and also expressed gratitude towards her mother for her support and her encouragement to keep going.

Feeling understood by others seemed to be important both in terms of the pain itself as well as associated physical limitations. Sally said members of her extended family had experienced similar health problems and this was helpful: *"We all understand one another, we know what one another is going through"*. Kim also described how supportive it was to have friends accommodate her physical limitations, without needing to discuss them:

> *It's just having them show a little bit of understanding. We don't have to have a big conversation about it, but if a friend says…it would probably be better for you if we drove rather than catch the tram or the train, just that one little thing.... showing me that they understand*. (Kim, aged 33)

6.1.4 Negative pain-related experiences.

6.1.4.1 Pain related losses.

All participants reported multiple pain-related losses. These included physical, social, sleep and work-related losses. Physical and social losses were reported with almost equal frequency. Michael stated his physical function had been impacted by pain.

> *It's not being able to… do the physical things the way that you have done them in the past, you try to do something and you just get stopped by a wall of pain. So you don't do it, and then you don't do it again.* (Michael, aged 65)

Many social losses resulted from participants withdrawing from activities because they felt they couldn't manage them. Emotional reactions to these losses, such as frustration, sadness, grief, anger and anxiety were frequently reported. Jane, aged 51, commented that "*Every team or work Christmas party is ten-pin bowling or doing something really active and I have to say no and I'm really annoyed that I say no because I feel like I'm isolating myself from the group*." Several participants also described pain negatively impacting on their sleep. Bettina and Kim reported that lack of sleep impacted on partner relationships and contributed to social withdrawal due to fatigue. Jane said that knowing how to manage sleep was an important part of her overall pain management strategy.

6.1.4.2 Negative social experiences.

Participants reported a range of negative social experiences as a result of having chronic pain. These included negative communication within couples and lack of understanding and invalidation by others. Pain was reported to impact negatively on relationships, especially with partners. Some said pain made them feel irritable and partners took the brunt of this. Difficulties associated with negotiating daily chores were mentioned. This was particularly so for those with

fluctuating function. Jane, aged 51, said that one cause of interpersonal stress was when partners felt guilty and felt forced to help with daily chores: *"(Support) does come with a down side because he doesn't like doing it but he says, "Oh, I've done this vacuuming all morning, you've just sat there and done nothing."*

Other negative social experiences included lack of understanding, lack of connection to others and invalidation. Having the validity of a diagnosis questioned was reported as particularly difficult. Some participants described wanting to reject the diagnosis in favour of something more 'real'. Participants also reported invalidation associated with the invisibility of chronic pain and having a diagnosis that may be seen by others as questionable.

> *Oh maybe just that with problems like fibromyalgia there has been that stigma around…is it real?… Because it's an invisible illness, and there's that constant feeling that....people don't think your pain is genuine, or they think it's all in your head*".
>
> (Kim, aged 33)

> *"Yeah, well that's something I didn't want, I didn't want one of these iffy sort of conditions. I wanted something that okay, we can give you a splint or artificial knee or something*.
>
> (Jane, aged 51)

6.1.4.3 Negative impacts on sense of self.

Two participants, Kim and Michael, reported negative effects of pain on their sense of themselves, their confidence and self-esteem. Other participants did not directly report detrimental effects of pain on their sense of self, but described earlier, preferred versions of themselves that pre-dated their pain. Two participants, Sally and Jane, reported that their core sense of self was unchanged by pain. Jane, aged 51, commented, "*No, I think I've just… proved probably that I'm*

tough, tougher than I thought I was." Jacqui said she had always been a 'hands-on person' and was determined to get back to that. With a sense of loss, Kim described her pre-pain self as busy and able to achieve a lot.

6.1.4.4 Negative effects of pain on mood.

All participants described negative pain-related emotional experiences. These included low mood, anger, anxiety and intolerance. Several participants reported feeling frustrated in regard to physical limitations. Michael, aged 65, said he often felt angry as a result of pain - "*I can lose my temper quite easily, where I was never short-tempered"*. Jane, aged 51, recognised a link between mood and pain, saying "*Yeah, I know if I'm having a down day for other reasons, if someone's upset me or something I will feel pain because of that….if I'm happy the pain sort of melts away a bit."*

6.2 Summary of Results and Implications for Study Three

Study Two provided further insights into the inter-relations identified in the literature as well as those that were demonstrated in Study One. Study Two highlighted factors that may contribute to improved adjustment in chronic pain and provided direction for Study Three model development. For example, participants described how social support and positive emotions appeared to promote confidence to manage pain. Participants described that acceptance was linked to improved physical function. Many resistance processes described by participants were consistent with those reviewed in Chapter Two. For example, feeling confident to manage pain and having a specific management plan reflected the concept of pain self-efficacy. Being physically active reflected the construct of pain self-efficacy as well as a lack of fear-avoidance. Maintaining a positive attitude and comparing to those worse off reflected the constructs of positive affect (PA)

and optimism. Positive social experiences appeared to foster an ability to live with pain and reflected the constructs of emotional and instrumental social support.

Of interest was the finding that some participants appeared to be motivated to manage their pain in order to be able to continue to engage in roles and activities that held meaning for them. One interpretation of this finding was that these participants were motivated to manage their pain in order to live according to their values. Both acceptance and values-based actions are central tenets of the psychological flexibility model of chronic pain and both are suggested to promote positive adjustment to pain (McCracken & Morley, 2014; McCracken & Vowles 2008; Vowles & McCracken, 2008). In this approach, values-related processes can be seen as behaviours that strengthen those aspects of a person's life that are important to them (Hayes, Strosahl & Wilson, 1999). The current results as well as existing literature, presented below in Section 6.2.1, indicate values-based living is linked to positive pain adjustment.

At the same time that resistance factors were described, a number of risk factors for poorer adjustment to pain were also reported. Categories of negative pain-related experiences included pain related losses, negative social experiences and impacts of pain on sense of self and mood. Pain-related losses appeared to be associated with negative emotional states that included sadness, frustration, anger and grief. Extensive negative effects of pain on mood were also described and included anxiety, anger, frustration and intolerance. Because these negative emotional experiences were conceptually distinct from anxiety and depression, negative affect (NA) was included as a personal risk factor in Study Three in place of anxiety and depression. The literature pertaining to NA as risk factor for pain-related disability and QOL was presented in Chapter Four.

Lastly, a major focus of discussion in Study Two related to social experiences, with invalidation frequently mentioned. Invalidation is generally considered to involve hostility or an ignoring response to an expression from another person. A small amount of research has

demonstrated direct negative associations between invalidation and pain-related function. This literature is presented below in Section 6.2.2. Invalidation was included as a social-ecological risk factor in Study Three.

6.2.1 Values-based living as a predictor of pain-related disability and quality of life.

The degree to which people with chronic pain are aware of their values and use them to motivate behaviour has been shown in multiple studies to be positively associated with improved pain-related function. McCracken and Yang (2006) investigated the relationship between living in accordance with values and disability in a heterogeneous pain clinic sample of 140 patients. Success in living in accordance with values contributed significantly to explaining the variance in the disability measure over and above that explained by measures of pain acceptance, suggesting that values awareness represents a resistance factor for pain adjustment that is distinct from acceptance. McCracken and Vowles (2008) also showed that values-based living measured at the time of an initial evaluation in 115 pain clinic patents with mixed diagnoses demonstrated a moderate strength negative association with physical disability on the first day of treatment, which occurred approximately 18 weeks later.

A commitment to living in accordance with values has also been shown to be positively associated with physical function in primary care pain patients. McCracken and Velleman (2010) investigated the relative contribution of values awareness and acceptance to a range of chronic pain outcomes including physical disability, in a heterogeneous sample of individuals with chronic pain recruited from general medical practices in England. Values awareness contributed significantly to the variance in disability after adjusting for the effects of both pain intensity and pain acceptance. Further, in heterogeneous community dwelling sample of adults with chronic pain, Trompetter and colleagues (2013) found that values-based living explained additional variance in measures of

anxiety and depression, positive mental health, and pain interference scores, over and above that explained by measures of acceptance and mindfulness. Reductions in disability have also been reported as a result of a values and acceptance-based psychological intervention in a primary care setting (Vowles, Wetherell & Sorrell, 2009).Together, these results suggest that values-based living may contribute uniquely to explaining variance in measures of chronic pain adjustment.

Fewer studies appear to have examined the relationship between values-based living and QOL, although a study of the relationships between values-based living, acceptance and mindfulness with health and vitality in a sample of 98 Singaporean health workers demonstrated strong positive correlations between these measures and explained a moderate and significant proportion of variance in measures of health and vitality (McCracken & Yang, 2006). The research examining a potential moderating or mediating role specifically of values-based living in cross-sectional research currently appears to be limited. However improvements in acceptance and the ability to live in accordance with values have been linked with functional gains in pain clinic patients, suggesting a potential mediating role of values in rehabilitation interventions (Vowles & McCracken, 2008; Vowles, McCracken & O'Brien, 2011). As the capacity for pain-affected individuals to live according to values appears to be associated with reduced pain-related disability, a measure of values was included as a resistance stress-processing factor in Study Three.

6.2.2 Invalidation as a predictor of pain-related function and quality of life.

Invalidation is generally seen as a hostile or an ignoring response to an expression from another person. This contrasts with validation, which is an expression of empathy, respect or acceptance in response to another's disclosure (Leong, Cano & Johansen, 2011). Invalidation has been shown in some research to be associated with negative effects on pain-related physical function and health-related quality of life (HRQOL). For example, Ghavidel-Parsa and colleagues

(2015) reported links between experiences of invalidation and QOL and impact of fibromyalgia in 112 Iranian females with fibromyalgia. Invalidation was measured by a Persian version of the Invalidation Inventory (3*I; Kool et al., 2010). Health-related QOL and impact of fibromyalgia were measured with Persian versions of the Short Form 12 (SF-12; Ware et al., 1993) and the Revised Fibromyalgia Impact Questionnaire (RFIQ; Bennett et al., 2009). Moderate strength negative correlations were reported between the discounting by medical professionals and the physical and social function, mental health and vitality subscales of the SF-12. Moderate strength negative correlations were also found between the discounting in work environments subscale of the 3*I and the physical and social function subscales of the SF-12. Only weak associations were observed between the 3*I subscales and the physical function subscale of the RFIQ, but a moderate strength positive correlation was reported between the discounting in work environments subscale of the 3*I and the overall impact score on the RFIQ. Taken together, these results suggest that experiences of invalidation may be linked to poorer physical function, disease impact and QOL for people with fibromyalgia.

Further support for possible links between experiences of invalidation and physical function were provided by Wernicke, de Witt Huberts and Wippert (2015) who demonstrated invalidation was predicted physical function in a predominantly female sample of 92 community-based individuals with chronic low back pain. Invalidation was measured with a German version of the 3*I (Kool et al., 2010), pain-related function was assessed as interference by pain with the Von Korff Questionnaire (Von Korff, Ormel, Keefe & Dworkin, 1992). Bivariate associations between invalidation and outcomes were not reported but discounting was a significant predictor in a regression analysis predicting pain-related function, explaining 11% of the variance in this measure.

Mavandadi, Rook and Newsom (2007) also found lower rates of invalidation were linked to lower disability in older, non-pain affected Canadians. Data was collected over two years in 482 adults reporting little or no disability at baseline. Adjusting for covariates such as gender, age, ethnicity and education level, low or absent negative social exchanges were associated with significantly lower levels of reported disability after two years. Contrasting somewhat with the above findings, Kool, Van Middendorp, Lumley, Bijlsma and Geenen (2013) explored relationships between invalidating and discounting social experiences, social support and HRQOL in 1455 patients with fibromyalgia, rheumatoid or osteoarthritis, ankylosing spondylitis or other rheumatic diseases. Health-related QOL was measured with the physical and mental component scores of the Short-Form 36 (Ware et al., 1993). Both discounting and lack of understanding were negatively associated with the SF-36 physical component score but only at weak strength. Discounting was a significant predictor of the physical component score in a regression analysis that also included age, gender, type of disease, social support and a measure of psychological morbidity. The amount of variance explained in the outcome measure was 11%. The association between invalidation and the mental component score of the SF-36 was stronger than for the physical component score.

Links have also been made between affective experiences and invalidation. For example, invalidation was associated with poorer maintenance of PA in healthy subjects (Linton, Boersma, Vangronsveld & Fruzzetti, 2012) and invalidation was associated with increased levels of NA in a small sample of nurses with chronic back pain (Vangronsveld & Linton, 2012). Invalidation has also been reported to be negatively associated with social functioning (Kool et al., 2010, Asbring & Narvanen, 2002; Werner, Steihaug & Malterud, 2003).

In summary, invalidation and other negative social experiences are reported to be common for those living with chronic pain. A small number of studies have identified that invalidating social

experiences may be linked to poorer physical function in individuals with chronic pain. It is was thus included as a social-ecological risk factor in Study Three. Based on the combined results of Studies One and Two, Study Three examines an expanded version of the risk-resistance model. The design for Study Three was cross-sectional and used purposely collected data. Chapter Seven describes Study Three methodology and sample. Chapter Eight presents Study Three results.

Chapter Seven – Study Three Rationale, Design and Method

7.1 Rationale

Deficits currently exist in the chronic pain literature in relation to indirect and interactive effects of risk factors associated with pain adjustment processes. There is also a lack of research addressing the influence of resistance factors on positive pain adjustment outcomes. In order to address these gaps, Study Three aimed to explore the direct, indirect and interactive effects of a range of risk and resistance factors on two pain adjustment outcomes, pain-related disability and quality of life (QOL). It was intended that Study Three findings could be used to inform positive clinical interventions for those with chronic pain as research has tended to focus instead on interventions addressing risk factors.

Study Three was informed by results of both Studies One and Two. It tested an expanded version of the theoretical model examined in Study One, which was adapted from Wallander and Varni's (Wallander et al., 1989; Wallander & Varni, 1998) risk-resistance model of adjustment to paediatric disability. Study Three aimed to extend the results of Study One by increasing the range of risk and resistance factors in the model. This was important because variables included in Study One were constrained to those available in the clinical database. Secondly, Study Three also aimed explore positive, as well as negative, adjustment processes. The model used to guide the Study Three research aims and hypotheses can be seen below in Figure 7.1. The current chapter outlines the design, aims, hypotheses and method of this final study. The method section describes participant characteristics and statistical analyses.

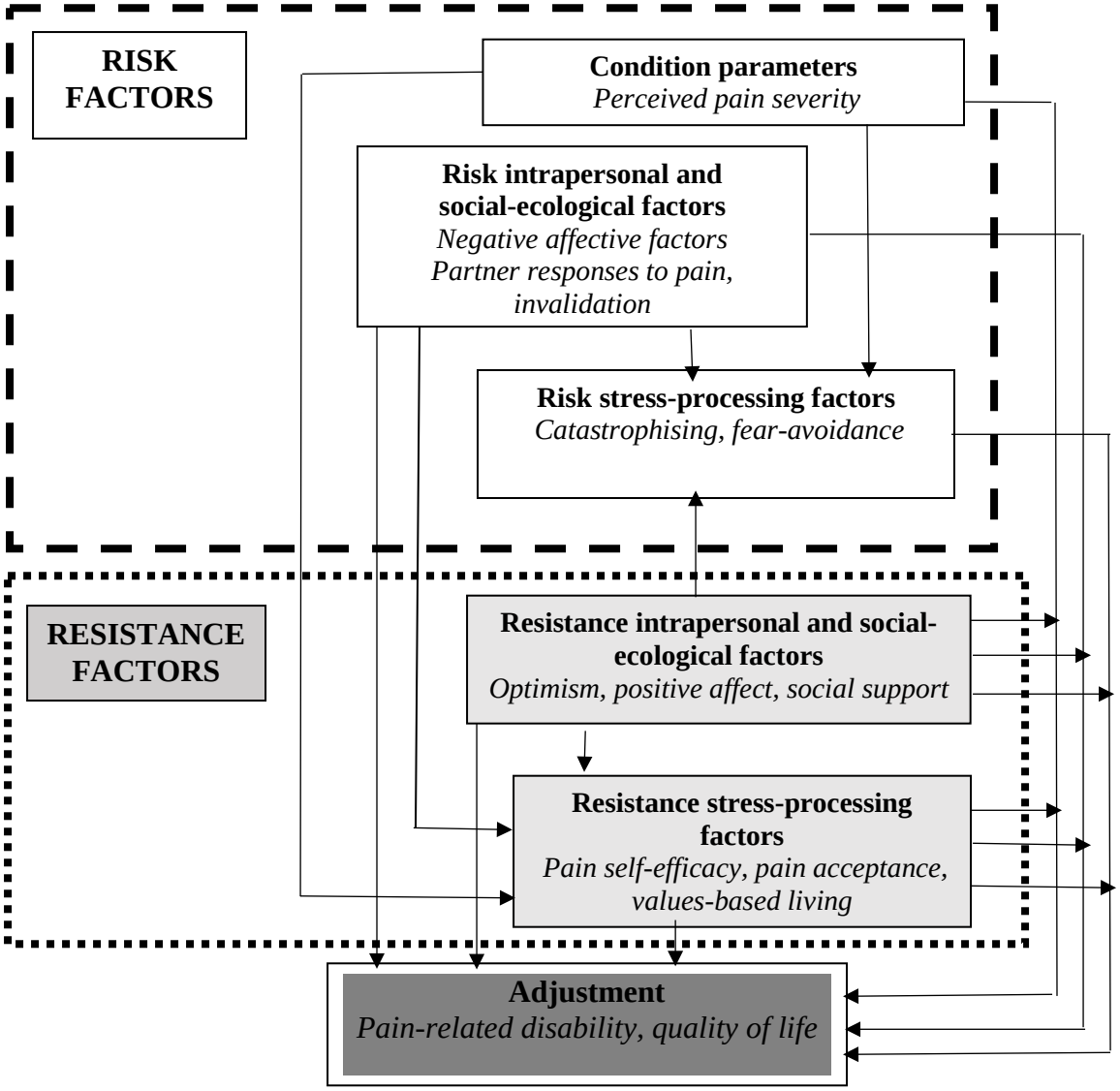

Figure 7.1 Proposed model of adjustment to chronic pain (adapted with permission from Wallander et al., 1989; Wallander & Varni, 1998).

Note 1: Study hypotheses are indicated by arrows.
Note 2: Existing literature was used to guide placement of variables within the model, hence stress-processing and social-ecological variables are placed as both risk and resistance factors.

7.2 Aims

Guided by the adapted risk and resistance conceptual model (Wallander et al., 1989; Wallander & Varni, 1998) depicted above in Figure 7.1, Study Three aimed to investigate, using cross-sectional data, the direct, moderating and mediating effects of the identified risk and resistance factors on the pain-related disability and QOL of those living with chronic pain. Consistent with the theoretical model, three statistical effects of psychosocial factors on pain-related disability were hypothesised:

d. Direct effects

e. Mediated effects

f. Moderated effects

The research aims therefore were:

1. To assess the strength of the relationships between risk and resistance factors with pain-related disability and QOL.
2. To assess the extent to which the risk factors (perceived pain severity, catastrophising, fear-avoidance, negative affect (NA), solicitous and punishing partner responses to pain and invalidation) explain variation in pain-related disability and QOL.
3. To assess if the resistance factors (pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support) contribute unique variance to pain-related disability and QOL over and above that accounted for by the risk factors.
4. To explore whether the stress-processing factors (catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living) mediate the effects of condition parameters, intrapersonal and social-ecological variables on pain-related disability and QOL.

5. To investigate whether the resistance factors (pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support) moderate the relationships between risk factors (perceived pain severity, catastrophising, anxiety, depression and solicitous and punishing partner responses) with pain-related disability and QOL.
6. In order to place the current results in the context of the existing literature, a final aim was to compare the psychosocial and functional profile of the current sample to normative pain clinic data.

7.3 Design

Study Three design replicated that of Study One. It employed a quantitative approach, using cross-sectional data, to investigate factors associated with pain-related physical function in a community-based sample of individuals with chronic pain. Data were collected online and in hard copy from a range of recruitment sites. Risk factors were pain severity (condition parameters), NA (intrapersonal factor), punishing and solicitous partner responses to pain and invalidation (social-ecological factors) and catastrophising and fear-avoidance (stress-processing factors). Resistance factors were, optimism and positive affect (intrapersonal factors), social support (social-ecological factor) and pain self-efficacy, pain acceptance and values-based living (stress-processing factors).

7.4 Hypotheses

Study Three hypotheses were guided by the conceptual model and by the literature review. The model in its entirety as it applies to Study Three can be seen in Figure 7.1. The literature review demonstrated that significant direct relationships exist between pain severity (Arnow et al., 2011; Raftery et al., 2011), NA (Agar-Wilson & Jackson, 2012; Karsdorp & Vlaeyen, 2011; Seebach et al., 2012), catastrophising (Jensen et al., 2011) and fear-avoidance (Perry & Francis, 2013; Zale et

al., 2013) with pain-related disability and QOL. There is also some evidence to suggest that solicitous and punishing partner responses to pain exert a negative influence on physical adjustment (Leonard et al., 2006) and that invalidation experiences are linked with poorer physical health (Ghavidel-Parsa et al., 2015; Wernicke et al., 2015).

The literature review also demonstrated that significant direct relationships exist between pain self-efficacy (Burke et al., 2015), pain acceptance (McCracken, 1998; Vowles, Sowden, & Ashworth, 2014), values-based living (Vowles et al., 2014), optimism (Tsakogia et al., 2011; White et al; 2011) and positive affect (Seebach et al., 2012; White et al., 2012) with pain-related disability and QOL. The evidence for a direct influence of social support on chronic pain adjustment outcomes is inconsistent (Campbell et al., 2011), however, the literature suggests that social support may be associated with improved pain adjustment outcomes in some settings (Demange et al., 2004; Evers et al., 2003) and social support has been shown to have protective effects on adjustment outcomes in broader health settings (Penninx et al., 1997). Therefore, consistent with this literature, the following hypotheses were proposed:

1. Higher levels of risk factors; perceived pain severity, NA, catastrophising, fear-avoidance, punishing and solicitous partner responses to pain and invalidation will be associated with greater pain-related disability and lower QOL.
2. Higher levels of resistance factors; pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support will be associated with lower pain-related disability and higher QOL.

In line with research suggesting that adjustment to chronic conditions is better predicted by a combination of risk and resistance factors than by single factors alone (Greenberg et al, 2001; Wallander et al., 1989; Wallander & Varni, 1998), the third hypothesis is:

3. Resistance factors (pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support) will account for additional variance, over and above that explained by the risk factors (perceived pain severity, catastrophising, NA, fear-avoidance, punishing and solicitous partner responses to pain and invalidation), in explaining pain-related disability and QOL.

The adapted version of Wallander's risk-resistance model (Wallander et al., 1989; Wallander & Varni, 1998) (see Figure 7.1) being tested in this study proposes two types of indirect effects: a) mediator effects when some or all of the direct relationship between pain intensity, intrapersonal and social factors and disability is attributed to stress-processing factors, and (b) moderator effects where the relationship between the risk factors and adjustment is moderated by levels of the resistance factors. Therefore the following indirect effects are hypothesised:

4. Catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living, will mediate the relationships between condition parameters and intrapersonal and social-ecological factors (both risk and resistance) with pain-related disability and QOL. These proposed effects can be seen in Figure 7.2.

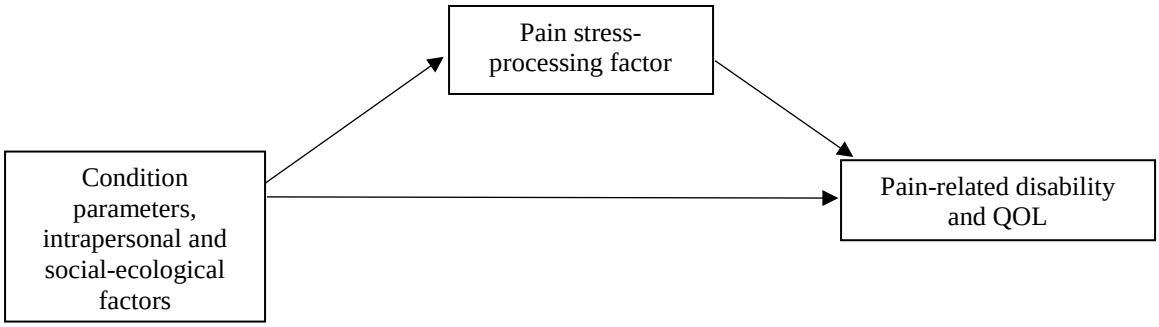

Figure 7.2 Mediator model.

5. Resistance factors (pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support) will moderate, or lessen, the negative relationships that exist between risk factors (perceived pain severity, NA, partner responses to pain, invalidation catastrophising and fear-avoidance) with pain-related disability and QOL. Refer to Figure 7.3.

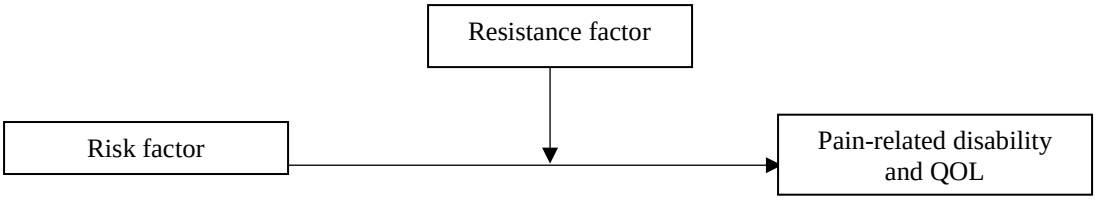

Figure 7.3 Moderator model.

6. As the current sample is a community-based one, with at least some participants who were no-treatment seeking, a final hypothesis was that the current sample will report significantly lower levels pain related-disability compared to normative pain clinic data.

As outlined in Chapter Three, the hypothesised models depict unidirectional relationships between predictors and outcomes. Although it is acknowledged these relationships may in reality be better represented as bi-directional, unidirectional relationships allow a complex model such as the one guiding this research to be statistically explored. Due to the cross-sectional data, causality cannot again be established. Therefore, comments in the current results section regarding the influence of one variable on another refer to statistical associations, not cause and effect relationships.

7.5 Method

7.5.1 Participants.

The final study sample comprised 281 participants, 222 women (M_{age} = 50.86 years, SD = 14.22) and 59 men. Participant recruitment sites are listed in Table 7.1. Participant demographics are listed in Table 7.2. The majority of participants identified English as their first language (97.3%). In relation to treatments received or medications taken for pain, only three of the 281 participants reported having attended a multidisciplinary pain treatment program, although a large number of participants reported treatment from a range of health disciplines that included medical, physiotherapy, psychology, massage therapy, acupuncture and naturopathy. A range of different pain medications were also reported.

Table 7.1

Participant Recruitment Sites

	Frequency	Percent
Online pain forum or other online link	152	54.09
Community Health Centre	36	12.81
Other	27	9.61
Medical, physiotherapy or other health practice	26	9.25
Exercise facility or club	33	11.73
Community Group	4	1.42
Missing	3	1.07
Total	281	-

Table 7.2

Demographic Details of Study Three Participants

Demographic		
Gender		
	Male	59 (21%)
	Female	222 (79%)
Age		
	Mean (SD)	50.86 years (14.22)
	Range	18 – 87 years
Pain Duration		
	Mean (SD)	12.19 years (11.02)
	Range	1 – 69 years
Education post Year 9		
	Mean (SD)	5.14 years (3.47)
	Range	0 – 27 years
	0-5 years	151(53.7%)
	6-10 years	112 (39.9%)
	>10 years	16 (5.7%)
	Missing	2 (.71%)
Marital status		
	Married / defacto	171 (60.8%)
	Single	59 (21.3%)
	Divorced / separated	51 (18.1%)
Employment status		
	Unemployed	95 (33.8%)
	Working part-time	76 (27.0%)
	Working full time	38 (13.5%)
	Retired	42 (14.9%)
	Studying	15 (5.3%)
	Carer/parent	14 (5.0%)
	Missing	1 (.35%)
Compensation Status		
	Non-Compensable	236 (84.0%)
	Traffic Accident Commission	6 (2.1%)
	WorkSafe	39 (13.9%)
Income		
	Less than $30,000	98 (34.9%)
	Between $30,000 and $70,000	90 (32.0%)
	Between $70,000 and $120,000	66 (23.5%)
	More than $120,000	22 (7.8%)
	Missing	5 (1.8%)
Diagnoses		
	Multiple sites +/- low back pain and fibromyalgia	49 (17.4%)
	Low back or pelvic pain	43 (15.3%)
	Osteoarthritis or peripheral joint pain	34 (12.1%)
	Fibromyalgia	33 (11.7%)
	Complex regional pain syndrome	19 (6.8%)
	Neuropathic pain	14 (5.0%)
	Rheumatoid arthritis / ankylosing spondylitis	14 (5.0%)
	Headache / Migraine / whiplash / neck pain	12 (4.3%)
	Missing or unspecified chronic pain	63 (22.4%)

7.5.2 Measures.

Several criteria were used to select Study Three measures including that the scale was psychometrically sound, it was suitable to the planned analyses and that it reflected the constructs identified in the literature review. Using these criteria, two measures were retained from Study One; the Punishing and Solicitous partner responses to pain from the Multidimensional Pain Inventory (MPI; Kerns et al., 1985) and the Pain Self-Efficacy Questionnaire (PSEQ; Nicholas, 1995). The two other variables that were also measured in Study One (pain severity, catastrophising) were measured by alternate scales in Study Three because these latter scales were psychometrically superior or better suited to the analyses.

7.5.2.1 Measures of adjustment.

7.5.2.1.1 Pain-related disability.

Pain-related disability was measured by the Pain Disability Index (PDI, Tait, Chibnall & Krause, 1990). The PDI is a brief measure of pain-related disability designed to assess the functional impact of chronic pain. The PDI consists of seven items reflecting seven different functional aspects of life; family and home responsibilities, recreation, social activity, occupation, sexual behavior and self-care activities. Items are scored on a 0 to 10 Likert scale, where zero indicates no disability and 10 indicates total disability. Respondents are asked to indicate the number that best reflects the extent to which they typically experience pain-related disability. Occupation is described in the scale as including non-paying jobs such as housewife or volunteer worker so this category is applicable even to those not formally employed. The questionnaire is scored by summing the number of items checked as true to calculate one total scale score. Scores range from 0 to 70, with higher scores indicative of greater pain-related disability. The PDI is

reported to have acceptable concurrent validity with other measures of pain-related function, has high reported levels of test-retest reliability and has been demonstrated to be sensitive to change (Tait et al., 1990). Cronbach's alpha is reported as .87 (Tait, Pollard, Margolis, Duckro & Krause, 1987), in this study it was .88.

7.5.2.1.2 Quality of life.

Quality of Life was measured by the Quality of Life Scale (QOLS; Flanagan, 1978; Burckhardt, Woods, Schultz & Ziebarth, 1989). The original QOLS (Flanagan, 1978) was developed in sample of 3000 American adults and measures quality of life in 15 items across five domains; material and physical well-being, relationships with others, social and community activities, personal fulfilment and recreation. Subsequent work was undertaken with the QOLS to validate its use in populations with chronic illness and an additional item 'Independence, doing for yourself' was added to the scale (Burckhardt et al; 1989). The revised QOLS is a 16 item questionnaire that is scored by summing individual item responses to form a total scale score. Respondents are asked to indicate on a seven point Likert scale, the degree to which they are satisfied by different aspects of their lives. Responses range between one (*terrible*) to seven (*delighted*). Thus scale scores range between 16 and 112, with higher overall scores indicating higher quality of life. The scale instructs respondents to respond to all items even if they are not currently engaged in that activity, as the scale instructions state that a person can be satisfied or not by the absence of that activity in their life. Sample items include satisfaction with 'Material comforts, home, food, conveniences, financial security', 'Expressing yourself creatively' and 'Independence, doing for yourself'.

The QOLS has been shown to have good criterion and discriminant validity (Burckhardt et al., 2003). Strong positive correlations of the scale have been demonstrated with measures of life

satisfaction and negative correlations with measures of disease impact (Burckhardt et al., 2003; Burckhardt, Archenholtz, & Bjelle, 1992). These associations have been demonstrated in samples of individuals with arthritis, lupus erythematosus and fibromyalgia (Burckhardt et al., 2003; Burckhardt et al., 1992). Reported Cronbach's alphas for the scale range between .82 and .92 (Archenholtz, Burckhardt & Segesten, 1999; Burckhardt et al; 1989; Burckhardt et al., 1992; Liedberg, Burckhardt & Henriksson, 2005). In this study, Cronbach's alpha was .93.

7.5.2.2 Risk factors.

7.5.2.2.1 Condition parameters – pain severity.

Pain severity was measured by the pain intensity items of the Brief Pain Inventory (BPI; Cleeland, 1991). The BPI was originally developed for measuring pain intensity and interference in malignant pain but has since been validated for use in chronic non-malignant pain conditions (Tan et al; 2004). Pain intensity is measured by four items designed to assess the variability of perceived pain severity over time. Items ask respondents to rate the severity of their pain on a scale of 0 to 10, rating their pain at its worst, least, average and 'right now'. Use of all four intensity items using a mean has been recommended (Cleeland, 1991). Specific permission was granted by the author's affiliated organisation (Department of Symptom Research, The University of Texas M. D. Anderson Cancer Center, personal communication, July 7, 2014) to use a modified version of the scale without a body chart that was suitable for online administration for this study.

Sound psychometrics of the BPI have been reported in non-malignant pain samples. The three factor structure of the scale was replicated and concurrent validity of the scale was demonstrated in a sample of 250 adults with arthritis or low back pain (Bann, Dodd, Schein, Mendoza & Cleeland, 2004). High internal consistency and criterion validity of the scale were also demonstrated in a heterogeneous sample of 440 individuals with chronic non-malignant pain (Tan,

Jensen, Thornby & Shanti, 2004). Cronbach's alpha for the pain intensity subscale of the BPI was originally reported as .85 (Cleeland, 1991), in this study it was.89.

7.5.2.2.2 Intrapersonal risk factor - negative affect.

Negative and positive affect was measured by Positive and Negative Affect Scale, Short Form (PANAS; Watson, Clark & Tellegen, 1988). The original 20 item form of the PANAS has been widely used in research to examine trait affect and has good reliability and validity (Watson et al., 1988). Each item of the PANAS describes an affective experience and respondents are asked to indicate the extent to which they generally feel that way. Items are scored between one (*very slightly or not at all*) and five (*extremely*). The subscales are scored by summing the item responses; higher scores therefore, reflect higher levels of positive or NA. Two-factor invariance of the PANAS reflecting the two subscales of positive and NA has been demonstrated in a non-clinical community sample of 1003 adults (Crawford & Henry, 2004). This same study demonstrated concurrent validity of the scale with strong correlations noted between the NA subscale with other well-validated measures of anxiety and depression (Crawford & Henry, 2004).

A shortened version of the PANAS was used in the current study which consists of 10 items, divided evenly into two subscales assessing positive and NA. Negative affect items of the PANAS short-form are afraid, nervous, upset, hostile and ashamed. Positive affect items include determined, alert and attentive. This shortened form of the PANAS has been validated in a in a cross-cultural sample of 1789 adults with comparable psychometrics reported to the original form of the scale (Thompson, 2007). For example, the two subscales of the 10-item PANAS short form were shown to correlate slightly more strongly with each other compared to the cross-scale correlations reported in the original validation of the long form of the scale (Watson et al., 1988), but are similar to cross-scale correlations reported by subsequent validation studies of the original

PANAS (Thompson, 2007). Concurrent validity of the short from of the PANAS was demonstrated with moderate to strong negative correlations reported between the NA subscale with measures of subjective well-being and happiness (Thompson, 2007). Cronbach's alphas for the NA subscale of the 10 item version of the PANAS were reported as .76 (Thompson, 2007), in this study it was calculated as .84.

7.5.2.2.3 Social-ecological risk factor - partner responses to pain.

Punishing partner responses to pain were measured by the punishing responses subscale in Section Two of the MPI (Kerns et al., 1985). Respondents are asked to score the frequency with which their partner engaged in punishing behaviours when the respondent was in pain. Scores range from zero to six, with higher scores reflecting greater frequency of that behaviour. The subscale is scored by calculating the response mean of the three items. Example items include (when I am in pain) ‘my partner expresses irritation at me’ and ‘my partner ignores me’. The MPI has established acceptable levels of reliability and concurrent validity (Kerns et al., 1985). Cronbach’s alpha for the punishing responses subscale of the MPI is reported as .84 (Kerns et al., 1985), in this study it was .89.

Solicitous partner responses to pain were measured by the solicitous responses subscale in Section Two of the MPI (Kerns et al., 1985). Respondents are asked to score the frequency with which their partner engaged in solicitous behaviours when the respondent was in pain. Scores range from zero to six, with higher scores reflecting greater frequency of that behaviour. The subscale is scored by calculating the response mean of the three items. Example items include (when I am in pain) ‘my partner asks me what he/she can do to help’ and ‘my partner takes over my jobs or duties’. Cronbach’s alpha for the solicitous support subscale of the MPI is reported as .78 (Kerns

et al., 1985), in this study it was estimated as .81. Together, these results indicate adequate scale reliability.

7.5.2.2.4 Social-ecological risk factor - invalidation.

Invalidation was assessed using the Illness Invalidation Inventory (3*I; Kool et al; 2010). This scale was developed to measures experiences of rejection, disbelief and lack of understanding in those with chronic pain conditions across a range of social situations including with family, partners, medical professionals, social services and in work situations. This produces a scale with 10 subscales, 'discounting' and 'lack of understanding', for each of the social situations listed above (Kool et al; 2010). A two factor structure of the 3*I was demonstrated in 927 patients with rheumatoid arthritis and fibromyalgia (Kool & Geenen, 2012). Construct validity of the scale was demonstrated by correlations between higher discounting and lack of scores with lower mental well-being and social functioning scores in 309 adults with rheumatoid arthritis and fibromyalgia (Kool et al; 2010).

The scale is scored on a scale of one (*never*) to five (*very often*). Higher scores indicate more frequent perceptions of invalidating social experiences. Three items comprise the lack of understanding subscales and five items comprise the discounting subscales. Each subscale is scored by calculating the mean score of summed items; lack of understanding items are reverse scored. Example items include 'My family makes me feel like I am an exaggerator' and 'Medical professionals give me unhelpful advice'. As the scale instructs respondents to leave blank sections that do not apply to them, it is likely to produce high levels of missing data for some subscales. The scale's author (Kool, M., personal communication, May 11, 2015) suggested that a total factor score for either discounting or lack of understanding could be calculated if at least three subscales were available for each factor. Cronbach's alphas for each subscale are all reported to be above .79

except lack of understanding by spouse, which was .67, just below the recommended threshold for reliability estimates of .70 (Kool et al; 2010). Cronbach's alphas in this study (for the subscales included in the analyses) ranged from .84 (discounting by medical professionals) to .91 (invalidation by family members), demonstrating acceptable scale reliability.

7.5.2.2.5 Stress-processing risk factor – catastrophising.

Catastrophising was measured by the Pain Catastrophising Scale (PCS; Sullivan, Bishop & Pivik, 1995). The PCS is a widely used, 13 item instrument that assesses catastrophic pain-related thinking. It consists of three subscales – rumination, magnification and helplessness. Items are scored on a five point Likert scale, from one to five, indicating how often respondents experience that thought or feeling when they have pain. Higher scores indicate more frequent catastrophising. Subscale and total scale scores are calculated by summing the relevant items with total scale scores ranging from 13 to 65. Items include 'I worry all the time about whether the pain will end' and 'It's awful and I feel that it overwhelms me'. Total scale scores were used in the current research. The PCS has been shown to discriminate between a community sample of 215 adults and a sample of 60 adults with chronic pain recruited from a pain clinic (Osman et al., 2000). This same study demonstrated acceptable criterion validity of the scale with moderate correlations reported between the PCS and measures of perceived pain severity and pain-related function. The Cronbach's alpha of the total scale is reported as .87 (Sullivan et al; 1995), in this study it was .95.

7.5.2.2.6 Stress-processing risk factor – fear-avoidance.

Fear-avoidance was assessed using the Tampa Scale for Kinesiophobia (TSK; Kori, Miller & Todd, 1990). The original 17 item version of this scale was developed to measure fear of movement and/or re-injury in chronic pain patients and its use has been validated in varying groups of pain patients including those with chronic low back pain and fibromyalgia (Goubert et al., 2004;

Vlaeyen, et al., 1995a) and osteoarthritis (Heuts et al., 2004). The 17 item TSK is reliable, with a reported Cronbach's alpha of .84 (French, France, Vigneau, French & Evans, 2007).

A shortened 11 item version of the original scale was used in this study. The psychometrics of the TSK 11 are reported to be similar to the original scale (Woby, Roach, Urmston & Watson, 2005). Discriminant and criterion validity of the TSK 11 has been demonstrated in sample of individuals with low back pain (Woby et al., 2005) and in those with general musculoskeletal pain conditions that included back pain, osteoarthritis and fibromyalgia (Roelofs et al., 2007; Tkachuk & Harris, 2012). Items are scored on a scale of one to four, to indicate the degree to which respondents agree with that fear-avoidant belief. Sample items include 'I wouldn't have this much pain if there weren't something potentially dangerous going on in my body' and 'Pain always means I have injured my body'. Items are summed to produce a total scale score; higher scores indicate higher levels of fear-avoidance beliefs. Cronbach's alphas for the TSK 11 in varying groups of pain patients have been reported to range between .76 and .80 (Roelofs et al., 2007; Tkachuk & Harris, 2012). In this study Cronbach's alpha was .84.

7.5.2.3 Resistance factors.

7.5.2.3.1 Intrapersonal resistance factor – optimism.

Optimism was assessed with the Life Orientation test – Revised (LOT-R, Scheier, Carver & Bridges, 1994). The LOT-R is widely used, brief measure of dispositional optimism. It consists of 10 items, three of which measure optimism and three, pessimism (reverse scored). The remainder items are filler items and are not scored. Respondents are asked to indicate the extent to which they agree with each item on a five point Likert scale ranging from zero (*strongly disagree*) to four (*strongly agree).* Example items include 'In uncertain times I usually expect the best'

(optimism) and ‘I hardly ever expect things to go my way’ (pessimism). Scores range from 0 to 12, with higher scores indicating a more optimistic outlook.

The scale has been shown to discriminate from other measures of mental health functioning such as neuroticism (r = -.36) and self-esteem (r = .50) (Scheier et al; 1994). Additional criterion validity of the scale was demonstrated in a sample of 128 substance-abusing adults via strong negative correlations of the LOT-R with measures of hopelessness and depression (Hirsch, Britton & Conner, 2010). Cronbach’s alpha for the scale is reported as .78 (Scheier et al; 1994), in this study it was .86.

7.5.2.3.2 Intrapersonal resistance factor - positive affect.

Positive affect was measured by Positive and Negative Affect Scale, Short Form (PANAS; Watson, Clark & Tellegen, 1988). This scale is described above in Section 7.7.2.2.4.

7.5.2.3.3 Social-ecological resistance factor - social support.

Social support was measured with the Medical Outcomes Study Social Support Survey (MOS-SS; Sherbourne & Stewart, 1991). The MOS-SS was developed from the Medical Outcomes Study (Hays, Sherbourne & Mazel, 1995) to measure perceptions of social support in community based, chronically unwell individuals. The original version of the MOS-SS is a 19 item scale that assesses social support across four domains - emotional and instrumental support, positive social interaction and affection. This version was validated in an adult sample of 2349 patients with chronic health conditions recruited from American health practices and was shown to have good convergent validity as all scale items correlated at .70 or greater with ther hypothesised subscales (Sherbourne & Stewart, 1991). Moderate to strong positive correlations of the tangible and emotional subscales of the original MOS-SS with measures of family and marital functioning, mental and general health and perceived pain intensity provided evidence of good criterion validity

of the scale (Sherbourne & Stewart, 1991). Cronbach's alphas of the original version of the MOS-SS subscales and total scale score were reported to range between .91 (affection) to .97 (overall scale) (Sherbourne & Stewart, 1991).

The short form of the MOS-SS, the mMOS-SS (Moser, Stuck, Silliman, Ganz & Clough-Gorr, 2012), was used in this study. The mMOS-SS is an eight item modified version of the original scale that measures emotional and instrumental support. It has demonstrated validity in a range of chronic illness populations including diabetes, heart disease, depression and breast cancer (Moser et al., 2012). Construct validity was demonstrated by a moderate positive correlation of the mMOS-SS with marital status and living alone (Moser et al., 2012). The mMOS-SS was also shown to correlate moderately in expected directions with other health status measures, providing further evidence of criterion validity (Moser et al., 2012). Correlations between the mMOS-SS and the original version of the MOS-SS were reported to differ minimally in the trial populations listed above (Moser et al., 2012).

The mMOS-SS asks to respondents to indicate the extent to which they perceive support is available to them if they need it on a scale of one (*none of the time*) to five (*all of the time*). Items scores are summed to produce subscale scores, with higher scores indicating higher levels of perceived support. Example items include 'To prepare your meals if you are unable to do it yourself' and 'Who understands your problems?' Cronbach's alpha for the overall mMOS-SS scale score in the chronic illness patient group was reported as .93 (Moser et al., 2012). High reliability of the two subscales was also demonstrated in the current research. Cronbach's alpha values were calculated for the instrumental and emotional subscales respectively as .95 and .91.

7.5.2.3.4 Stress-processing resistance factor - pain self-efficacy.

Pain self-efficacy was measured by the Pain Self-Efficacy Questionnaire (PSEQ; Nicholas, 2007). This 10-item inventory assesses beliefs about the confidence to accomplish activities despite pain. The PSEQ is scored by summing the item responses on a scale of zero to six, where zero indicates not at all confident and six represents complete confidence. Possible scale scores range from 0 to 60, with higher scores indicating stronger self-efficacy beliefs (Nicholas, 2007). Items include 'I can enjoy things, despite the pain' and 'I can cope with my pain in most situations'. The PSEQ has high concurrent validity, reliability and internal consistency (Nicholas, 2007). Cronbach's alpha for the PSEQ is reported as high, .92 (Nicholas, 2007), in this study it was .93.

7.5.2.3.5 Stress-processing resistance factor - pain acceptance.

Pain acceptance was measured with the Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004). The original CPAQ is a 20 item scale, consisting of two subscales. Subscale One, Activity Engagement, assesses willingness to engage in life activities despite pain. Sample items include 'I am getting on with the business of living no matter what my level of pain is'. Subscale Two, Pain Willingness, measures the degree to which respondents are willing to accept the experience of pain rather than attempt to control it. Sample items include 'I would gladly sacrifice important things in my life to control this pain better' (reverse scored). The scale asks respondents to rate the degree to which each item applies to them on a scale of zero (*never*) to six (*always*), with higher scores indicating greater pain acceptance and willingness for activity despite pain. The CPAQ has been demonstrated to be a reliable and valid measure of pain acceptance and has been widely used for research purposes (McCracken et al., 2004; Vowles et al., 2014). Cronbach's alpha for the Activities Engagement and Pain Willingness subscales in the original version of the questionnaire are reported to be .82 and .78 respectively (McCracken et al., 2004).

Subsequent to original CPAQ, a short form of the questionnaire, the CPAQ 8, used in this study, was developed (Fish, McGuire, Hogan, Morrison & Stewart, 2010). Psychometrics of the CPAQ 8 have been examined in several large samples with good psychometrics reported (Fish et al; 2010, Fish, Hogan, Morrison, Stewart & McGuire, 2013; Rovner, Årestedt, Gerdle, Börsbo & McCracken, 2013). For example, concurrent validity of the CPAQ 8 was demonstrated via moderate to strong negative correlations of the CPAQ 8 with measures of anxiety, depression and perceived pain intensity in a sample of 428 adults with chronic pain (Fish et al; 2010).

The CPAQ 8 is particularly suitable for the current purposes as it has been validated in a sample of 334 Australian adults with chronic pain (Baranoff, Hanrahan, Kapur & Connor, 2014). Concurrent validity of the CPAQ 8 was demonstrated via moderate negative correlations with measures of anxiety, depression and pain intensity (Baranoff et al., 2014). This same study reported a high correlation with the original CPAQ (r = .93). Cronbach's alphas of the CPAQ 8 are reported to be similar to the original CPAQ; for the Activities Engagement subscale reported alpha values range between .79 and .89. For the Pain Willingness subscale alpha values range between .69 and .77 (Fish et al; 2010, Fish et al., 2013; Rovner et al., 2013). Cronbach's alpha for the whole scale score of the CPAQ 8 used in the current study was .79 which demonstrates acceptable scale reliability.

7.5.2.3.6 Stress-processing resistance factor –values-based living.

Values-based living was measured with the Engaged Living Scale (ELS; Trompetter et al; 2013). The ELS is a 16 item scale that is divided into two subscales – Values Living and Life Fulfilment. Respondents are asked to indicate the extent to which they agree with statements in the scale, with items scored on a five point Likert scale ranging from one (*completely disagree*) to five (*completely agree*). Subscale scores are calculated by summing all items for that subscale with

higher scores indicative of higher level of values awareness and degree of fulfilment as a result of living in accordance with one's values. Example items include 'I have values that give my life more meaning' (Values Living) and 'I live the way I always intended to live' (Life Fulfilment).

The ELS was used in this research because, unlike other measures of values validated for use in chronic pain populations such as the Chronic Pain Values Inventory (CPVI; McCracken & Yang, 2006), the ELS assesses commitment and success in living in accordance with values generally and does not specify values according to domains (Trompetter et al; 2013). Although the CPVI has been used more broadly in the related research, it was felt the similarity of some items on the CPVI to items on the QOLS may have contributed to a high degree of variance overlap and subsequent potential problems with interpretation of results. For example, all six domains identified on the CPVI (family, intimate relations, friends, work, health and growth and learning) are also represented within the QOLS.

Concurrent validity of the ELS was demonstrated in a clinical sample of 238 adults with chronic pain as well as a non-clinical sample of 439 adults via moderate positive correlations of ELS scale scores with validated measures of acceptance, physical and mental health and psychological wellbeing (Trompetter et al; 2013). Cronbach's alphas for both subscales in the chronic pain population were reported to be .89 (Values Living), .87 (Life Fulfilment) and .91 for the total scale (Trompetter et al; 2013). In this study high scale reliability was demonstrated; Cronbach's alpha for the total scale score of the ELS was .93. Measures used in Study One (PSEQ, MPI) can be found in Appendix B. The remaining Study 3 measures can be found in Appendix F.

7.5.3 Procedure.

Participants were pain-affected individuals recruited from online pain groups, community health centres, medical and physiotherapy practices and exercise facilities such as council operated

gymnasium and pool facilities within a regional Victorian area. Online pain groups included Fibromyalgia Victoria and Chronic Pain Australia. Participants completed a series of questionnaires, either online or in hard copy. Posters were placed in health centre waiting rooms and in common areas of exercise facilities, for example on noticeboards in corridors. Paper and pencil versions of the research questionnaires were left at the points of advertisement (for example, in waiting rooms) as well as a link to the research website. The questionnaires were administered in the same order in both hard copy and online. The explanatory statement was available on the website as a downloadable document. For those completing the research in hard copy, the explanatory statement was attached to the questionnaires. Completed copies of the paper and pencil version of the questionnaire were posted back to a post office box address. A stamped addressed envelope was provided with the paper and pencil version of the survey. Consent to participate was implied from completion of the questionnaire.

7.5.3.1 Ethics approval.

Ethics approval was obtained from Monash University and Barwon Health Human Research Ethics Committees. Barwon Health Ethics approval was required because the community health centres that acted as recruitment sites for the research were part of Barwon Health. Copies of the ethics approval certificates can be found in Appendix A.

7.5.4 Statistical analyses.

This section outlines the analytic approach. All data were analysed using the Statistical Package for the Social Sciences, Versions 20.0 and 24.0 (SPSS; IBM Corp., 2010 & 2015). The risk only and risk-resistance models were explored using multiple hierarchical regression. Indirect effects were assessed with ordinary least squares regression, using PROCESS (Hayes, 2013), the same SPSS macro that was used in Study One to calculate indirect effects. Data cleaning and

preparation processes are described including the process used to replace missing values. This is followed by a description of the analytic approach used to investigate the study hypotheses. In summary, the same steps as those used in Study One were used to address the research hypotheses. These are outlined below:

1. Data were screened for missing values, these were replaced using the Expectation-Maximisation algorithm (Section 7.5.4.1).
2. Variable distributions were assessed. Required assumptions for correlational, *t*-tests and multiple regression analyses were checked (Section 7.5.4.2).
3. Power calculations were completed to ensure adequate sample size (Section 7.5.4.3).
4. Scale reliability was assessed (Sections 7.5.2.1, 7.5.2.2 and 7.5.2.3).
5. Single sample t-tests were used to compare the Study Three data to normative pain clinic data (Section 7.5.4.4)
6. Pearson correlation co-efficients were calculated to assess relationships between risk and resistance factors with pain-related disability and QOL (Section 7.5.4.5).
7. Hierarchical linear regression was used to assess the proportion of variance in pain-related disability and QOL explained by the risk-resistance model that was over and above that explained by the risk only model (Section 7.5.4.6).
8. Indirect effects were assessed with ordinary least squares regression using PROCESS (Hayes, 2013), a macro for SPSS that calculates these effects (Section 7.5.4.7).

7.5.4.1 Data screening – missing data, univariate distributions and assumptions.

Prior to assessing missing data, the data were screened for errors and negatively worded items were reversed scored. Missing data patterns were assessed using frequency tables, graphs and Little's Missing Completely at Random (MCAR) test (Little, 1988). This test indicates whether

missing data are missing at random or in a systematic pattern. A statistically significant result is indicative of data that are not missing at random. Missing data were replaced using the Expectation-Maximisation algorithm (EM). As explained in Chapter Three, Section 3.5.4.1, the EM algorithm uses the means, variances, and co-variances of the available data to calculate replacement values and is as an accurate and robust procedure for replacing values that are missing at random (Graham, 2009).

Of all the respondents to the online survey, only complete surveys were used in the analysis. Many of the online surveys that were commenced had only a small number of items completed. These surveys were discarded. Of the 304 total number of surveys commenced, 223 were complete and were retained for analysis. Of the 59 hard copy surveys received, only one was incomplete. This survey was not included. The majority of scales had less than 5% missing data. The only exceptions to this noted were the sexual activity item of the PDI (5.4%), several items on the QOLS pertaining to satisfaction with job (6.1%), partner (6.8%) and children (8.1%). These data were not missing at random according to Little's MCAR test, but because the proportions missing were only marginally more than 5% they were replaced using the EM algorithm. On average, across all items of the QOLS only 1.61% of total data points were missing.

Additionally, some subscales of the 3*I (Kool et al; 2010) contained large amounts of missing data. The 3*I instructs participants to leave subscales blank if they are not applicable to them. Proportions of missing data for the subscales assessing invalidation and discounting by spouses, in work environments and by social services were high, ranging between 28% and 59%. These scales were omitted from the analyses. The proportion of missing data across all items on the remaining subscales of the 3*I (discounting and invalidation by medical professionals and family members) ranged between .12% (lack of understanding by medical professionals) to 3.6%

(discounting by family members). Because these proportions were less than 5%, these values were replaced using the EM algorithm. The only other statistically significant result of Little's test (Little, 1988) was noted for the instrumental support subscale of the MOS (Moser et al., 2012). However, as the proportion of missing values on items of this subscale was less than 5% these values were also replaced using the EM algorithm.

Following imputation, univariate distributions of all variables were examined to ensure the assumption of normality was not violated. Univariate outliers were assessed by inspection of frequency tables and box plots. Few outliers were identified as a result of the forced response style of the online questionnaires. One extreme outlier was identified in the years of education (27 years, variable mean 5.06 years). This value was replaced with the variable mean, as is suggested by Tabachnik and Fidell (2013). Skewness and kurtosis values, together with Shapiro-Wilk and Kolmogorov Smirnoff univariate normality statistics were assessed. Plots, including histograms with normality plots, box plots and detrended normal quantile-quantile (Q-Q) plots were also inspected to ensure normality. Visual inspection of histograms revealed substantively normal distributions. After removal of the single outlier in years of education, all kurtosis and skewness values fell within the recommended range of +/- 1.5 (Tabachnik & Fidell, 2013, see Table 3, Appendix G).

Non-statistically significant Kolmogorov-Smirnov and Shapiro-Wilk test statistics are indicative of normal distributions. However, significant values of these statistics are not uncommon in larger sample sizes of greater than 200 and inspection of box plots and histograms has been recommended as a better assessment of distribution (Pallant, 2010). Non-statistically significant Kolmogorov-Smirnov test statistics were noted for the pain self-efficacy, the pain acceptance and the QOL scales, indicating normal distributions of these variables. Non-statistically significant

Shapiro-Wilk test statistics were also noted for mean pain scores, the fear-avoidance, the pain acceptance and the QOL scales also indicating normal distributions. In addition to these tests, inspection of histograms with normality plots, the 5% trimmed mean values, box plots and Q-Q plots demonstrated largely normal distributions of all variables. Skewness and kurtosis values and trimmed mean values of all measures can be found in Appendix G.

7.5.4.2 Data screening – univariate and multivariate assumptions.

The assumption of equality of variance was met for most *t*-tests and *ANOVA*s. Where this assumption was not met, results were reported with the appropriate test statistic and the caveat 'equal variances not assumed'. For all correlations, bivariate scatterplots of the relationships were inspected to ensure linearity and homoscedasticity. These assumptions were met. Examination of the normal probability plots of the standardised residuals and the scatterplot of the standardised residuals in both hierarchical regression analyses indicated that the assumptions of linearity, homoscedasticity and independence of residuals were not violated.

The multiple regression assumptions of multicollinearity and singularity were checked in SPSS 20.0 (IBM Corp., 2012) by three methods. Firstly, correlation coefficients were checked to ensure that these did not exceed the recommended levels for multicollinearity of $r = .90$ (Tabachnik & Fidell, 2013). Because the correlations remained below this threshold, these variables were retained in their original form in the analyses. Multicollinearity was further assessed by examining variance inflation factors (VIFs) for the hierarchical regression equations. In support of the decision to retain all measures as separate variables in initial analyses, VIFs for all measures were less than 3.3 in both initial hierarchical regression analyses, below the recommended threshold of 10 (Hair et al., 2010; Tabachnik & Fidell, 2013). Lastly, standard errors of the regression co-efficients were

checked to ensure they were not excessively large, as standard errors can become inflated when issues with multicollinearity occur (Tabachnik & Fidell, 2013).

As in Study One, the significance level of statistical tests, the alpha level, was set at .05 unless otherwise specified. Given multiple tests were used, a Bonferroni adjustment of the alpha level could have been conducted however this was not deemed necessary because all analyses were specified a priori. It has been also been suggested that Bonferroni adjustments should not be used even in the setting of multiple tests because the Type 1 error rate cannot be decreased without increasing the Type II error rate, which is equally as incorrect (Perneger, 1998). As a way of increasing confidence in the reliability of estimates of indirect effects, bootstrapped confidence intervals were used in place of alpha levels.

It is important in multiple regression to ensure that any multivariate outliers in the dataset are not exerting an influence on results (Tabachnik & Fidell, 2013). Therefore, Mahalanobis distances were calculated. The critical value for Mahalanobis distance for the risk-resistance regression analysis predicting pain-related disability was 29.59 as indicated by the Chi square critical values table for 10 predictors at the level of .001 (Tabachnik & Fidell, 2013). One case (35.41) was noted to be above this value. In relation to the regression analysis predicting QOL, the critical value for Mahalanobis distance was 31.26 as indicated by the Chi square critical vales table for 11 predictors at the level of .001 (Tabachnik & Fidell, 2013). No cases were above this value. Cook's distances were also checked to assess multivariate outliers. Tabachnik and Fidell (2013) suggest that cases with values greater than one represent multivariate outliers. No values were noted greater than .08 in either analysis.

To assess the impact of the single multivariate outlier on Mahalanobis distance for the risk-resistance model regressing pain-related disability, this single case was removed and the analysis repeated. Removal of this outlier resulted in the regression co-efficient of pain acceptance changing

from being significant at the level of .05 to being non-statistically significant. Because of this influence, this outlying case was removed as is recommended by Tabachnik and Fidell (2013). The results reported in Chapter Eight are therefore those obtained from the data that did not include the outlier. Multivariate outliers for the risk-resistance models are listed in Appendix G.

Multivariate outliers were also checked for the analyses of indirect effects using ordinary least squares regression and the PROCESS macro (Hayes, 2013). All relevant variables for each model were entered into linear regression analyses and Mahalanobis distances checked. A single multivariate outlier was identified in each of these models. To ensure this outlier did not influence results, these analyses were examined in a dataset that had this single case deleted. Results were unchanged.

7.5.4.3 Power calculations.

Power calculations were made in order to ensure a reasonable likelihood of detecting a given effect size (Tabachnik & Fidell, 2013) and as in Study One, these were completed using GPOWER (Faul et al., 2007). The ability to detect moderate to large effect sizes (between .15 and .35) in a multiple regression analysis with 11 predictors at the level of .95 power was estimated as requiring a sample size of between 83 and 178 participants. Therefore the current sample size of 281 participants can be assumed to provide sufficient power for multiple regression analyses.

7.5.4.4 Comparison of the current sample to Study One and normative data.

To explore differences between the Study One and Study Three samples, mean age, levels of pain self-efficacy and partner responses to pain were compared using t-tests. These measures were the same in both studies. In order to place the current results in the context of the existing literature, age, pain duration and reported disability levels Study Three data were compared to normative pain clinic data using single sample t-tests.

7.5.4.5 Relationships between predictors and pain-related disability and quality of life.

Hypothesis One proposed that higher levels of risk factors (perceived pain intensity, catastrophising, fear-avoidance, NA, punishing and solicitous partner responses to pain and invalidation), would be associated with greater disability and lower QOL. Hypothesis Two proposed that higher levels of resistance factors (pain self-efficacy, pain acceptance, values-based living, positive affect, optimism and social support), would be associated with less disability and greater perceived QOL. These two hypotheses were examined by using Pearson's product-moment correlation coefficients using total scale scores. As in Study One, the guidelines adopted for interpretation of the strength of correlation co-efficients were those provided by Cohen (1988), which suggest that small correlations are those ranging between .10 to 29, medium correlations range between .30 and .49 and large correlations are those above .50. Variables that demonstrated statistically significant correlation coefficients of at least moderate magnitude ($r \geq .30$, Cohen, 1988) were retained for further analysis of direct and indirect effects using both hierarchical multiple regression and ordinary least squares regression using PROCESS, a macro for SPSS (Hayes, 2013).

7.5.4.6 Risk only and risk-resistance models.

Hypothesis Three proposed that the resistance factors pain self-efficacy, pain acceptance, values-based living, positive affect, optimism and social support, would account for additional variance, over and above that explained by the risk factors (perceived pain intensity, catastrophising, fear-avoidance, NA, punishing and solicitous partner responses to pain and invalidation), in pain-related disability and QOL. A hierarchical regression model was specified such that demographic predictors were entered in Step One, followed by risk predictors in Step Two and resistance predictors in Step Three. The amount of variance explained by both the risk

only and the risk-resistance model was assessed. Statistical significance of individual predictors was examined. Only those risk and resistance factors demonstrating a moderate or greater correlation with either adjustment outcome were entered in the regression models.

7.5.4.7 Testing indirect effects.

As in Study One, all proposed indirect effects were tested with Hayes' (2013) PROCESS macro for SPSS. PROCESS uses an ordinary least squares regression-based path analytic approach to estimate direct and indirect effects. Single and multiple mediator models can be assessed. In this research, 10,000 bootstrap iterations and 95% bias-corrected confidence intervals were used. For a full discussion about the use of bootstrap confidence intervals for estimating indirect effects, please see Section 3.7.4.7 in Chapter Three. Results were interpreted as statistically significant if the bias-corrected confidence intervals did not cross or include zero (Hayes, 2013).

As in Study One, mediator and moderator effects were only assessed for relationships that correlated between a predictor and outcome variable at moderate or greater strength ($r \geq .30$) as this indicated that a relationship existed to be mediated or moderated (Holmbeck, 2004). Unstandardised effect sizes are reported in relation to mediation effects. For a full discussion regarding this issue, please see Section 3.5.4.7 in Chapter Three. As outlined in this section, magnitude of effects sizes were interpreted according to guidelines provided by Cohen (1988), in which effect sizes of .01, .09, and .25 are defined as small, medium, and large respectively.

7.5.4.7.1 Mediation effects.

Hypothesis Four proposed that five pain appraisal processes, catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living, would mediate the relationships between condition parameters, intrapersonal and social-ecological factors (perceived pain severity, NA, solicitous and punishing partner responses to pain, invalidation, positive affect,

optimism and social support) with pain-related disability and QOL. As explained in Section 3.5.4.7 in Chapter Three, a mediator can be seen as a mechanism through which a predictor influences an outcome (Rose et al., 2004). As in Study One, the current study used the PROCESS macro Model Four to test mediation effects first in single mediator models. Then, where significant mediation effects were identified, these were examined in parallel mediator models. For a full discussion of single and parallel mediator models please see Section 3.5.4.7 in Chapter Three. In summary, single mediator models calculate the indirect effect of a single predictor on an outcome via a third variable, the mediator (Hayes, 2013). In parallel mediator models, the relative indirect effect of each mediator is adjusted for the effects of other mediators in the model. That is, the predictor variable is hypothesised to exert its effect on the outcome partly via at least two mediators (Hayes, 2013).

As outlined in Section 3.5.4.7 in Chapter Three, PROCESS facilitates formal testing of differences between specific indirect effects. As explained in Section 3.5.4.7 in Chapter Three, PROCESS provides confidence intervals for pairwise comparisons of specific indirect effects, thereby enabling comparison of statistical differences between mediation effects (Hayes, 2013). Conclusions about the relative strength of the compared indirect effects is possible when the point estimates of the two indirect effects are of the same sign (Hayes, 2013).

To test Hypothesis Four, separate parallel mediator models were specified for each predictor for each outcome, pain-related disability and QOL. PROCESS calculates a number of different effects within parallel mediator models. These include the direct effect (c') which refers to the relationship between the predictor and outcome after controlling for the mediators in the model. Specific indirect effects (for example a_1b_1) are also calculated which refer to the role of a specific mediator in the relationship between the predictor and outcome. As in Study One, unstandardised effect sizes are reported in the current Study. For a full discussion of reporting of

effect sizes in mediation analyses see Section 3.5.4.7 in Chapter Three. A pictorial representation of single and parallel mediator models are shown below in Figures 7.4 and 7.5.

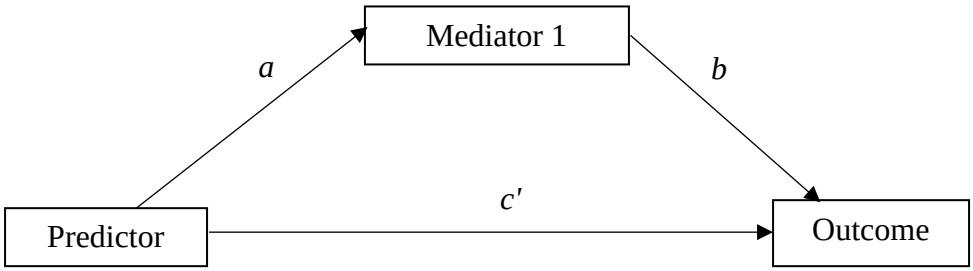

Figure 7.4 Simple mediator model.

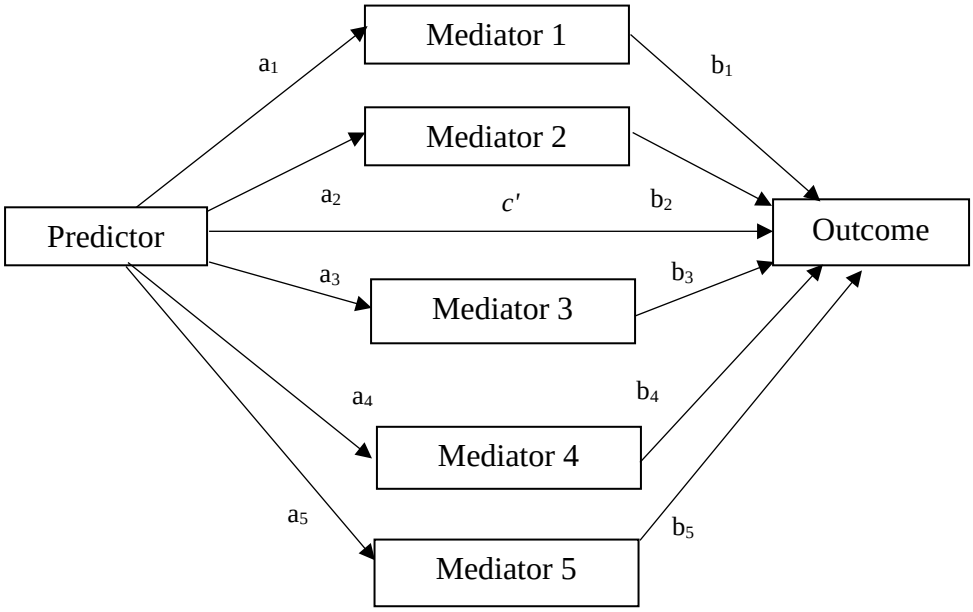

Figure 7.5 Parallel mediator model showing five potential mediators.

7.5.4.7.2 Moderation effects.

Addressing the fifth hypothesis, the potential moderating effects of the resistance factors (pain self-efficacy, pain acceptance, values-based living, optimism, positive affect and social support) on the relationships between risk predictors and pain-related disability and QOL were also assessed using ordinary least paths regression and the PROCESS macro (Hayes, 2013). PROCESS calculates two way interactions in moderation models. Specifically, PROCESS Model 1 was used to test moderation effects. The estimation of moderation effects is described more fully in Section 3.5.4.7 in Chapter Three. As in mediation testing, bootstrapping is also used within PROCESS to calculate confidence intervals for moderation effects. In this research, 10,000 bootstrap iterations were used to calculate 95% bias-corrected confidence intervals.

Where significant interactions were demonstrated post-hoc probing identified the level of the moderator that interacted with risk predictor to affect its relationship with disability or QOL. PROCESS was used to conduct post-hoc probing of significant interactions firstly, by plotting simple slopes of the relationship between the predictor and outcome at three levels of the moderator (Aitken & West, 1991). Secondly, a regions of significance analysis was conducted with PROCESS using the Johnson-Neyman technique (Johnson & Neyman, 1936). This technique calculates the specific values of the moderator at which the relationship between the predictor and outcome variable becomes statistically significant.

7.6 Threats to validity

As in Study One, various potential validity threats were considered. These included internal validity threats such as effects of missing data and biased effects estimates. Threats to external validity were also considered, such as common method bias and adequacy of sample size. Methods for addressing these potential threats are described below.

7.6.1 Effects of missing data.

The proportion of missing data was generally low. The only exceptions to this were some subscales of the 3*I, which instructed participants to leave blank the sections that did not apply to them. The subscales were omitted from the analyses. Several items of the QOLS (Burckhardt et al., 1989) and one item of the PDI (Tait et al., 1990) also had proportions of missing data that were marginally greater than 5%. Because missing data across the whole of these scales was low and the proportions were only slightly greater than 5%, they were replaced using the EM algorithm. This is a robust procedure to deal with missing values (Graham, 2009). Thus, any effects of missing data on the analyses were considered to be negligible.

7.6.2 Biased effects estimates.

Bootstrapped bias-corrected confidence intervals were calculated for all indirect effects to improve reliability of estimates of indirect effects. None of the assumptions for regression were violated and apart from the risk-resistance model predicting pain-related disability, results were the same in datasets containing the small number of multivariate outliers as those that did not contain multivariate outliers. This latter analysis therefore omitted the multivariate outlier. The parameter estimates in the analyses were thus considered reliable.

7.6.3 Common method bias.

Common method bias (CMB) can bias results of research that is conducted using a single method of assessment, for example a paper and pencil or online questionnaire. A more extensive discussion of CMB can be found in Section 3.9.3 in Chapter Three. As in Study One, the possibility that CMB may have biased estimates in the current research is acknowledged but due to existing controversy about appropriate assessment and correction techniques, it was not specifically assessed. Results of Study Three are presented in Chapter Eight.

Chapter Eight – Study Three Results

8.1 Overview

Chapter Eight presents Study Three results. Study Three aimed to add to the chronic pain literature by using Wallander and Varni's risk-resistance model (Wallander et al., 1989; Wallander & Varni, 1998) as a framework to statistically explore the direct, mediating and moderating effects of a range of risk and resistance factors on two chronic pain adjustment outcomes, pain-related disability and quality of life (QOL) in a community-based sample of adults living with chronic pain. Descriptive statistics are reported, followed by results of each of the study hypotheses.

8.2 Descriptive Statistics and Adjustment Profile

Range, means and standard deviations of scale scores by gender can be seen in Table 8.1. Several significant gender differences were noted. Males reported significantly higher scores for fear-avoidance and instrumental social support compared to females but reported significantly lower levels of values-based living and discounting by family.

8.2.1 Covariates of pain-related disability and quality of life.

Potential covariates were explored using correlational analyses, *t*-tests and one way *ANOVAS*. No significant differences were evident between mean scores on the disability measures for men (M = 43.38, SD = 15.76) compared to women (M = 41.85, SD = 14.98), t (278) = .69, p = .49. Neither were gender differences apparent in perceived QOL [Men (M = 62.02, SD = 18.79), women (M = 64.36, SD = 17.77)], t (278) = -.89, p = .38. Years of education was significantly negatively correlated with pain-related disability at weak magnitude (r [281] = -.14, p =.02) but was not significantly associated with QOL (r [281] = .11, p = .06). Longer pain duration was not associated with pain-related disability (r [281] = -.10, p =.10) but was positively associated at weak magnitude with QOL (r [281] = .17, p = .01). Increased age was weakly associated with lower

levels of pain-related disability (r [281] = -.24, p = .00) and moderately associated with higher QOL (r [281] = .30, p = .00). Six people reported their pain condition was or had been compensable under the Victorian Traffic Accident Commission (TAC) and 39 people reported their pain was or had been compensable under the Victorian WorkSafe Authority. Due to the small number of people reporting compensability under the TAC, two groups were created for the purpose of comparison for covariates; compensable or non-compensable. A *t*-test showed that people whose pain was currently or had previously been compensable reported significantly higher disability scores (M = 47.21, SD = 13.89) than did those whose pain had never been compensable (M = 41.23, SD = 15.21) t (278) = 2.45, p = .02, eta squared = .02. Similarly, a *t*-test showed that people who reported their pain was currently or had previously been compensable reported significantly lower QOL scores (M = 57.57, SD = 17.66) than did those whose pain had never been compensable (M = 65.07, SD = 17.82) t (278) = -2.59, p = .01, eta squared = .02. The correlations and effect sizes of most potential covariates except age were small, therefore only age was included in analyses as a covariate.

Table 8.1

Means, Standard Deviations of Study Variables Comparing Males to Females

	Mean (SD)		Range	*t* (p)
	Male (N=59)	Female (N=222)		
Covariates				
Age (years)	56.34 (14.57)	48.51 (13.76)	18 - 87	3.84 (.00)***
Years Education (after Year 9)	4.67 (3.71)	5.16 (3.06)	0 - 27	-1.06 (.29)
Years Pain Duration	11.77 (12.65)	12.30 (10.57)	1 - 69	-.29 (.77)
Adjustment measures				
Pain-Related Disability	43.38 (15.76)	41.92 (14.98)	0 - 69	.66 (.51)
Quality of Life	62.02 (18.79)	64.33 (17.73)	18- 112	-.88 (.38)
Risk factors				
Condition parameters				
Mean Pain Severity	5.40 (1.63)	5.39 (1.77)	.75 - 10	.04 (.97)
Stress-processing factor				
Catastrophising	37.55 (14.29)	35.15 (11.80)	13 - 65	1.68 (.10)
Fear of movement	28.51 (5.40)	25.54 (5.84)	11 - 43	3.53 (.00) ***
Intrapersonal factors				
Negative Affect	12.76 (5.49)	12.13 (4.46)	5 - 25	.82 (.41)
Social-ecological factors				
Solicitous partner responses	3.07 (1.11)	3.23 (1.03)	0 - 5	-1.03 (.31)
Punishing partner responses	1.77 (1.12)	1.85 (1.12)	0 - 5	-.50 (.62)
Lack of understanding - medical	2.20 (.86)	2.39 (.95)	1 - 5	-1.41 (.16)
Lack of understanding - family	2.55 (1.00)	2.83 (1.12)	1 - 5	-1.75 (.08)
Discounting - medical	2.09 (.88)	2.25 (.93)	1 - 5	-1.23 (.22)
Discounting - family	2.27 (1.10)	2.62 (.93)	1 - 5	-2.46 (.02)*
Resistance Factors				
Appraisal and coping factors				
Pain Self-Efficacy	30.10 (11.86)	31.32 (11.34)	10 - 58	.74 (.46)
Pain Acceptance	19.78 (8.38)	21.22 (7.55)	3 - 41	-1.27 (.20)
Values-Based Living	35.19 (6.84)	37.61 (6.29)	22 – 80	-1.91 (.06)
Intrapersonal factors				
Positive affect	15.10 (4.42)	15.25 (3.65)	5 - 23	-.27 (.79)
Optimism	5.60 (3.04)	6.25 (2.67)	0 - 12	-1.62 (.11)
Social-ecological factors				
Emotional social support	14.00 (4.59)	13.24 (4.14)	4 - 20	1.23 (.22)
Instrumental social support	14.23 (5.36)	12.67 (4.93)	4 - 20	2.13 (.03)*

Note. ***$p < .001$, **$p < .01$, *$p < .05$

8.2.2 Comparison of Study One sample to Study Three sample.

In order to consider results in light of differences between the Study One and Study Three samples, variables that were assessed by the same measures were compared using independent sample *t*-tests. Mean ages and perceived levels of partner responses to pain of the two samples did not differ but Study Three participants reported significantly higher pain self-efficacy levels compared to Study One participants. Results can be seen in Table 8.2.

Table 8.2

Means, Standard Deviations of Study Variables Comparing Study One to Study Three

	Mean (SD)		*t* (p)
	Study 1 (N=330)	Study 3 (N=281)	
Age	51.54 (13.50)	50.24 (14.22)	1.17 (.24)
Pain self-efficacy	21.62 (13.94)	31.08 (11.46)	-9.35 (.00)
Punishing partner responses	2.02 (1.47)	1.84 (1.12)	1.60 (.11)
Solicitous partner responses	3.39 (1.50)	3.19 (1.04)	1.93 (.05)

Table 8.3

Comparison of Study 3 Adjustment Profile to Normative Pain Clinic Data

	M (SD) normative data (n=6124)	*M* (SD) Study 3 (n=281)	*t* (p)
Age	43.4 (13.1)	52.24 (14.22)	7.93 (.00)
Pain Duration (Years)	4.79 (7.23)	12.04 (11.61)	11.23 (.00)
Pain-Related Disability	44.6 (13.4)	42.18 (15.13)	-2.63 (.01)

Note. ***** Normative data obtained from Chibnall & Tait (1994)

Normative data for community-based samples of individuals with chronic pain is not available, however, two sources of reference data for the PDI as a measure of pain-related disability are available (Chibnall & Tait, 1994; Soer et al., 2015). These data were based on a large (*n* = 6997) sample of Canadian and Dutch patients seeking treatment at pain clinics for chronic musculoskeletal pain. Diagnoses of the reference group included non-specific back pain,

discogenic back pain, soft tissue pain such as fibromyalgia and whiplash injuries. Thus, the diagnostic profile of the two groups was similar. Participants in Study Three reported a significantly higher disability score (M = 42.18, SD = 15.13) compared to the normative data for the PDI (M = 37.8, SD = 14.2), t (279) = 4.84, p = .00.

8.3 Study Hypotheses

8.3.1 Hypothesis one – relationships between risk factors and adjustment.

Hypothesis One that risk factors would be associated with higher levels of pain-related disability and lower QOL was largely supported. These relationships were assessed using bivariate Pearson-product moment correlations. As hypothesised, moderate positive correlations were observed between risk intrapersonal and stress-processing factors (catastrophising, fear-avoidance and negative affect) with pain-related disability. Moderate negative associations were noted between these same factors and QOL. Social-ecological risk factors (solicitous and punishing partner responses to pain and invalidation by family members and medical professionals) were negatively associated with pain-related disability and positively associated with QOL all at weak strength. Correlation co-efficients between risk factors and adjustment measures can be seen in Table 8.4.

Table 8.4

Bivariate Correlation Matrix of Risk Factors and Outcome Measures

	1	2	3	4	5	6	7	8	9	10	11	12
1. Pain-Related Disability	1											
2. Quality of Life	-.63**	1										
3. Mean pain	.57**	-.48**	1									
4. Catastrophising	.45**	-.45**	.36**	1								
5. Fear-Avoidance	.34**	-.28**	.27**	.61**	1							
6. Negative Affect	.47**	-.59**	.39**	.63**	.39**	1						
7. Punishing Responses	.20**	-.23**	.18**	.33**	.23**	.34**	1					
8. Solicitous Responses	.11	.07	-.03	.06	.10	-.06	-.28**	1				
9. Lack Family Understanding	.09	-.18*	.14*	.15*	.05	.17*	.17**	-.17**	1			
10. Family Discounting	16**	-.22**	.16**	.23**	.12*	.25**	.29**	-.12*	.76**	1		
11. Lack Medical Understanding	.14*	-.18**	.16**	.13	.10	.14*	.16**	-.15**	.44**	.42**	1	
12. Medical Discounting	.24**	-.23**	.19**	.23**	.16*	.25**	.24**	-.06	.32**	.44**	.74**	1

Note. * $p < .05$, ** $p < .01$

8.3.2 Hypothesis two – relationships between resistance factors and adjustment.

Hypothesis Two that resistance factors would be associated with lower pain-related disability and higher QOL was also largely supported. Strong relationships in the hypothesised directions were noted between most resistance factors and pain-related disability and QOL. The social-ecological resistance factors (emotional and instrumental social support) were negatively associated with pain-related disability at weak strength. However, these same factors were positively associated with QOL at moderate strength. Correlation co-efficients between resistance factors and adjustment measures can be seen in Table 8.5.

Table 8.5

Bivariate Correlation Matrix of Resistance Factors and Outcome Measures

	1	2	3	4	5	6	7	8	9
1. Pain-Related Disability	1								
2. QOL	-.63**	1							
3. Pain Self-efficacy	-.74**	.74**	1						
4. Pain Acceptance	-.63**	.65**	.76**	1					
5. Optimism	-.24**	.34**	.42**	.37**	1				
6. Positive Affect	-.44**	.53**	.55**	.49**	.34**	1			
7. Values-Based Living	-.49**	.56**	.54**	.47**	.44**	.50**	1		
8. Instrumental. Social Support	-.15*	.30**	.20**	.19**	.17**	.20**	.23**	1	
9. Emotional Social Support	-.26**	.48**	.39**	.28**	.33**	.29**	.41**	.72**	1

Note. * $p < .05$, ** $p < .01$

Moderate strength negative associations were also noted between perceived pain severity, negative affect, catastrophising and fear-avoidance with most intrapersonal and stress-processing resistance factors. The exceptions only to this were the associations between pain and fear-avoidance with positive affect which were weak and negative $r(281) = -.28$, $p < .01$ and -.29, $p < .01$ respectively and the association between pain and optimism which was also weak and negative $r(281) = -.08$, ns.

8.3.3 Hypothesis three – risk-only model compared to risk-resistance model predicting pain-related disability.

Hierarchical multiple regression was employed to test whether the resistance factors would account for additional variance in pain-related disability, over and above that explained by the risk factors. Only those variables that demonstrated correlation coefficients of at least moderate magnitude ($r \geq .30$, Cohen, 1988) were entered in the initial multiple regression analysis. In the model predicting pain-related disability, age was entered as a covariate in Step 1 (Model 1). Risk factors were entered in Step 2 (Model 2). Finally, resistance factors were entered in Step 3 (Model 3). As shown in Table 8.6, after adjusting for age, the combined risk and resistance factors

accounted for a significant 62% of the variance in pain-related disability (F (9, 270) = 48.91, p < .001). The addition of the resistance factors accounted for a significant 19% of the variance in pain-related disability over and above that accounted for by the risk factors, ΔR^2 = .19, ΔF (4, 270) = 34.03, p < .001. Only perceived pain severity and pain self-efficacy made significant unique contributions to pain-related disability in the final equation. The direction of the association between fear-avoidance and values-based living with pain-related disability in the final risk-resistance model was in the opposite direction to that which was hypothesised, although the magnitude of these regression weights was very small.

Table 8.6

Hierarchical Multiple Regression of Pain-related Disability on Risk and Resistance Factors

	B (SE)	R_2	$\triangle R_2$	F	$\triangle F$
Step 1		.06	.06	17.35***	17.35***
Age	-.05 (.04)				
Step 2		.43	.37	41.02***	44.25***
Pain severity	.28 (.38)***				
Fear-avoidance	-.03 (.13)				
Catastrophising	.00 (.07)				
Negative Affect	.06 (.18)				
Step 3		.62	.19	48.91***	34.03***
Pain self-efficacy	- .51 (.09)***				
Pain acceptance	-.12 (.13)				
Positive affect	-.01 (.20)				
Values-based living	.06 (.08)				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The standardised regression weights for risk factors shown above are those from Model 3, the risk-resistance model.

The reversed regression co-efficients were suggestive of potential issues with multicollinearity (Ludlow & Klein, 2014). Therefore, model simplification was undertaken to explore these potential effects. Each of the predictors were removed and replaced iteratively in the model. Catastrophising, fear-avoidance and negative affect remained non-significant risk factors in all models, even those in which pain severity was the only other risk factor. Pain self-efficacy was a significant predictor of disability in all models. However, whenever models included pain self-efficacy, the sign of the regression weight for values-based living was reversed. This may have been due to suppressor effects or an interaction between these variables, however it may also have been because of multicollinearity (Ludlow & Klein, 2014) as pain self-efficacy was strongly correlated with disability and pain acceptance. The variance inflation factor (VIF) for pain acceptance in the initial model was 2.92 and for pain self-efficacy was 3.15. Although these values were well below the recommended threshold of 10 (Tabachnik & Fidell, 2013) and even below other more conservative thresholds such as five (Rogerson, 2001) and four (Pan & Jackson, 2008), alternative models were still explored to investigate potential multicollinearity effects in the model. As the simultaneous effects of positive and negative social support were of interest in the research and because solicitous partner responses was a significant predictor of disability in the Study One risk-resistance model, different model iterations were explored that also included the social-ecological risk and resistance factors, despite that these were only weakly associated with disability at a bivariate level.

The model explaining the largest proportion of variance with regression co-efficients that were all in the hypothesised directions was a model in which pain severity and solicitous partner responses were significant risk predictors and pain acceptance was the only significant resistance predictor. In this model, age was entered as a covariate in Step 1 (Model 1). Risk factors were

entered in Step 2 (Model 2) and resistance factors were entered in Step 3 (Model 3). As shown in Table 8.7, after adjusting for age, the combined risk and resistance factors accounted for a significant 55% of the variance in pain-related disability (F (10, 269) = 32.87, p < .001). The addition of the resistance factors accounted for a significant 12% of the variance in pain-related disability over and above that accounted for by the risk factors, ΔR^2 = .12, ΔF (5, 269) = 14.22, p < .001.

Table 8.7

Hierarchical Multiple Regression of Pain-related Disability on Risk and Resistance Factors – Revised Model

	B (SE)	R_2	$\triangle R_2$	F	$\triangle F$
Step 1		.06	.06	17.35***	17.35***
Age	-.04 (.05)				
Step 2		.43	.37	41.52***	44.82***
Pain severity	.35 (.40)***				
Negative Affect	.04 (.19)				
Solicitous Partner Responses	.12 (.72)*				
Punishing Partner Responses	.07 (.63)				
Step 3		.55	.12	32.87***	14.22***
Pain acceptance	-.35 (.11)***				
Positive affect	-.09 (.21)				
Values-based living	-.06 (.07)				
Instrumental support	-.01 (.19)				
Emotional support	-.03 (.25)				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The standardised regression weights for risk factors shown above are those from Model 3, the risk-resistance model.

When all the non-significant predictors were removed from the model seen in Table 8.7, after adjusting for age, only pain severity and pain acceptance were significant predictors of pain-related disability, explaining 52% of the variance (F (10, 269) = 32.87, $p < .001$). Pain acceptance contributed an additional 17% unique variance to the prediction of disability, over and above that explained by pain intensity, $\Delta R^2 = .18$, ΔF (5, 269) = 14.22, $p < .001$. This final model, seen in Table 8.8, was similar to the initial model in which pain severity and pain self-efficacy were the only significant predictors of disability. Social support was not a significant predictor of disability in any model.

Table 8.8

Hierarchical Multiple Regression of Pain-related Disability on Risk and Resistance Factors – Revised Model

	B (SE)	R_2	$\triangle R_2$	F	$\triangle F$
Step 1		.06	.06	17.35***	17.35***
Age	-.07 (.05)				
Step 2		.35	.29	73.89***	122.83***
Pain severity	.38 (.40)***				
Step 3		.52	.18	100.98***	101.53***
Pain acceptance	-.46 (.09)***				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The standardised regression weights for risk factors shown above are those from Model 3, the risk-resistance model.

8.3.4 Hypothesis three – risk-only model compared to risk-resistance model predicting quality of life.

Hierarchical multiple regression was also employed to test whether the resistance factors would account for additional variance in QOL, over and above that explained by the risk factors. Age was entered in Step One (Model One), risk factors were entered in Step Two (Model Two) and resistance factors were entered in Step Three (Model Three). Hypothesis Three was supported.

As shown in Table 8.9, after adjusting for age, the combined risk and resistance factors accounted for a significant 70% of the variance in QOL (F (11, 268) = 56.53, $p < .001$). The addition of the resistance factors accounted for a significant 26% of the variance in QOL over and above that accounted for by the risk factors, $\Delta R^2 = .26$, ΔF (7, 268) = 32.70, $p < .001$. In Model 3, perceived pain severity, catastrophising, negative affect, pain self-efficacy, pain acceptance, values-based living and emotional social support all made significant unique contributions to QOL. However, the direction of the regression coefficients for catastrophising, optimism and instrumental social support with QOL in this model were all in the opposite direction to what was hypothesised. Despite that catastrophising had a moderate negative correlation with QOL, once the other risk and resistance factors were taken into account, higher catastrophising scores were a significant predictor of better QOL.

Table 8.9

Hierarchical Multiple Regression of Quality of Life on Risk and Resistance Factors

	B (SE)	R_2	△ R_2	F	△ F
Step 1		.09	.09	28.05***	28.05***
Age	.09 (.05)*				
Step 2		.44	.35	54.37***	57.44***
Pain severity	-.10 (.41)*				
Catastrophising	.11 (.07)*				
Negative Affect	-.14 (.20)**				
Step 3		.70	.26	56.23***	32.70***
Pain self-efficacy	.32 (.10)***				
Pain acceptance	.16 (.13)**				
Values-based living	.26 (.09)***				
Optimism	-.08 (.27)				
Positive affect	.02 (.21)				
Instrumental support	-.01 (.18)				
Emotional support	.17 (.24)**				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The standardised regression weights for risk factors shown above are those from Model 3, the risk-resistance model.

Again, further model exploration was undertaken to explore potential multicollinearity issues The VIF for emotional social support was 2.74 and for instrumental social support was 2.16. Although these values were again below recommended thresholds (Pan & Jackson, 2008; Rogerson, 2001; Tabachnik & Fidell, 2013), emotional and instrumental support subscales were averaged to provide a total score for social support. Variance inflation factors for pain acceptance and pain self-efficacy were 2.95 and 3.30 respectively. The VIFs for catastrophising and negative affect were 2.05 and 2.30 respectively. Alternative models were specified, where each predictor

was removed one by one and replaced iteratively in different models. Pain severity, negative affect, pain self-efficacy, pain acceptance and social support were significant predictors in all models. Catastrophising continued to demonstrate a reversed sign in all models, even in those models where pain severity was the only other risk factor. A positive regression weight for catastrophising was only observed in a model that contained neither pain acceptance nor pain self-efficacy.

The final model, seen in Table 8.10, demonstrated regression signs all in the hypothesised directions. After adjusting for age, this model accounted for a significant 68% of the variance in QOL (F (6, 273) = 96.91, $p < .001$). Pain severity and negative affect were significant risk factors and pain self-efficacy, values-based living and social support were significant resistance factors. The addition of the resistance factors accounted for a significant 24% of the variance in QOL over and above that accounted for by the risk factors, $\Delta R^2 = .24$, ΔF (3, 273) = 69.09, $p < .001$. Social-ecological risk factors were entered in this final model as potential risk factors for exploratory purposes but they did not contribute additional unique variance in the measure of QOL over and above that of the other significant predictors.

Table 8.10

Hierarchical Multiple Regression of Quality of Life on Risk and Resistance Factors – Revised Model

	B (SE)	R_2	△ R_2	F	△ F
Step 1		.09	.09	28.05***	28.05***
Age	.10 (.05)*				
Step 2		.44	.35	71.69***	85.02***
Pain severity	-.11 (.41)**				
Negative Affect	-.11 (.18)*				
Step 3		.68	.24	96.19***	69.09***
Pain self-efficacy	.39 (.08)***				
Values-based living	.27 (.08)***				
Social support	.12 (.16)**				

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. The standardised regression weights for risk factors shown above are those from Model 3, the risk-resistance model.

8.3.5 Hypothesis four – mediation effects.

The fourth hypothesis proposed that the stress-processing factors (catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living) would mediate the relationships between risk factors and pain-related disability. As described in Chapter Three, Section 3.5.4.7, a mediator is an explanatory link in the relationship between one variable and another. It can be conceptualized as a mechanism through which a predictor influences an outcome (Rose et al., 2004).

Of the condition parameters, intrapersonal and social-ecological risk and resistance factors in the model, only pain severity and negative and positive affect were associated with pain-related disability at a moderate or greater strength. Therefore, the five stress-processing factors

(catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living) were tested as mediators of the relationships between pain severity and negative and positive affect with pain-related disability. Mediation effects were assessed first in single mediator models, then, where significant indirect effects were identified, in parallel mediator models. Parallel mediator models are discussed in detail in Chapter Three, Section 3.5.4.7. In essence, a parallel mediator model assesses the relative strength of multiple mediators when they act together to mediate (Hayes, 2013). Diagrams of a single mediator model and a parallel mediator model can be seen below in Figures 8.1 and 8.2.

As explained in Section 3.5.4.7 in Chapter Three, PROCESS facilitates comparison of indirect effects when tested in parallel mediator models. PROCESS provides confidence intervals for pairwise comparisons of specific indirect effects, allowing assessment of statistically significant differences between indirect effects (Hayes, 2013). Where more than one significant mediation effect was identified in a parallel mediator model, these effects were compared. As explained in Section 3.5.4.7 in Chapter Three, conclusions about the relative magnitudes of the indirect effects can only be made when comparing the indirect effects are in the same direction.

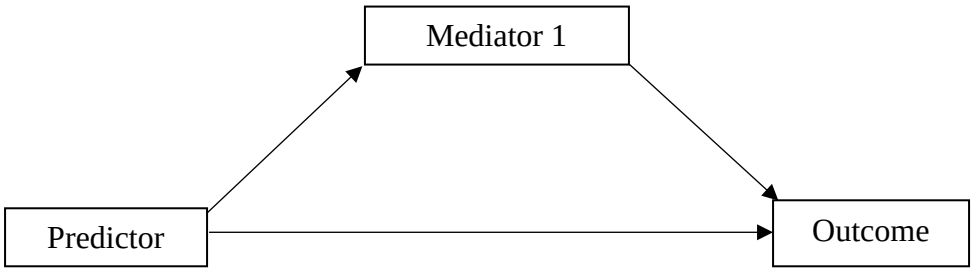

Figure 8.1 Simple mediation model.

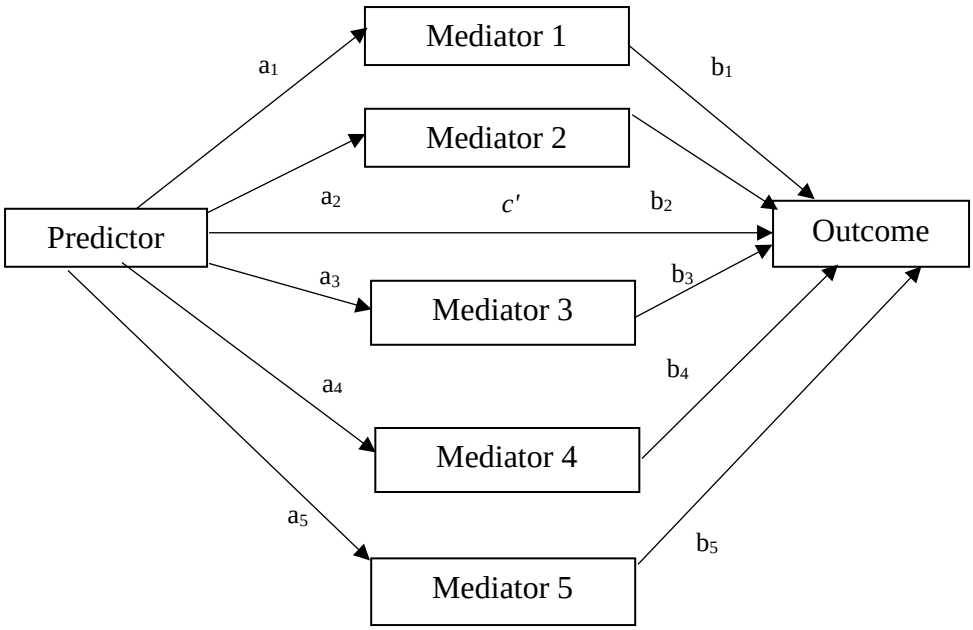

Figure 8.2 Parallel mediator model showing five potential mediators.

8.3.5.1 Single mediator models – risk factors predicting pain-related disability.

Hypothesis Four was supported in single mediator models. Pain severity and negative affect were associated with pain-related disability indirectly through an association with all five stress-processing factors. That is, pain severity and negative affect were indirectly associated with pain-related disability via an association with increased catastrophising and fear-avoidance. Pain severity and negative affect were also associated with pain-related disability via an association with lower pain self-efficacy, pain acceptance and values-based living. Results can be seen in Table 8.11.

In single mediator models, bias-corrected bootstrap intervals for the indirect effects of pain severity and negative affect on pain-related disability via catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living (paths *ab*) were all entirely above or below zero, indicating significant indirect effects. There was evidence that perceived pain severity and negative

affect also influenced pain-related disability directly (path c'), as the unstandardised regression weights for these paths remained statistically significant when the mediators were in these models. These results can also be seen in Table 8.10.

8.3.5.2 Single mediator models – resistance factors predicting pain-related disability.

Positive affect also influenced pain-related disability indirectly through an association with all five of the proposed mediators. That is, positive affect was indirectly associated with pain-related disability via an association with lowered levels of catastrophising and fear-avoidance and via higher pain self-efficacy, pain acceptance and values-based living. Bias-corrected bootstrap intervals for the indirect effect of positive affect on pain-related disability via catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living (paths *ab*) were all entirely above or below zero, indicating significant indirect effects.

There was evidence that positive affect influenced pain-related disability directly (path c') when its association with pain-related disability was mediated by catastrophising, fear-avoidance, pain acceptance and values-based living. This was because the direct path between positive affect and pain-related disability remained statistically significant in these mediated models. However, when the influence of positive affect on pain-related disability was mediated by pain self-efficacy, there was no evidence that positive affect influenced pain-related disability directly. This was because the unstandardised regression weight for this direct path was not statistically significant. Results of single mediator models can be found in Table 8.11.

Table 8.11

Single Mediation Models Predicting Pain-Related Disability (10,000 Bootstrap Samples)

Predictor (X)	Coefficient (SE) Predictor to M (Path *a*)	Coefficient (SE) M to Disability (Path *b*)	Indirect Effect of X on Disability (CIs) (Path *ab*)	Coefficient (SE) X to disability (Path *c'*)	Model summary R^2, F
Mediator: Catastrophising					
Pain Intensity	2.41 (.40)***	.32 (.06)***	.78 (.19) [.46; 1.21]#	3.97 (.43)***	.41, *F*(3, 276) = 63.43***
Negative Affect	1.67 (.13)***	.31 (.08)***	.51 (.14) [.27; .81]#	.91 (.22)***	.27, *F*(3, 276) = 34.84***
Positive Affect	-1.21 (.19)***	.40 (.07)***	-.48 (.11) [-.72; -.30]#	-1.13 (.23)***	.29, *F*(3, 276) = 37.76***
Mediator: Fear-avoidance					
Pain Intensity	.96 (.20)***	.51 (.13)***	.48 (.17) [.20; .88]#	4.27 (.44)***	.38, *F*(3, 276) = 56.97***
Negative Affect	.54 (.07)***	.48 (.15)**	.22 (.09) [.09; .47]#	1.17 (.19)***	.26, *F*(3, 276) = 33.16***
Positive Affect	-.47 (.09)***	.62 (.14)***	-.29 (.09) [-.53; -.13]#	-1.32 (.23)***	.25, *F*(3, 276) = 31.44***
Mediator: Pain Self-Efficacy					
Pain Intensity	-2.97 (.35)***	-.77 (.06)***	2.30 (.33) [1.70; 3.00]#	2.45 (.37)***	.61, *F*(3, 276) = 144.53***
Negative Affect	-1.24 (.13)***	-.88 (.06)***	1.09 (.13) [.84; 1.36]#	.34 (.16)*	.56, *F*(3, 276) = 115.94***
Positive Affect	1.64 (.16)***	-.93 (.06)***	-1.53 (.17) [-1.88; -1.22]#	-.08 (.20)	.55, *F*(3, 276) = 112.51***
Mediator: Pain Acceptance					
Pain Intensity	-1.61 (.25)***	-.91 (.09)***	1.46 (.25) [1.00; 2.00]#	3.30 (.39)***	.52, *F*(3, 276) = 110.32***
Negative Affect	-.84 (.09)***	-1.01 (.11)***	.85 (.12) [.63; 1.12]#	.58 (.18)*	.42, *F*(3, 276) = 67.88***
Positive Affect	.94 (.11)***	-1.05 (.10)***	-.99 (.14) [-1.30; -.74]#	-.60 (.22)**	.42, *F*(3, 276) = 66.49***
Mediator: Values-Based Living					
Pain Intensity	-2.08 (.35)***	-.43 (.07)***	.90 (.21) [.55; 1.37]#	3.85 (.43)***	.43, *F*(3, 276) = 69.20***
Negative Affect	-1.38 (.12)***	-.44 (.09)***	.61 (.13) [.38; .87]#	.82 (.21)***	.30, *F*(3, 276) = 39.31***
Positive Affect	1.58 (.15)***	-.50 (.09)**	-.78 (.15) [-1.10; - .51]#	-.83 (.26)**	.29, *F*(3, 276) = 37.26***

Note 1. ***p<.001, **p<.01, *p<.05

Note 2. Unstandardized b coefficients reported, effects adjusted for age.

Note 3. # Indicates significant indirect effect based on bias-corrected confidence intervals, Note 3. CI indicates confidence interval; M, mediating variable.

The fourth hypothesis also proposed that catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living would mediate the relationships between condition parameters, intrapersonal and social-ecological factors in the model with QOL. Pain severity, negative and positive affect, optimism and emotional and instrumental social support were all correlated with QOL at moderate or greater strength. Therefore, the model stress-processing factors (catastrophising, pain self-efficacy, pain acceptance and values-based living) were examined as mediators of the relationships between these condition parameters, intrapersonal and social-ecological factors with QOL. Fear-avoidance was only weakly associated with QOL so was not tested as a mediator.

8.3.5.3 Single mediator models – risk factors predicting quality of life.

Hypothesis Four was supported in single mediator models where risk factors predicted QOL. Pain severity and negative affect negatively influenced QOL indirectly through their effects on all four of the proposed mediators. That is, pain severity and negative affect indirectly decreased QOL by increasing catastrophising and by decreasing pain self-efficacy, pain acceptance and values-based living. Pain severity and negative affect also influenced QOL directly (path c'), as the unstandardised regression weights for these paths were statistically significant when the mediators were in the models. Results can be seen in Table 8.12.

8.3.5.4 Single mediator models resistance factors predicting quality of life.

Hypothesis Four was supported as positive affect, optimism and emotional and instrumental social support were all indirectly associated with QOL via decreased catastrophising and by increased pain self-efficacy, pain acceptance and values-based living. As can be seen in Table 8.11, bias-corrected bootstrap intervals for the indirect effects of perceived pain severity, negative affect, positive affect, optimism and social support on QOL via catastrophising, pain self-efficacy, pain

acceptance and values-based living (paths *ab*) were all entirely above or below zero, indicating significant indirect effects.

Positive affect and social support also influenced QOL directly (path c'), as the unstandardised regression weights for these paths were statistically significant when the mediators were in the models. Optimism also influenced QOL directly (path c') when its effects were mediated by catastrophising and pain acceptance. However, when the influence of optimism on QOL was mediated by pain self-efficacy and values-based living, optimism was only associated with QOL indirectly via these mediators as the unstandardised regression co-efficients for the direct paths between optimism and QOL in these models were not statistically significant. These results can be seen in Table 8.12.

Table 8.12

Summary of Simple Mediation Analyses Predicting Quality of Life (10,000 Bootstrap Samples)

Predictor (X)	Coefficient (SE) Predictor to M (Path a)	Coefficient (SE) M to QOL (Path b)	Indirect Effect of X on QOL (CIs) (Path *ab*)	Coefficient (SE) X to QOL (Path *c'*)	Model summary R^2
Mediator: Catastrophising					
Pain Intensity	2.43 (.40)***	-.43 (.08)***	-1.03 (.26) [-1.63; -.60]#	-3.49 (.54)***	.35, *F*(3, 277) = 50.02***
Negative Affect	1.67 (.13)***	-.19 (.09)***	-.32 (.15) [-.62; -.04]#	-1.79 (.24)***	.38, *F*(3, 277) = 56.05***
Positive Affect	-1.15 (.19)***	-.42 (.07)***	.48 (.12) [.27; .75]#	1.77 (.26)***	.14, *F*(3, 277) = 23.00***
Optimism	-1.68 (.25)***	-.48 (.08)***	.82 (.18) [.27; .75]#	1.25 (.36)***	.29, *F*(3, 277) = 36.86***
Emotional SS	-.83 (.17)***	-.43 (.07)***	.35 (.10) [.19; .58]#	1.66 (.21)***	.39, *F*(3, 277) = 60.12***
Instrumental SS	-41 (.14)**	-.54 (.07)***	.22 (.09) [.06; .43]#	.79 (.18)***	.30, *F*(3, 277) = 39.84***
Mediator: Pain Self-Efficacy					
Pain Intensity	-2.96 (.35)***	1.00 (.07)***	-2.95 (.40) [-3.78; -2.22]#	-1.57 (.45) ***	.59, *F*(3, 277) = 130.86***
Negative Affect	-1.22 (.13)***	.91 (.07)***	-1.12 (.13) [-1.39; -.87]#	-.99 (.17) ***	.62, *F*(3, 277) = 147.50***
Positive Affect	1.62 (.16)***	1.00 (.07)***	1.62 (.19) [1.28; 2.00]#	.62 (.23) **	.58, *F*(3, 277) = 127.38***
Optimism	1.68 (.22)***	1.08 (.07)***	1.81 (.25) [1.34; 2.33]#	.26 (.28)	.57, *F*(3, 277) = 122.31***
Emotional SS	1.04 (.15)***	-.95 (.06)***	1.00 (.14) [.72; 1.29]#	1.02 (.17)***	.62, *F*(3, 277) = 149.24***
Instrumental SS	.44 (.13)***	1.06 (.06)***	.47 (.14) [.19; .74]#	.54 (.14)***	.59, *F*(3, 277) = 133.21***
Mediator: Pain Acceptance					
Pain Intensity	-1.58 (.24)***	1.21 (.11)***	-1.92 (.37) [-2.66; -1.66]#	-2.60 (.48)***	.50, *F*(3, 277) = 93.00***
Negative Affect	-.82 (.09)***	1.07 (.11)***	-.87 (.13) [-1.16; -.63]#	-1.24 (.19)***	.52, *F*(3, 277) = 100.39***
Positive Affect	.96 (.11)***	1.18 (.12)***	1.13 (.17) [.82; 1.51]#	1.12 (.24)***	.49, *F*(3, 277) = 87.85***
Optimism	1.00 (.15)***	1.32 (.11)***	1.33 (.23) [.91; 1.82]#	.74 (.31)*	.46, *F*(3, 277) = 74.47***
Emotional SS	.49 (.10)***	1.20 (.10)***	.59 (.14) [.34; .87]#	1.42 (.18)***	.55, *F*(3, 277) = 113.61***
Instrumental SS	.27 (.09)**	1.34 (.11)***	.37 (.13) [.14; .63]#	.64 (.16)***	.48, *F*(3, 277) = 85.09***
Mediator: Values-Based Living					
Pain Intensity	-2.08 (.35)***	.97 (.07)***	-2.03 (.35) [-2.74; -1.38]#	-2.50 (.44)***	.57, *F*(3, 277) = 121.08***
Negative Affect	-1.37 (.12)***	.89 (.08)***	-1.22 (.15) [-1.53; -.95]#	-.89 (.20)***	.55, *F*(3, 277) = 112.65***
Positive Affect	1.55 (.15)***	.98 (.08)***	1.59 (.19) [1.17; 1.91]#	.73 (.24)**	.53, *F*(3, 277) = 105.28***
Optimism	1.83 (.20)***	1.10 (.08)***	2.02 (.25) [1.55; 2.54]#	.05 (.31)	.52, *F*(3, 277) = 98.96***
Emotional SS	1.18 (.13)***	.94 (.08)***	1.11 (.15) [.83; 1.41]#	.90 (.19)***	.55, *F*(3, 277) = 113.98***
Instrumental SS	.56 (.12)***	1.06 (.07)***	.59 (.13) [.34; .85]#	.42 (.15)**	.53, *F*(3, 277) = 104.17***

Note 1. ***p<.001, **p<.01, *p<.05
Note 2. Unstandardized b coefficients reported, effects adjusted for age.
Note 3. #Indicates significant indirect effect based on bias-corrected confidence intervals, CI indicates confidence interval; M, mediating variable

8.3.5.5 Parallel mediator models predicting pain-related disability.

Hypothesis Four was partially supported in parallel mediator models predicting disability. The variance explained in pain-related disability by pain severity, positive and negative affect occurred via statistically significant indirect effects through pain self-efficacy. Indirect effects of

pain severity, positive and negative affect on disability through catastrophising, fear-avoidance, pain acceptance and values-based living were not statistically significant.

Pain severity also influenced pain-related disability directly (path *c*') as well as indirectly because the unstandardised regression co-efficient for this direct path was statistically significant. By contrast, in the parallel mediator models the relationships between positive and negative affect and disability was only indirect, via self-efficacy. Pair-wise comparison of specific indirect effects was not conducted as only one significant indirect effect in these models was identified. These models can be seen in Figures 8.3 to 8.5.

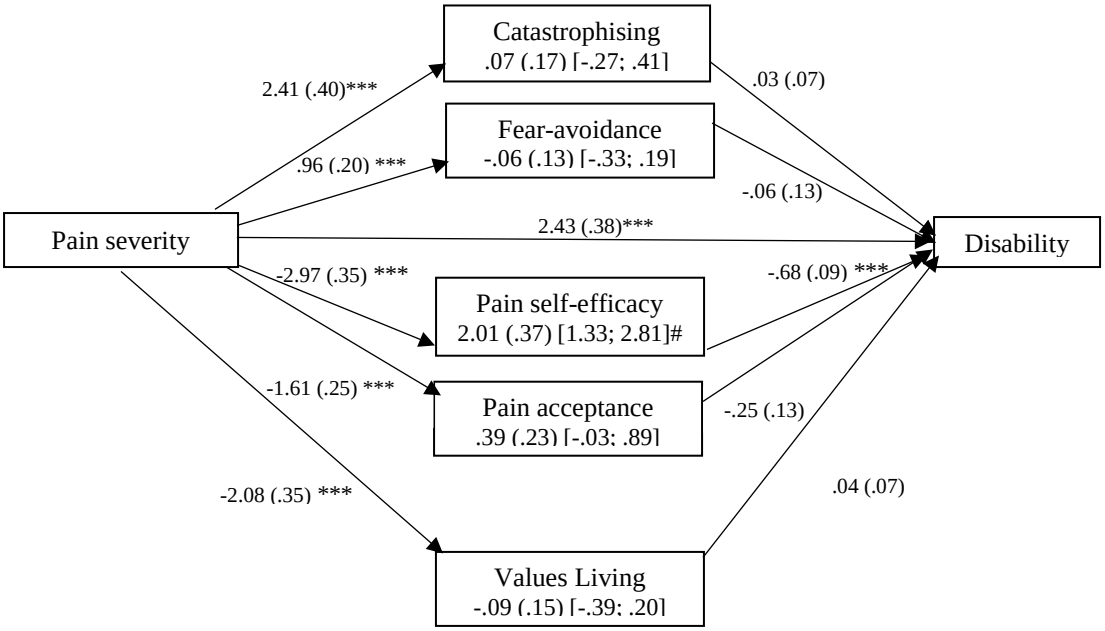

Figure 8.3 Only pain self-efficacy mediates the relationship between pain severity and pain-related disability.

Note 1.Total effect of pain intensity on disability = 4.75 (.43), model summary: R_2: .62, $F(7, 272) = 62.82$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

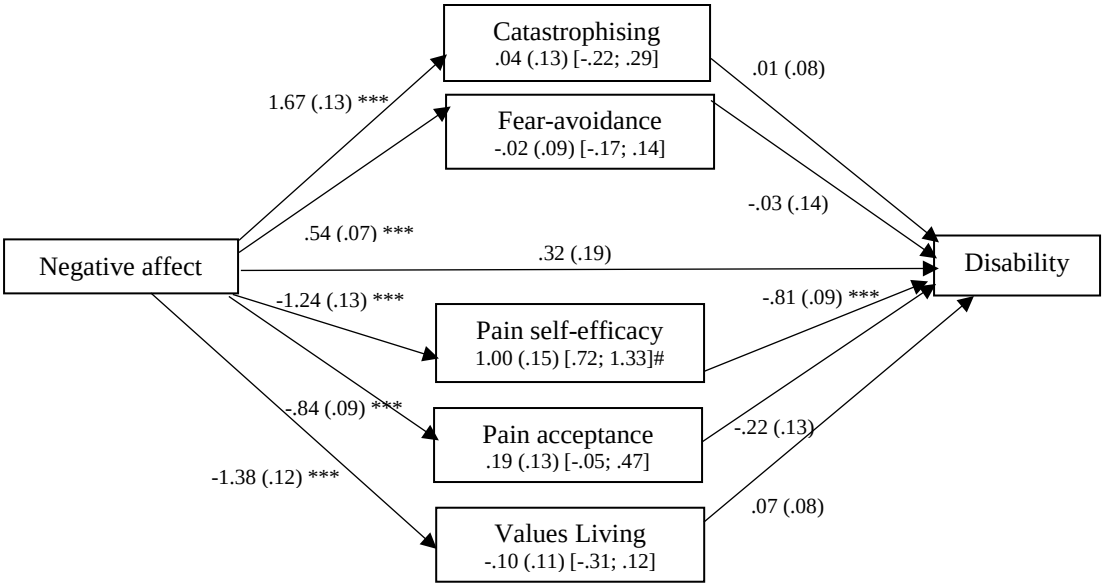

Figure 8.4 Only pain self-efficacy mediates the relationship between pain severity and pain-related disability.

Note 1. Total effect of negative affect on disability = 1.43 (.18), model summary: R_2: .56, $F(7, 272) = 50.19$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

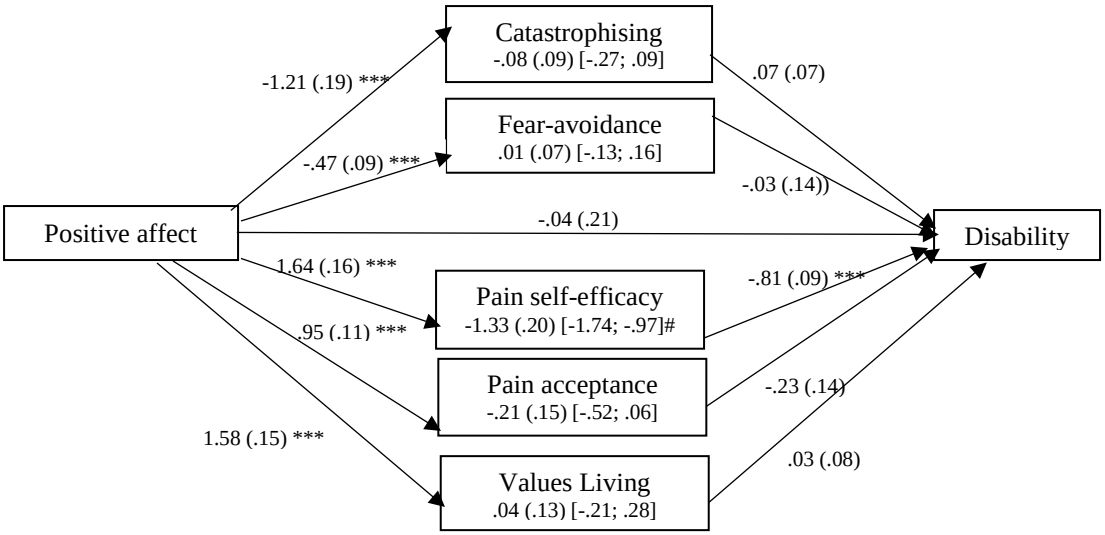

Figure 8.5 Only pain self-efficacy mediates the relationship between pain severity and pain-related disability.

Note 1. Total effect of positive affect on disability = -1.61 (.23), model summary: R_2: .56, $F(7, 272) = 49.28$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

8.3.5.6 Parallel mediator models predicting quality of life – pain severity.

Hypothesis 4 was partially supported for mediation models predicting QOL. The variance explained in QOL by pain severity occurred via statistically significant indirect effects through pain self-efficacy, pain acceptance and values-based living. The indirect effect of pain severity on QOL through catastrophising was not statistically significant. As well as being associated with QOL indirectly, via self-efficacy, acceptance and values-based living, pain severity also influenced QOL directly (path c'), as the unstandardised regression co-efficient for this direct path was statistically significant. This model can be seen in Figure 8.6.

Pair-wise comparison of specific indirect effects indicated the specific indirect effect of pain severity on QOL via pain self-efficacy was larger than its indirect effect via pain acceptance. Similarly, the indirect effect of pain severity on QOL via values-based living was significantly larger than its indirect effect via pain acceptance. No significant differences were noted when the indirect effect of pain severity on QOL via values-based living was compared to its indirect effect via pain self-efficacy. Comparison of indirect effects can be seen in Table 8.13.

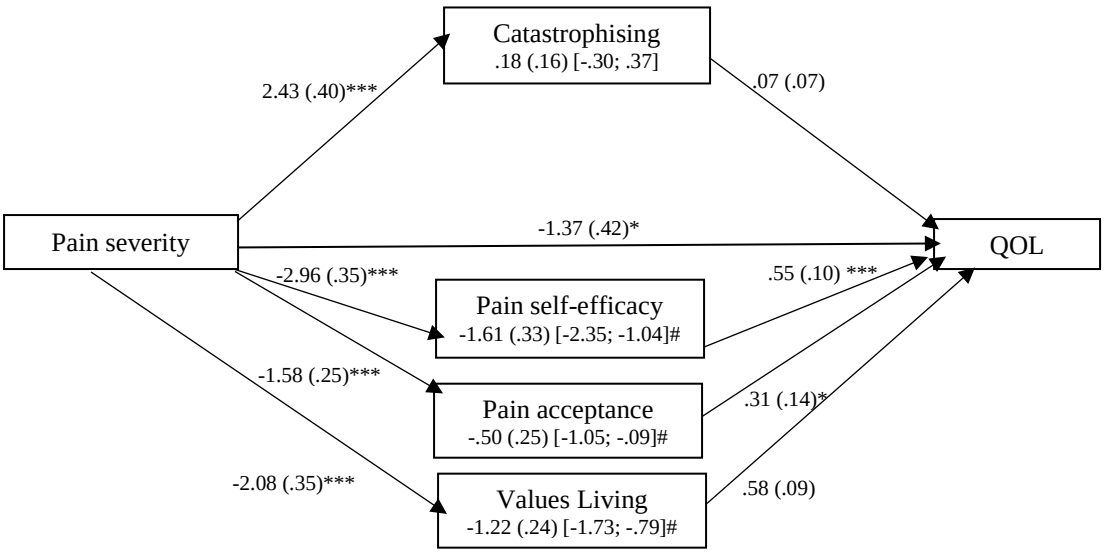

Figure 8.6 Pain self-efficacy, pain acceptance and values-based living mediate the relationship between pain severity and QOL.

Note 1. *Total effect of pain intensity on QOL = -4.52 (.54) ***, model summary* R_2*: .67,* F*(6, 274) = 53.37***,* ***p*<.001,* **p*<.01,* *p*<.05*

Note 2. ****p*<.001, ***p*<.01, **p*<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets

Table 8.13

Comparison of Indirect Effects in Parallel Mediator Model Pain Severity Predicting Quality of Life

Pain self-efficacy v Acceptance	-1.12 (.48) [-2.15; -.24]#
Pain self-efficacy v Values	-.40 (.39) [-1.18; .36]
Values v Acceptance	.72 (.35) [.03; 1.40] #

Note 1. # = significant difference in indirect effect

8.3.5.7 Parallel mediator models predicting quality of life – negative affect.

The variance explained in QOL by negative affect occurred via statistically significant indirect effects through all four mediators. Negative affect also influenced QOL directly (path c'), as the unstandardised regression co-efficient for this direct path was statistically significant. Thus, hypothesis 4 was supported in this model. This model can be seen in Figure 8.7.

Pair-wise comparison of indirect effects indicated that the indirect effect of negative affect on QOL through catastrophising was significantly different from the indirect effects through pain self-efficacy, pain acceptance and values-based living. Comparison of mediator effects that were the same direction found that the indirect effect of negative affect on QOL via values-based living was significantly larger than its indirect effect via pain acceptance. When the indirect effect via values-based living was compared to the indirect effect via pain self-efficacy no significant differences were noted. Comparison of indirect effects can be seen in Table 8.14.

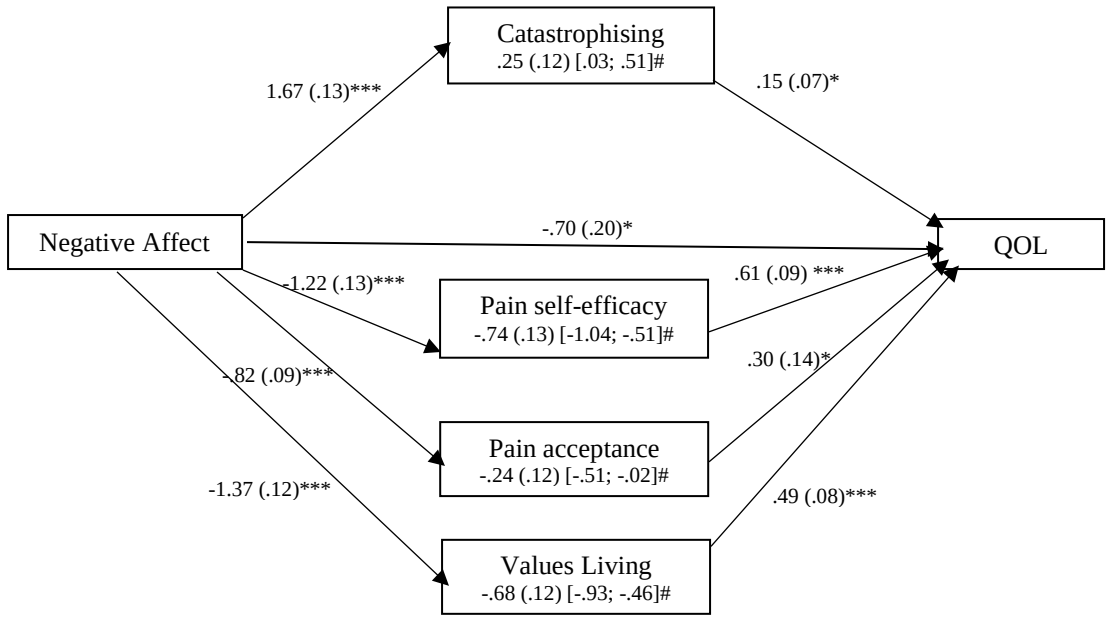

Figure 8.7. Catastrophising, pain self-efficacy, pain acceptance and values-based living all mediate the relationship between negative affect and QOL.

Note 1. Total effect of negative affect on QOL = -2.11 (.19) ***, model summary R_2: .67, $F(6, 274) = 91.89$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.14

Comparison of Indirect Effects in Parallel Mediator Model Negative Affect Predicting Quality of Life

Self-Efficacy v Acceptance	-.48 (.24) [-.97; -.02]#
Self-Efficacy v Values	-.06 (.19) [-.42; .32]
Acceptance v Values	.42 (.19) [.04; .81]#

Note. # = significant difference in indirect effect

There were signs in most of the above models of reversal of regression co-efficients of the paths from some mediators to outcomes. In all cases except the model above, in which the relationship between negative affect and QOL was mediated by catastrophising, pain self-efficacy, pain acceptance and values-based living, the indirect effect associated with the reversed co-efficient was

not statistically significant. To ensure any effects of collinearity did not influence indirect effects via the other mediators, an additional model was specified. This model can be seen below in Figure 8.8. Results in relation to the mediators in this latter model were unchanged.

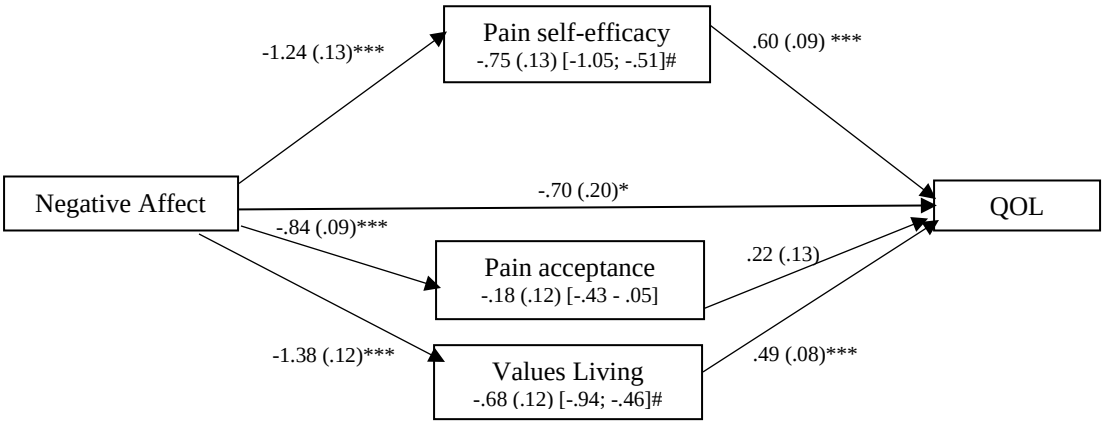

Figure 8.8. Pain self-efficacy, pain acceptance and values-based living all mediate the relationship between negative affect and QOL

Note 1. Total effect of negative affect on QOL = -2.12 (.19) ***, model summary R_2: .66, F(5, 274) = 107.54***

Note 2. ****p*<.001, ***p*<.01, **p*<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.15

Comparison of Indirect Effects in Parallel Mediator Model Negative Affect Predicting Quality of Life

Self-Efficacy v Acceptance	-.56 (.22) [-1.03; -.15]#
Self-Efficacy v Values	-.07 (.18) [-.43; .29]
Acceptance v Values	.50 (.18) [.14; .85]#

Note. # = significant difference in indirect effect

8.3.5.8 Parallel mediator models predicting quality of life – positive affect and optimism.

The variance explained in QOL by positive affect and optimism occurred via statistically significant indirect effects only through the resistance stress-processing factors, pain self-efficacy, pain acceptance and values-based living. The indirect effect of these predictors via catastrophising was not statistically significant. Positive affect and optimism were associated with QOL only indirectly (paths c'), as the unstandardised regression co-efficients for these direct paths were not statistically significant. These model can be seen in Figures 8.8 and 8.9.

The indirect positive effects of positive affect and optimism on QOL via pain self-efficacy were significantly larger than their indirect influence via pain acceptance. The indirect effects of positive affect and optimism on QOL expressed via pain self-efficacy was not significantly different from indirect effects via values. The indirect effect of positive affect and optimism on QOL via values differed significantly from indirect effects via pain acceptance. Because the indirect effects for pain self-efficacy, pain acceptance and values-based living were in the same direction, this indicates that the strength of the indirect pathway of positive affect and optimism on QOL was greater via pain self-efficacy and values than it was via acceptance. Pairwise comparisons can be seen in Tables 8.15 and 8.16.

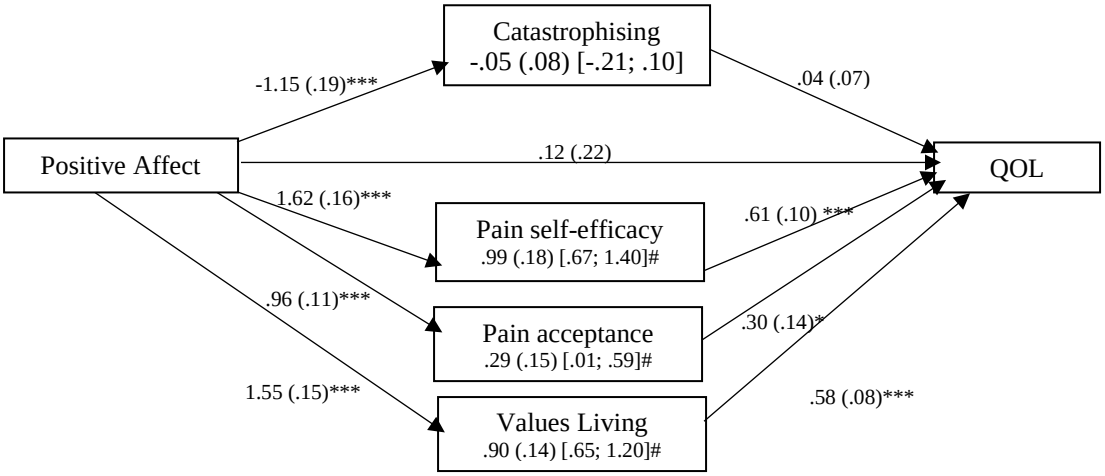

Figure 8.9. Catastrophising, pain self-efficacy, pain acceptance and values-based living all mediate the relationship between negative affect and QOL.

Note 1. Total effect of positive affect on QOL = 2.24 (.25)***, model summary R_2: .65, $F(6, 274) = 85.92$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.16

Comparison of Indirect Effects in Parallel Mediator Model Positive Affect Predicting Quality of Life

Self-Efficacy v Acceptance	.71 (.28) [.18; 1.29]#
Self-Efficacy v Values	.09 (.23) [-.36; .55]
Acceptance v Values	-.62 (.22) [-1.05; -.20]#

Note. # = significant difference in indirect effect

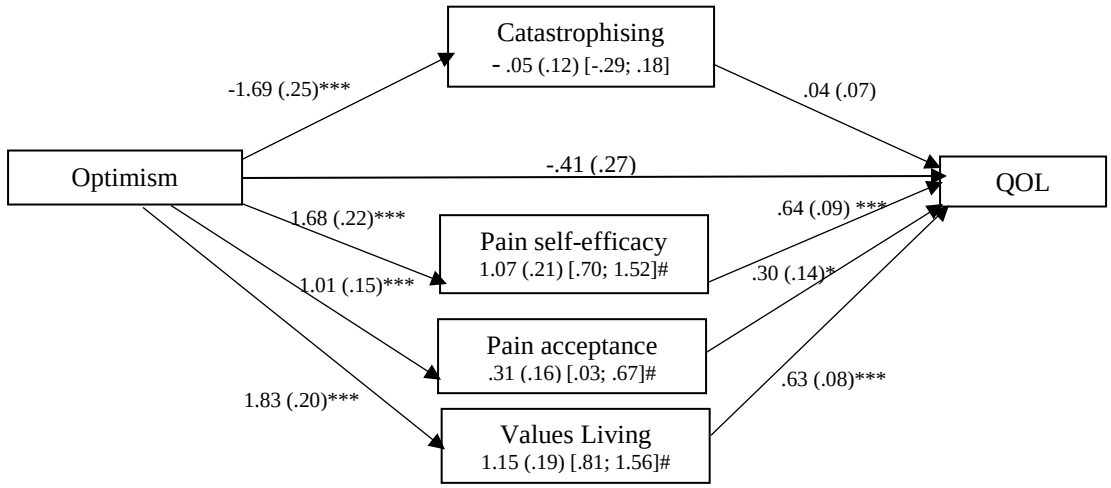

Figure 8.10 Pain self-efficacy, pain acceptance and values-based living all mediate the relationship between optimism and QOL

Note 1. Total effect of optimism on QOL = 2.07 (.35)***, model summary R_2: .66, $F(6, 274) = 86.89$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.17

Comparison of Indirect Effects in Parallel Mediator Model Optimism Predicting Quality of Life

Self-Efficacy v Acceptance	.76 (.31) [.19; 1.40]#
Self-Efficacy v Values	-.09 (.28) [-.63; .45]
Acceptance v Values	-.85 (.25) [-1.34; -.37]#

Note. # = significant difference in indirect effect

8.3.5.9 Parallel mediator models predicting quality of life – social support.

The variance explained in QOL by emotional and instrumental social support occurred via statistically significant indirect effects only through pain self-efficacy, pain acceptance and values-based living. The indirect effect of these predictors via catastrophising was not statistically significant. Emotional and instrumental social support were associated with QOL directly as well as indirectly via the mediators paths c'), as the unstandardised regression co-efficients for these direct paths were statistically significant. These models can be seen in Figures 8.10 and 8.11.

The strength of the indirect effect of social support on QOL via pain self-efficacy and values were significantly larger than the indirect effects via pain acceptance. The strength of the indirect effects of social support on QOL via pain self-efficacy did not differ significantly from the strength of the indirect effects via values. Because the indirect effects for pain self-efficacy, pain acceptance and values-based living are all of the same sign, these results indicate that the strength of the indirect pathway of social support to QOL was greater via pain self-efficacy and values than by via acceptance. Results of pairwise comparisons cane be seen in Tables 8.17 and 8.18.

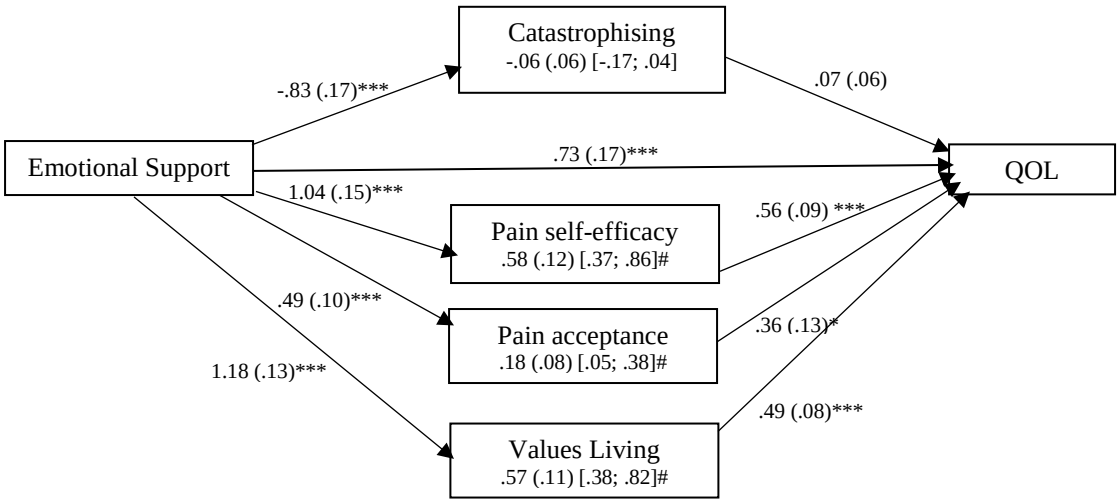

Figure 8.11 Pain self-efficacy, pain acceptance and values-based living all mediate the relationship between emotional social support and QOL

Note 1. Total effect of emotional support on QOL = 2.01 (.21)***, model summary R_2: .67, $F(6, 274) = 94.80$***

Note 2. ***p<.001, **p<.01, *p<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.18

Comparison of Indirect Effects in Parallel Mediator Model Emotional Social Support Predicting Quality of Life

Self-Efficacy v Acceptance	.40 (.16) [.11; .75]#
Self-Efficacy v Values	.01 (.16) [-.30; .32]
Acceptance v Values	-.39 (.14) [-.67; -.12]#

Note. # = significant difference in indirect effect

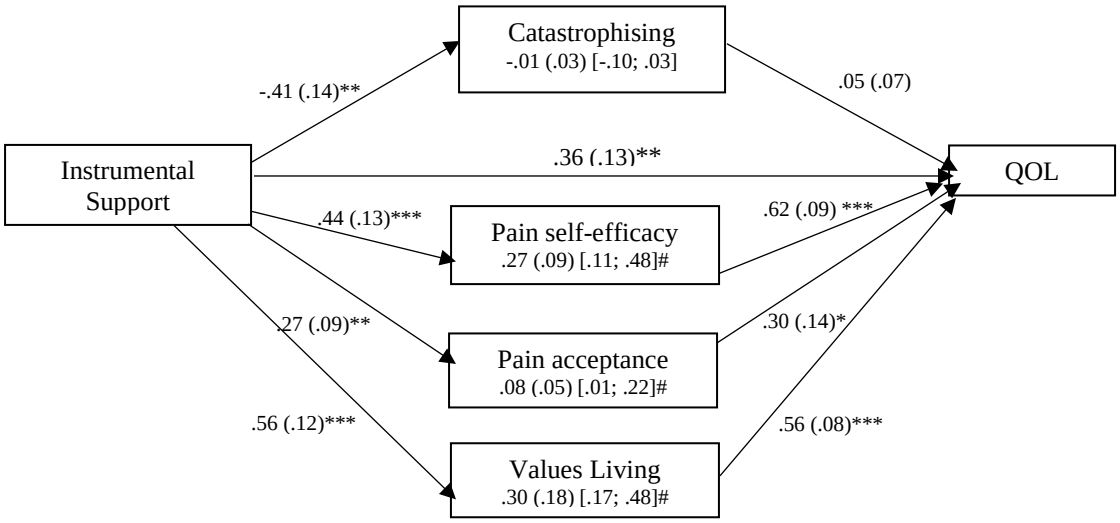

Figure 8.12 Pain self-efficacy, pain acceptance and values-based living all mediate the relationship between instrumental social support and QOL

Note 1. Total effect of instrumental social support on QOL= 1.01 (.19)***, model summary R_2: .66, $F(6, 274)$ = 89.50***

Note 2. ****p*<.001, ***p*<.01, **p*<.05, # = significant indirect effect

Note 3. Models adjusted for age.

Note 4. Unstandardized regression co-efficients with standard errors in parentheses are shown on the depicted paths. Indirect effects are shown in the boxes with standard errors in parentheses and associated bias-corrected confidence intervals in brackets.

Table 8.19

Comparison of Indirect Effects in Parallel Mediator Model Emotional Social Support Predicting Quality of Life

Self-Efficacy v Acceptance	.19 (.10) [.03; .42]#
Self-Efficacy v Values	-.04 (.09) [-.21; .16]
Acceptance v Values	-.23 (.09) [-.42; -.07]#

Note. # = significant difference in indirect effect

8.3.6 Hypothesis five – moderation models

Hypothesis Five proposed that resistance factors, pain self-efficacy, pain acceptance, values-based living, optimism, positive affect, and instrumental and emotional social support, would moderate the negative relationships between risk predictors with pain-related disability and

QOL. All risk factors that demonstrated a moderate or stronger ($r \geq .30$) relationship with these outcomes were included in these analyses. Resistance factors were seen as potential moderators that may specify the conditions under which a risk factor might exert a negative influence (Holmbeck, 1997). Thus, the moderating effects of pain self-efficacy, pain acceptance, optimism, positive affect, values-based living and social support were assessed for the relationships between pain severity, catastrophising, fear-avoidance and negative affect with pain-related disability and QOL.

As explained in Section 7.5.4.7 in Chapter Seven, moderation effects were calculated using the PROCESS macro for SPSS (Hayes, 2013). PROCESS calculates two-way interactions in moderation models, using 10,000 bootstrap iterations to calculate 95% bias-corrected confidence intervals of the moderation effects. Where significant interactions were identified, these were probed using PROCESS by plotting simple slopes at three levels of the moderator, low, high and medium (Aitken & West, 1991). A regions of significance analysis was then conducted using the Johnson-Neyman technique (Johnson & Neyman, 1936) in PROCESS. This analysis is explained in detail in Section 7.5.4.6.2 in Chapter Seven.

8.3.6.1 Moderation models predicting pain-related disability.

In partial support of Hypothesis Five, three significant moderation effects were noted. Pain acceptance moderated the negative relationship between both pain severity and negative affect with pain-related disability and values-based living moderated the negative relationship between negative affect and pain-related disability. Unstandardised regression co-efficients for these three models can be seen in Table 8.16. No significant moderation effects were noted in the models predicting QOL.

Results of all moderation tests can be found in Appendix I. The three significant interactions are in explored in detail below.

Table 8.20

Regression Models Estimating Pain Acceptance and Values-Based Living as Moderators of the Relationships between Risk Predictors and Pain-Related Disability

		Co-efficient	*SE*	*t*	*BC CIs*
Model 1					
R^2=.54, *F*(4, 275) = 79.64***					
	Pain Severity	.48	1.07	.46	-1.62; 2.59
	Pain Acceptance	-1.62	.27	-6.03	-2.15; -1.09
	Interaction	.13	.05	2.82	.04; .22
Model 2					
R^2=.44, *F*(4, 275) = 43.25***					
	Negative Affect	-.28	.38	-.75	-1.02; .46
	Pain Acceptance	-1.56	.24	-6.55	-2.02; -1.09
	Interaction	.05	.02	2.58	.01; .08
Model 3					
R^2=.32, *F*(4, 275) = 25.49***					
	Negative Affect	-.96	.75	-1.27	-2.44; .53
	Values-Based Living	-.85	.19	-4.36	-1.23; -.47
	Interaction	.03	.01	2.40	.01; .06

Note. ****p*<.001, ***p*<.01, **p*<.05, models adjusted for age.

8.3.6.2 Post-hoc probing of interaction between pain severity and acceptance.

The relationship between pain severity and pain-related disability was significant at all three levels of the moderator. As shown in Figure 8.3, individuals reporting low levels of pain acceptance were overall more disabled than those reporting high levels of pain acceptance, however perceived pain severity was not negatively associated with pain-related disability at low levels of pain acceptance. The Johnson-Neyman technique (Johnson & Neyman, 1936) demonstrated specifically that the negative relationship between pain severity and pain-related disability became statistically significant when pain acceptance levels were greater than 8.70. These results can be seen in Table 8.21.

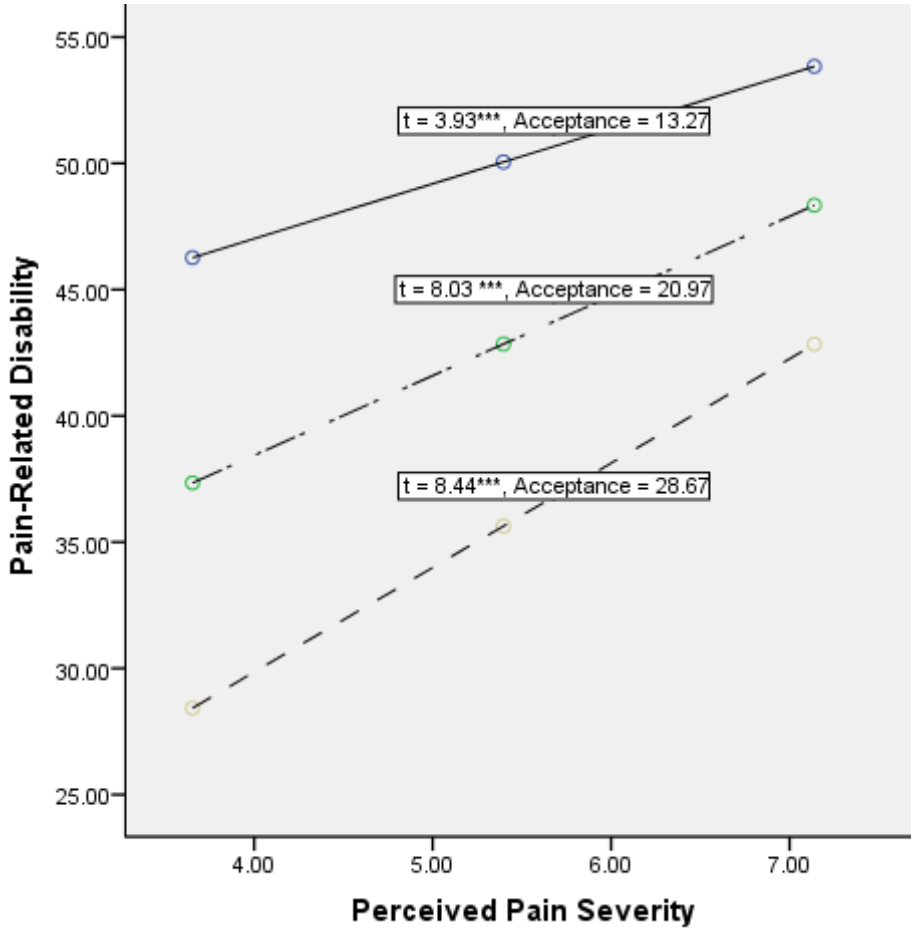

Figure 8.13 Pain acceptance moderates the relationship between pain severity and pain-related disability.

Table 8.21

Conditional Effect of Pain Acceptance on Relationship Between Pain Severity and Disability at Increasing Values of Pain Acceptance

Level of Pain Acceptance	Effect (SE) [BC CIs]	t (p)
3.00	.87 (.95) [-1.00; 2.73]	.90 (.36)
4.90	1.11 (.87) [-.60; 2.82]	1.28 (.20)
6.80	1.35 (.79) [-.21; 2.91]	1.70 (.09)
7.83	1.48 (.75) [.00; 2.96]	1.97 (.05)
8.70	1.59 (.72) [.18; 3.01]	2.21 (.03)
10.60	1.83 (.65) [.56; 3.11]	2.83 (.01)
12.50	2.08 (.58) [.93; 3.22]	3.56 (.00)
14.40	2.32 (.52) [1.29; 3.35]	4.44 (.00)
16.30	2.56 (.47) [1.64; 3.48]	5.45 (.00)
20.10	3.04 (.40) [2.26; 3.83]	7.61 (.00)
22.00	3.29 (.39) [2.52; 4.05]	8.43 (.00)
25.80	3.80 (.42) [2.94; 4.60]	8.89 (.00)
27.70	4.01 (.46) [3.10; 4.93]	8.63 (.00)
31.50	4.50 (.58) [3.36; 5.63]	7.80 (.00)
33.40	4.74 (.64) [3.47; 6.00]	7.38 (.00)
37.20	5.22 (.78) [3.68; 6.77]	6.62 (.00)
39.10	5.46 (.86) [3.77; 7.16]	6.35 (.00)
41.00	5.71 (.94) [3.86; 7.55]	6.09 (.00)

Note ***p<.001, **p<.01, *p<.05, model adjusted for age.

8.3.6.3 Post-hoc probing of interaction between negative affect and acceptance.

Overall, those with low levels of pain acceptance had the highest levels of pain-related disability, regardless of their level of negative affect. However, the relationship between negative affect and pain-related disability was significant only at moderate or high levels of pain acceptance. A plot of this interaction can be seen in Figure 8.4. The Johnson-Neyman technique (Johnson & Neyman, 1936) showed that the relationship between negative affect and pain-related disability became statistically significant when pain acceptance levels were greater than or equal to 16.30. These results can be seen in Table 8.22.

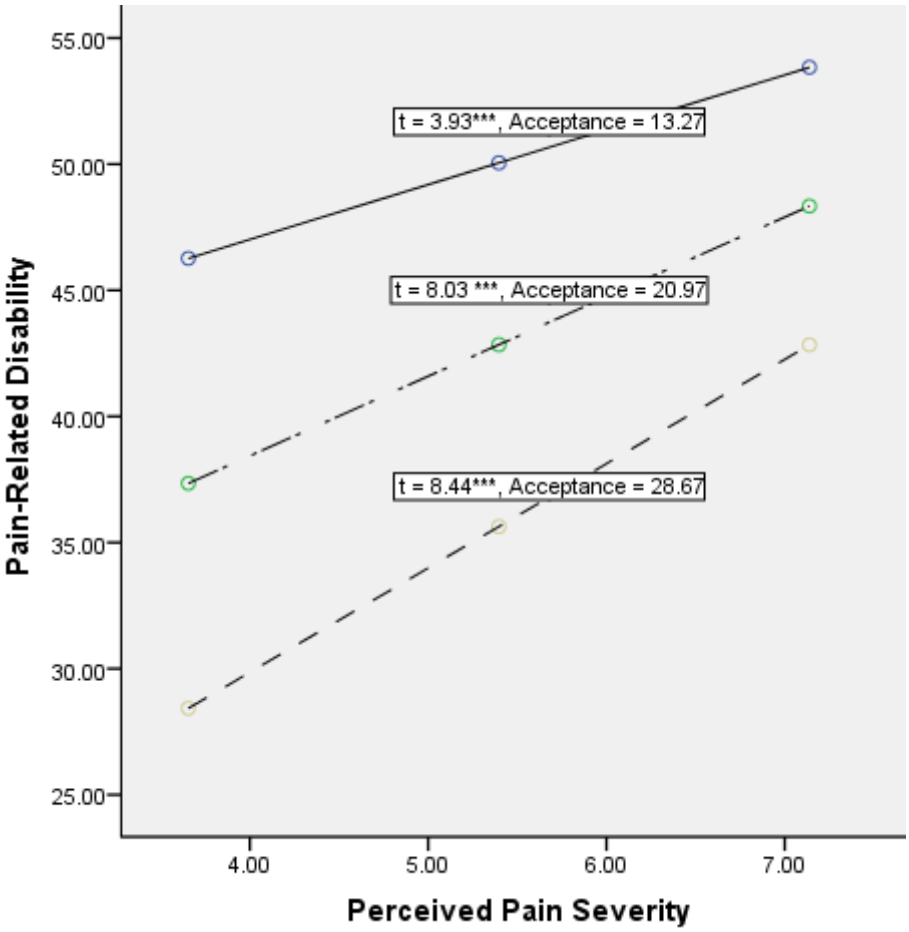

Figure 8.14 Pain acceptance moderates the relationship between negative affect and pain-related disability.

Table 8.22

Conditional effect of Pain Acceptance on Relationship Between Negative Affect and Disability at Increasing Values of Pain Acceptance

Level of Pain Acceptance	Effect (SE) [BC CIs]	t (p)
3.00	-.16 (.34) [-.83;.50]	-.49 (.63)
4.90	-.08 (.31) [-.68; .53]	-.25 (.80)
6.80	.01 (.28) [-.54; .56]	.04 (.97)
10.60	.18 (.23) [-.28; .64]	.79 (.43)
12.50	.27 (.21) [-.15; .69]	1.27 (.20)
14.40	.36 (.20) [-.03; .74]	1.82 (.07)
16.30	.45 (.18) [.10; .81]	2.41 (.02)
18.20	.53 (.18) [.20;.89]	2.99 (.00)
20.10	.62 (.18) [.27; .97]	3.49 (.00)
22.00	.71 (.18) [.34; 1.07]	3.85 (.00)
25.80	.88 (.21) [.46; 1.30]	4.17 (.00)
27.70	.97 (.23) [.51; 1.42]	4.18 (.00)
29.60	1.05 (.25) [.55; 1.55]	4.15 (.00)
33.40	1.23 (.31) [.63; 1.83]	4.02 (.00)
35.30	1.32 (.33) [.66; 1.97]	3.95 (.00)
37.20	1.40 (.36) [.69; 2.12]	3.87 (.00)
39.10	1.49 (.39) [.72; 2.26]	3.72 (.00)
41.00	1.58 (.42) [.75; 2.41]	3.74 (.00)

Note. ***p<.001, **p<.01, *p<.05, model adjusted for age.

8.3.6.4 Post-hoc probing of interaction between negative affect and values-based living.

The relationship between negative affect and pain-related disability was significant at all three levels of values-based living, however the strength of this relationship increased at higher levels of values-based living. Overall, those reporting low levels values-based living reported the highest levels of pain-related disability, regardless of their level of negative affect. Simple slopes plots of this interaction effect can be seen in Figure 8.5. The Johnson-Neyman technique (Johnson & Neyman, 1936) showed that the relationship between negative affect and pain-related disability became significant when the reported level of values-based living was greater than 41.83. These results can be seen in Table 8.23.

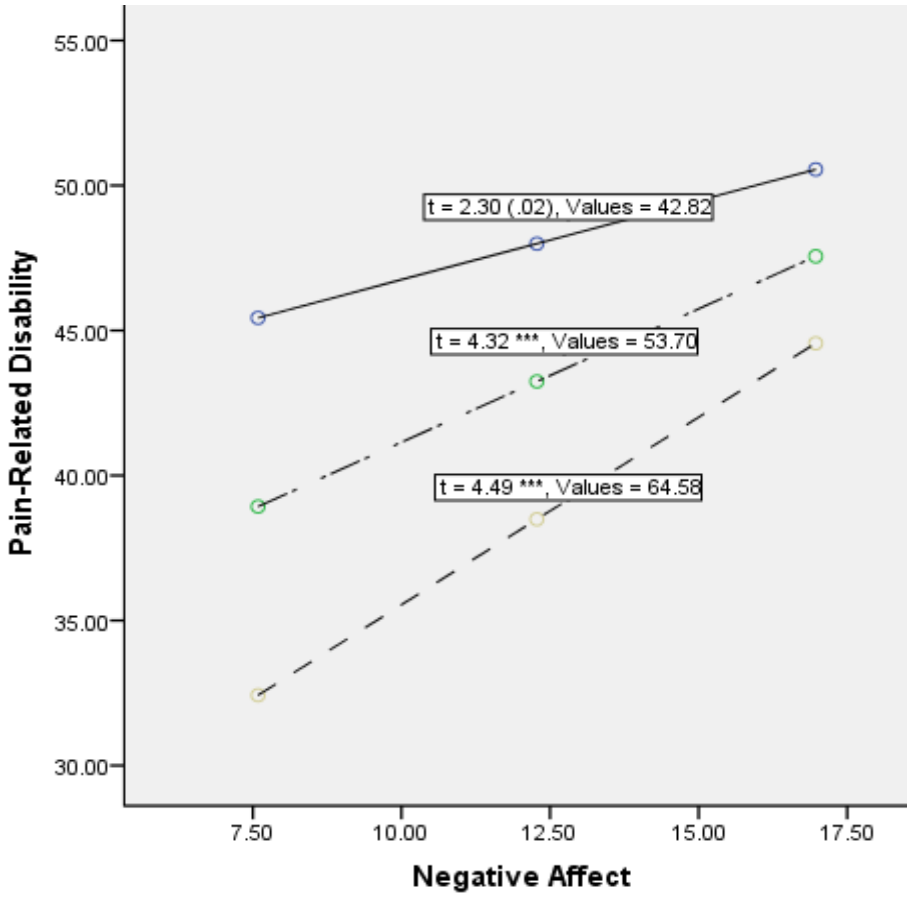

Figure 8.15 Values-based living moderates the relationship between negative affect and pain-related disability.

Table 8.23 *Conditional Effect of Values-Based Living on Relationship Between Negative Affect and Disability at Increasing Values of Pain Acceptance*

Level of Pain Acceptance	Effect (SE) [BC CIs]	t (p)
22.00	-.17 (.46) [-1.08; .74]	-.37 (.71)
24.90	-.07(.42) [-.91; .76]	-.17 (.87)
27.80	.03 (.39) [-.74; .79]	.08 (.94)
30.70	.13 (.35) [-.57; .83]	.37 (.72)
33.60	.23 (.32) [-.40; .86]	.71 (.48)
36.50	.33 (.29) [-.25; .90]	1.13 (.26)
41.21	.48 (.25) [.00; .98]	1.97 (.05)
42.30	.53 (.24) [.05; 1.00]	2.19 (.03)
45.20	.63 (.22) [.19; 1.07]	2.81 (.01)
48.10	.73 (.21) [.31; 1.14]	3.43 (.001)
51.00	.83 (.21) [.42; 1.24]	3.96 (.001)
53.90	.93 (.21) [.42; 1.24]	3.96 (.001)
59.70	1.13 (.25) [.64; 1.61]	4.59 (.000)
65.50	1.33 (.30) [.74; 1.91]	4.46 (.000)
68.40	1.42 (.33) [.78; 2.07]	4.34 (.000)
74.20	1.62 (.40) [.84; 2.40]	4.10 (.000)
77.10	1.72 (.43) [.87; 2.57]	3.99 (.000)
80.00	1.82 (.47) [.90; 2.75]	3.89 (.000)

Note. ***p<.001, **p<.01, *p<.05

8.4 Summary of Results

The first two hypotheses were largely supported. All risk factors except social-ecological factors demonstrated moderate to strong positive and negative associations respectively with pain-related disability and QOL. Resistance stress-processing and personal factors also demonstrated moderate to strong negative and positive associations respectively with pain-related disability and QOL. Resistance social-ecological factors (social support) were moderately positively associated with QOL but were only weakly associated with pain-related disability.

The third hypothesis that resistance factors would account for additional variance in the measures of disability and QOL, over and above that explained by the risk factors was supported. The risk-resistance model predicting pain-related disability explained 19% more variance than the risk-only model. The risk-resistance model predicting QOL explained 26% more variance than the risk-only model.

The fourth hypothesis that the stress-processing factors would mediate the relationships in the model between condition parameters, personal and social-ecological factors with adjustment outcomes was supported. When tested in single mediator models, each hypothesised indirect effect was significant. However, when these effects were analysed in parallel mediator models predicting pain-related disability, pain severity and negative and positive affect influenced pain-related disability indirectly only by an association with pain self-efficacy but not by an association with catastrophising, fear-avoidance, pain acceptance or values-based living.

When mediation effects were analysed in parallel mediator models predicting QOL, pain severity, positive affect, optimism and emotional and instrumental social support were associated with QOL indirectly via pain self-efficacy, pain acceptance and values-based living but not via

catastrophising. By contrast, negative affect was associated with QOL indirectly via catastrophising, pain self-efficacy, pain acceptance and values-based living.

When the indirect effects were compared in these latter models predicting QOL, the strength of the indirect effect of pain severity and negative affect on QOL was stronger via pain self-efficacy and values than it was via acceptance. Similarly, the strength of the indirect effect of positive affect, optimism and social support on QOL was stronger via pain self-efficacy and values than it was via acceptance. There were indications of effects of multicollinearity in most parallel mediator models. In all cases but one, the indirect effects associated with the reversed path co-efficients were not significant. In this model, the indirect effects were replicated in an alternative model in which the mediator with the reversed co-efficient was removed.

A final hypothesis regarding moderating effects of resistance factors on the relationships between risk factors and pain-related disability and QOL was only partially supported. Pain acceptance moderated the negative relationship between pain severity and pain-related disability. Both pain acceptance and values-based living moderated the negative relationship between negative affect and pain-related disability. However, these effects were in the opposite direction to what was hypothesised. Participants with high pain acceptance and values reported the lowest overall levels of disability, but the strength of the relationship between pain severity and negative affect with disability was stronger for those with high pain acceptance and high values compared to those reporting lower levels of these resistance factors. The implications of these results are discussed in Chapter Nine.

Chapter Nine – Discussion

9.1 Overview

This research aimed to investigate the direct and indirect effects of risk and resistance factors on pain-related disability and quality of life (QOL) for those living with chronic pain. It was guided by Wallander and Varni's (Wallander et al., 1989; Wallander & Varni, 1998) conceptual model of adjustment which was adapted from its original context according to the chronic pain literature. The model depicts three different effects that may foster improved understanding of pain adjustment processes. Specific effects tested were those hypothesised by the model; the unique and combined direct effects of risk (condition parameters, intrapersonal, social-ecological and stress-processing,) and resistance (intrapersonal, social-ecological and stress-processing) factors on pain-related disability and QOL; mediating effects of risk and resistance stress-processing factors in the relationships between condition parameters, risk and resistance intrapersonal and social-ecological factors and adjustment; and moderating effects of resistance factors on relationships between condition parameters, intrapersonal and social-ecological risk factors and adjustment.

It was postulated that understanding these potential effects would improve specificity of therapeutic targets and could inform further model development. Strong evidence existed to support many of the risk and resistance factors included in the current model. However, evidence for some of these factors and adjustment outcomes in heterogeneous community-based samples was lacking. Further, there was a paucity of research that explored ways that risk and resistance factors may influence adjustment indirectly. Wallander and Varni's (Wallander et al., 1989; Wallander & Varni, 1998) risk-resistance model provided a theory driven framework within which these potential effects could be explored.

Overall, results of the research were consistent with the hypotheses offered by the model. The direct effects hypothesis was largely supported. With a small number of exceptions, moderate or strong bivariate associations in the hypothesised directions were noted between pain severity, intrapersonal and stress-processing risk and resistance factors and adjustment outcomes. In contrast, only weak associations were found between social-ecological risk and resistance factors and pain-related disability. By contrast, a range of risk and resistance factors drawn from differing domains were linked to QOL in the Study Three sample. These findings are explored below.

As hypothesised, the resistance factors explained additional unique variance in the adjustment outcomes, over and above that explained by the risk factors. This highlights the importance in chronic pain rehabilitation interventions of simultaneously strengthening resistance factors while at the same time risk factors are addressed. In comparing the two risk-resistance regression models predicting pain-related disability and QOL, the resistance factors contributed more unique variance in the measure of QOL than they did in the measure of disability. This demonstrates that resistance factors play an especially important role in contributing to positive adjustment to pain. It also highlights that targeting resistance factors in treatment may be especially important for improving positive adjustment outcomes for those living with chronic pain.

The proposed mediation effects were, for the most part, supported. Stress-processing factors mediated relationships between pain severity, intrapersonal and social-ecological factors and adjustment. These mediator effects were evident in all single mediator models. However, when tested in parallel mediator models predicting pain-related disability, risk and resistance factors influenced adjustment only by associations with pain self-efficacy. In parallel mediator models predicting QOL, risk and resistance factors influenced adjustment predominantly by their association with resistance stress-processing factors, pain self-efficacy, pain acceptance and values-based living, but not via associations with catastrophising.

Significant moderation effects were identified, but only in the third study and in the opposite direction to what was hypothesised. Pain acceptance moderated the relationships between pain severity and negative affect (NA) and pain-related disability. Values-based living moderated the relationship between NA and pain-related disability. However, the direction of this effect was opposite to what was hypothesised. Despite that people with high acceptance and values were least disabled overall, the relationship between pain severity and disability and between negative affect and disability was stronger for people reporting high acceptance compared to those reporting low acceptance. Similarly, the relationship between negative affect and pain-related disability was stronger for those reporting high compared to low values. These findings are explored in detail below.

In order to place the current results in the context of the existing literature, this chapter first discusses the reported disability level of participants in Studies One and Three, comparing current findings to normative data. Then, consistent with the literature presented in Chapter Two and the order in which the study hypotheses were tested in Chapters Four and Eight, the current chapter discusses the research findings in relation to bivariate and combined direct effects before discussing findings in relation to mediation and moderation effects. Finally, overall implications of the research are presented followed by the strengths, limitations and directions for future research.

9.2 Reported disability levels

It was anticipated that participants in Study One, who were recruited from a pain clinic, would report disability levels comparable to other pain clinic samples and that the community-based sample in Study Three would report less disability compared to normative pain clinic data. The data only partly supported these expectations. Participants in Study One reported significantly higher disability levels compared to normative Australian pain clinic data provided by Nicholas

and colleagues (2008). As anticipated, the non-treatment seeking community-based participants in Study Three reported lower mean disability levels compared to normative pain clinic data for the Pain Disability Index (Chibnall & Tait, 1994). The Study Three sample reported a significantly higher pain self-efficacy level, a finding that was consistent with expectation given that a relatively higher functioning sample was sought.

Reasons to explain the higher disability levels of the Study One compared to the normative sample remain unclear. The significantly older age of the Study One sample compared to the normative sample may have been a contributing factor. However, mixed findings have been noted in research exploring the effect of age on pain-related disability (Hunter, 2001; Lachapelle & Hadjistavropoulos, 2005; Thomas et al., 2004; Turk et al., 1995) making the impact of age on disability in the current sample unclear. It is possible other demographic factors such as pain duration or number of medical comorbidities may have accounted for the noted differences but given no other demographic data were available such comparisons could not be made.

9.3 Risk factors

9.3.1 Condition parameters – direct effects of pain severity on adjustment.

Moderate strength positive bivariate associations were noted between pain severity and pain-related disability in both Studies One and Three. Of all the risk factors, pain severity demonstrated the strongest bivariate association with pain-related disability in both the pain clinic and community-based samples. These findings are consistent with a large volume of previous research that has shown perceived pain severity is strongly and independently associated with pain-related disability in chronic pain, even after adjusting for other psychosocial risk factors. Strong links between these factors have been demonstrated in community-based and primary care samples of individuals with chronic pain (Alcantara et al., 2010; Arnow et al., 2011; Campbell et al., 2013;

Hung et al., 2015; Nieto et al., 2013; Raftery et al., 2011; Sieben et al; 2005,). Positive and significant associations between pain severity and pain-related disability have also been noted in some pain clinic samples (Moix et al, 2011; Newton-John et al., 2014; Nicholas et al., 2009).

Two theoretical frameworks help explain how increased pain severity may contribute to pain-related disability. Operant behavioural paradigms suggest that when certain behaviours are linked with a punishing stimulus like pain, these behaviours tend to be extinguished over time (Bandura, 1977). That is, when something hurts, people stop doing it. The affective-motivational model of pain adjustment (Eccleston & Crombez, 1999) also suggests that the extent to which pain distracts people from other goal driven behaviours and instead drives avoidance is related at least in part to perceived pain severity. Michael, one of the Study Two participants, aptly described this experience, saying *"It's not being able to… do the physical things the way that you have done them in the past, you try to do something and you just get stopped by a wall of pain. So you don't do it, and then you don't do it again"*. As pain severity has been shown to be a strong driver of pain-related avoidance behaviours and appears to be related, at least partly, to threat interpretations (Crombez et al., 2013), education regarding these influences represents an important aspect of pain rehabilitation.

A moderate strength negative association was noted between pain severity and QOL in the community-based sample of Study Three. This finding reflects previous international research that has established links between perceived pain severity and QOL in both community-based and pain clinic samples (Lamé et al., 2005). Pain severity was a significant predictor of lowered physical QOL in a community-based sample of pain-affected adolescents (Merlijn et al., 2006) and was associated longitudinally with lower health-related QOL (HRQOL) over a period of six months in a large Canadian community-based (Nolet et al., 2015). Reductions in pain severity have also been

found to be linked to gains in perceived QOL in a large community-based sample of people with chronic pain related to fibromyalgia (Moore et al., 2010). The current results provide support in an Australian context for pain severity as an important risk factor not only for worsened physical function but also for lowered quality of life for those with chronic pain. They suggest that reduction in perceived pain severity in pain clinic and community-based samples might be expected to yield gains in both physical function and QOL.

9.3.2 Intrapersonal risk factors – direct effects of depression, anxiety and negative affect on adjustment.

The hypothesis that depression, anxiety and negative affect (NA) would be positively associated with pain-related disability was supported. These results are consistent with a large volume of previous research that has also demonstrated strong links between these risk factors and pain-related disability. For example, in a systematic review of psychological factors contributing to chronicity in low back pain, depression was identified as the strongest predictor of disability (Pincus et al., 2002). A strong relationship between NA and pain-related disability has been demonstrated in a heterogeneous community-based sample of people with chronic pain (Karsdorp & Vlaeyen, 2011), in a mixed pain clinic and community-based sample (Agar-Wilson & Jackson, 2012) and in individuals following spinal surgery (Seebach et al., 2012). Further a recent systematic review of treatment outcomes in fibromyalgia identified depression as a major predictor of poorer function (de Rooij et al., 2013). Links between anxiety and worsened physical function have also been reported in people with chronic low back pain (Moix et al., 2011). The current findings highlight the importance of addressing affective risk factors in pain rehabilitation.

The hypothesis that NA would be negatively associated with QOL in Study Three was also supported as a moderate strength negative correlation was noted. This finding expands the currently limited amount of literature that has demonstrated links between depression or NA and general

measures of QOL in mixed samples of individuals with chronic pain. A relatively small amount of research has found affective risk factors were significantly linked with worsened QOL in individuals with chronic pain. For example, Outcalt and colleagues (2015) established similar links in 250 primary care patients with chronic pain with and Elliott and colleagues (2003) reported a moderate negative correlation between depression and the mental but not physical component score of the Short-Form 36 (SF-36; Ware et al., 1993), a measure of HRQOL in a heterogeneous pain clinic sample of 242 individuals. Anxiety disorders were also linked to significantly lower HRQOL in a heterogeneous group of people with chronic pain (Kroenke et al., 2013).

The current results suggest interventions aimed at reducing NA could be expected to produce gains in both physical function and QOL for mixed groups of community-based individuals with chronic pain. Evidence exists for a number of such approaches including cognitive-behavioural therapy (Hofmann & Smits, 2008; Butler, Chapman, Forman & Beck, 2006), mindfulness-based approaches (Hofmann, Sawyer, Witt & Oh, 2010), medication (Gould, Otto, Pollack & Yap, 1997) and exercise (Krogh, Nordentoft, Sterne & Lawlor, 2011; Wipfli, Rethorst & Landers, 2008). As Kratz, Davis and Zautra (2007) pointed out, targeting NA in those with chronic pain may be particularly important as even small gains in mood over the lengthy time course associated with chronic pain may translate into substantial gains in function and life satisfaction.

The established links between pain perception and NA (Gore et al., 2005; Smedstad et al., 1995) were also supported in the current research. Pain severity was positively associated with depression, anxiety and NA all at moderate strength. These associations likely reflect bidirectional relationships whereby increased pain severity and NA mutually reinforce each other (Eccleston & Crombez, 1999; Sullivan et al., 1997). This idea was supported by Jane in Study Two who

recognised a link between mood and pain, saying "*Yeah, I know if I'm having a down day for other reasons, if someone's upset me or something I will feel pain because of that….if I'm happy the pain sort of melts away a bit.*"

Links between pain severity and NA may be explained by the shared anatomical and neurochemical pathways that have been suggested mediate the experiences of both pain and depression (Campbell et al.,2003; Fishbain et al; 1997; Shackman et al.,2011). Such ideas are beyond the scope of this thesis to explore in detail but highlight the reciprocity of many of the constructs explored in the current research.

9.3.3 Social-ecological risk factors.

9.3.3.1 Direct effects of solicitous and punishing partner responses to pain on adjustment.

Solicitous and punishing partner responses were weakly correlated with higher pain-related disability and lower QOL in Studies One and Three. Reported previous findings in relation to the relationship between both solicitous and punishing partner responses and pain-related disability have been mixed (Leonard et al., 2006). It is possible the current results may be partly understood in light of research that has identified the nature of the relationship between the individuals as important for determining the consequences of social interactions (Campbell, Jordan & Dunn, 2012; Krahe et al., 2013; Martire et al., 2011; Turk et al., 1995). Thus it is possible that the relationship between partner responses and adjustment outcomes may have been moderated by variables not assessed in the current research. Combined, these findings suggest that individualised assessment of the social context of pain patients may be important in order to prioritise treatment goals on an individual basis. Very little research appears to have explored links between partner responses to pain and QOL. Therefore the weak associations noted in Study Three extend the

current literature. It is possible this relationship was also modified by other contextual factors. Further research is needed to replicate this latter finding in other samples.

9.3.3.2 Direct effects of invalidation on adjustment.

In contrast to findings of Ghavidel-Parsa and colleagues (2015) and Wernicke and colleagues (2015) who reported moderate strength correlations between measures of invalidation and pain-related physical function, the current study identified instead only a weak association between invalidation and pain-related disability. The current finding is consistent with that reported by Kool and colleagues (2013) who identified similar strength associations between these constructs. Weak negative correlations were also noted between invalidation and QOL, suggesting little direct influence of these negative social experiences on pain-related function and QOL in the current sample. Participants interviewed in Study Two reported extensive experiences of invalidation. For example, invalidation was reported to be associated with the invisibility of chronic pain and having a diagnosis that may be seen by others as questionable.

> *Oh maybe just that with problems like fibromyalgia there has been that stigma around…is it real?… Because it's an invisible illness, and there's that constant feeling that....people don't think your pain is genuine, or they think it's all in your head*".
>
> (Kim, aged 33)

Reasons to explain the current findings in relation to some previous research (Ghavidel-Parsa et al., 2015; Wernicke et al., 2015) are unclear but may relate to differential effects of social experiences according to the characteristics of the person experiencing them, as noted above. The current findings may also be attributable to sample characteristics. For example, it is possible that the negative effects of invalidation on pain-related function are stronger in specific diagnostic groups such as people with fibromyalgia or chronic low back pain compared to mixed samples of

pain patients. Further research is needed to see if the current findings in relation to invalidation are replicated in other mixed groups of community-based individuals with chrnic pain.

9.3.4 Risk stress-processing factors.

9.3.4.1 Direct effects of catastrophising on adjustment.

The hypothesis that catastrophising would be positively related to pain-related disability and negatively associated with QOL was supported. Moderate strength correlations between catastrophising and both adjustment outcomes were noted in Studies One and Three. The relationship between catastrophising and pain-related disability identified in the current research reflects a large body of previous research that has demonstrated similar links in both pain clinic and community-based samples (Arnow et al., 2011; Bergbom et al., 2011; Smeets et al., 2006; Sullivan et al., 2001; Thompson et al., 2010). Some research has also identified links between early catastrophising and subsequent increased risk for pain chronicity (Pincus et al., 2002), suggesting a potentially causative role of catastrophising in pain and pain-related disability. However, the likelihood of a bi-directional relationship between these factors is also acknowledged. For example, when normal life activities are difficult because of pain, appraisals of pain are likely to become increasingly negative.

The moderate negative association in Study Three of catastrophising with QOL parallels previous research that has also demonstrated moderate to strong negative correlations between catastrophising and QOL in both pain-clinic (Borsbo et al., 2008, 2010; Lame et al., 2005) and community-based samples (Kall, 2009). The negative association of catastrophising with QOL suggests that reduction of catastrophising in treatment, for example using cognitive-behavioural approaches (Morley, Williams & Hussain, 2008; Thorn et al., 2007), could be expected to benefit not only physical function but also QOL.

9.3.4.2 Direct effects of fear-avoidance on adjustment.

The hypothesis that fear-avoidance would be positively associated with pain-related disability was supported with a moderate strength positive correlation observed. This reflects a large volume of previous research that has reported similar links in cross-sectional pain clinic and community-based data (Bean et al., 2014; Perry & Francis, 2013; Zale et al., 2013). Anecdotal links between fear-avoidance and pain-related disability were described in Study Two as several participants reported having to overcome a fear of physical activity in order to recover. Most participants had worked with health professionals to become more active and in doing so had faced fears about worsening pain with exercise. For example, Michael said he had to work with physiotherapist to gradually re-teach his body that not every movement was going to be associated with pain. Bettina also said that her GP had helped allay her anxiety about becoming more active and this was a turning point in her recovery.

> *Being more confident to take on physical exercise (helped me)….. my GP did help. She encouraged me, "Don't be scared of that. If you do it and it's too hard, don't push yourself, but if you try and you get some benefit from it, then that … would be better for you." Again, it was just hearing it from someone that I trusted that I thought, of course, I should be doing that sort of thing.* (Bettina, aged 44)

The relationship identified in the current research between fear-avoidance and functional impairment is consistent with much research that has identified fear-avoidance as a key therapeutic target in pain rehabilitation (Vlaeyen & Linton, 2000). Research exploring the effectiveness of interventions for reducing fear-avoidance has focused on both cognitive- behavioural interventions (Woby et al., 2004) and exposure approaches (Boersma, Linton, Overmeer, Jansson, Vlaeyen & de Jong, 2004) with benefits demonstrated for both.

Fear-avoidance demonstrated only a weak negative association with QOL. This is consistent with a small body of research that has also demonstrated weak links between fear-avoidance and both physical and mental component scores of a measure of HRQOL for those with chronic pain (Abbott et al., 2010; Kovacs et al., 2007). It is feasible that fear-avoidance would be more strongly linked to physical function than QOL because it assesses the extent to which people avoid physical tasks due to pain. It would thus be expected to relate more strongly to physical function than global measures of QOL.

9.3.5 Risk factors, summary.

The current findings support the relevance of the conceptual model to chronic pain adjustment processes and support previously observed direct links between risk factors and adjustment outcomes. In addition, two unique contributions to the existing literature are noted from the current research. Firstly the current research demonstrated only weak links to exist between partner responses to pain and perceived QOL. Secondly, despite experiences of invalidation being associated with poorer pain-related physical function in previous research, direct links between these measures and adjustment outcomes were weak in the current sample. Further research is needed to validate these findings in other chronic pain samples. The next section examines the direct associations between resistance factors and adjustment outcomes.

9.4 Resistance Factors

9.4.1 Intrapersonal resistance factors.

9.4.1.1 Direct effects of positive affect on adjustment.

In the current research, moderate strength negative correlations were noted between positive affect (PA) with pain-related disability. Anecdotal evidence regarding positive adjustment effects of PA were also provided by several Study Two participants. These seemed to relate mainly

to physical activity or positive social experiences. Kim said "*So I leave the pool feeling like my old self, you know, like my old self, really good… just feel really upbeat and positive*". The majority of participants in Study Two reported that a positive mental attitude promoted better pain coping. Bettina, for example, described making a conscious effort to keep her thinking positive, Jane thought developing a more positive attitude in her workplace promoted social connections and Michael said he tried to keep a positive attitude despite struggles with chores.

Relatively little quantitative research appears to have explored links between PA and physical function for those with chronic pain although lower PA has been linked at weak strength in some studies to greater functional limitation (Kothari et al., 2015; Seebach et al., 2012; White et al., 2012; Zautra et al., 1995). The moderate strength negative correlation found in Study Three between PA and pain-related disability may have been due in part to the fact that one of the items on the shortened form of the Positive and Negative Affect Scale (PANAS; Thompson, 2007; Watson et al., 1988) used in the current research asked participants to record the extent to which they felt 'active'. Due to obvious links between feeling 'active' and functional impairment, the associations between these two variables may have been inflated. Further research is needed to explore links between PA and adjustment to chronic pain. Validation of the short and long forms of the PANAS links in other chronic pain samples will contribute to reliability of findings in this area.

The finding that PA was moderately positively associated with QOL in Study Three expands the existing literature as few studies have examined these links in pain-affected samples. Positive associations between these factors have been identified in general illness samples that included individuals with chronic arthritis-related pain (Eaton et al., 2014) and in individuals with spinal cord injuries (Van Leeuwen et al., 2012). The current findings highlights that PA may represent a resistance factor associated with improved QOL for individuals with varying pain

diagnoses and suggests that targeting PA in pain rehabilitation may provide adjustment benefits for those with chronic pain. Research has demonstrated that skills associated with PA can be learned, although such interventions are not nearly as well-researched as those for depression. Early work by Fordyce (1981, 1983) showed that students who participated in a 'happiness intervention' that comprised teaching about behaviours associated with happiness such as being present oriented and keeping physically active, demonstrated improved levels of PA up to 18 months after the intervention. Subsequently, the positive psychology movement has demonstrated efficacy for happiness interventions in multiple studies (Emmons, & McCullough, 2003; Seligman, Steen, Park & Peterson, 2005; Sin & Lyubomirsky, 2009).

9.4.1.2 Direct effects of optimism on adjustment.

The hypothesis that optimism would be directly associated with improved pain adjustment outcomes was only partly supported. At the bivariate level, optimism was weakly negatively associated with pain-related disability. This finding is consistent with previous research that has also reported weak links between optimism and physical function for those with chronic pain (Ramirez-Maestre et al., 2012; Wright et al., 2011) but contrasts with a small amount of other research that has linked optimism to better physical function (Cruz-Almeida et al., 2013).

Seligman (2012) suggested that optimism may exert protective effects on health during the earlier stages of a disease but may have less impact as the disease progresses. Thus, it is possible the weak association between optimism and physical function in the current research reflected an erosion of optimism over time as participants in Study Three reported a mean pain duration of 12 years. This idea is supported by other research that also identified weak links between optimism and physical function in samples with lengthy pain durations (Ramirez-Maestre et al., 2012; Wright

et al., 2011). More research is needed to explore links between optimism and adjustment in other chronic pain samples and additional variables that may moderate these relationships.

The moderate strength positive bivariate association between optimism and QOL in the current research is consistent with a small amount of research that has identified optimism as associated with improved QOL for those with musculoskeletal pain (Tsakogia et al., 2011), rheumatoid arthritis (Treharne et al., 2005, 2007) and with life satisfaction in people with osteo-arthritis (Ferreira & Sherman, 2007). Very few studies appear to have explored links between optimism and QOL in heterogeneous community-based samples of adults with chronic pain. Accordingly, the current research provides new insights into links between optimism and QOL in this population. Despite the weaker bivariate associations between optimism and QOL when compared to other resistance factors, it is still possible that optimism represents an important resource for those with pain because optimism has been linked to disposition and thus may represent a stable personality-based resource that is relatively impervious to the ongoing challenge of living with pain.

Although optimism is often seen as dispositional (Scheier & Carver, 1987, Scheier et al., 2001) some theorists have also suggested it is amenable to change (Seligman, 2011). For example, cognitive techniques such as imagining a more positive future or the ‘best possible self’ intervention described by Hanssen and colleagues (2012) may help increase levels of optimism. It is possible that interventions aimed at increasing optimism for those with chronic pain might be expected to yield QOL benefits for those with chronic pain, even if not directly contributing to improved physical function. Further research is needed to validate these findings in other pain-affected samples.

9.4.2 Social-ecological resistance factors.

9.4.2.1 Direct effects of social engagement on adjustment.

As social support has been shown to confer protective effects for those with a range of chronic health conditions including chronic pain, it was hypothesised that social engagement would be negatively associated with pain-related disability. Results supported this hypothesis with a moderate strength negative correlation observed. This association suggested that socialising with others outside the house provided a protective benefit for pain-related physical function. However, it did not discount the possibility that greater pain-related disability precluded the socialising that would potentially serve as a protective mechanism (Newsom & Schulz, 1996).

It is possible that the social engagement measure used in Study One assessed factors other than, or in addition to, social support. For example, social engagement may have reflected a functional capacity of participants to get out of the house, rather than support derived from social activities. The extent to which the findings in relation to social engagement can be generalized to other more commonly assessed forms of social support remain unclear but may be elucidated in subsequent research.

9.4.2.2 Direct effects of instrumental and emotional social support on adjustment.

Emotional and instrumental support were negatively associated in Study Three with pain-related disability at weak strength but were positively associated with QOL at moderate strength. The literature exploring links between social support and pain-related disability has yielded mixed results. Chronic pain patients reporting high levels of social support are generally less disabled by pain (Campbell et al., 2003; Demange et al., 2004; Evers et al., 2003; Ferreira & Sherman, 2007; Marin, Holtzman, DeLongis, & Robinson, 2007) but inconsistencies in these associations have

been noted and negative effects of overly supportive partner responses to pain have been observed (Flor et al., 1989; Williamson et al., 1997).

The mixed findings pertaining to the influence of social support on pain-related function may also be a reflection of other factors that have been shown to influence the adjustment impact of social support in those with chronic pain. These factors include degree of satisfaction with the relationships that provided the support (Stark-Taylor et al., 2013), attachment style (Meredith et al., 2008), characteristics of the person receiving support (Martire et al., 2011; Romano et al., 1995) and the interpretation of the intention, desirability, and nature of the helping behaviour (Revenson Schiaffino, Majerovitz & Gibofsky, 1991). That such factors might impact the degree to which social support influences function were highlighted in Study Two by Jacqui who noted that practical support was helpful, but not if it was motivated by pity.

> *I don't want pity. That's one thing I don't want, you know. If someone says 'I'll give you a hand,' because they genuinely want to give me a hand, that's fine, but if they want to do something for me because they think I can't then I'm probably more likely to get my back up.* (Jacqui, aged 45)

Jane, aged 51, also noted mixed feelings in relation to instrumental support: *"(Support) does come with a down side because he doesn't like doing it but he says, "Oh, I've done this vacuuming all morning, you've just sat there and done nothing."* These complexities inherent within the construct of social support may help explain the weak links observed in the current research between social support and disability. Such factors could be explored as moderators in subsequent research. The stronger association of social support with QOL compared to disability noted in the current research likely reflects the social relations items within the QOLS but also suggests a protective effect of social support on QOL for those with chronic pain. Such links are

also consistent with a large volume of research that has supported direct protective effects of social support on QOL (Cohen & Wills, 1985; Stanton et al., 2006).

9.4.3 Stress-processing resistance factors

9.4.2.3 Direct effects of pain self-efficacy on adjustment.

Consistent with much previous research that has demonstrated pain self-efficacy as a powerful determinant of physical function for those with chronic pain (Arnstein et al., 1999; Keefe et al., 1997; Lefebre et al., 1999; Riesma et al., 1998), the current research also demonstrated a robust negative relationship between pain self-efficacy and pain-related disability. In Study Three pain self-efficacy was also strongly positively associated with QOL. This suggests that pain self-efficacy exerts a protective influence not only on physical function but also on QOL across a broad range of domains. The association in the current study of pain self-efficacy with improved QOL contributes to the literature because research exploring this relationship in community-based non treatment-seeking samples appears to be currently sparse.

Participants in Study Two described an ability to 'just keep going' which may have reflected a sense of self-efficacy for managing pain or may have reflected stoicism. Several participants described their confidence to manage pain arose from childhood or adult family relationships. For example, Jacqui said "*Yeah, I mean we've always been that sort of a family where you pick yourself up and get on with it 'what are you whinging about?' … and that's…what keeps me going*". This suggests a learned aspect to the acquisition of positive self-efficacy beliefs and is consistent with a qualitative study of 13 injured workers who described the ability to cope with pain as an intrinsic attitude that arose through modelling by parents and significant others (Carroll, Rothe & Ozegovic, 2013). This suggests that teaching patients that coping skills can be learned or modified may foster improved pain adjustment outcomes.

Bandura (1977) stated that the level and strength of a person's self-efficacy determines what goals are set, which behaviours are attempted, how much effort is expended and whether the effort is sustained. It is possible that chronic pain exerts a particularly strong negative influence on self-efficacy beliefs as it represents a constant aversive stimulus, the removal of which is beyond personal control. Thus, it might be expected that self-efficacy beliefs help determine the capacity of an individual to function despite pain, but equally that high levels of pain-related disability will have negative effects on self-efficacy beliefs. It has been suggested that the mechanisms through which self-efficacy beliefs are acquired are both cognitive and behavioural (Bandura, 1986). Clinicians working with individuals with chronic pain may aim to increase pain self-efficacy through use of modelling, mastery experiences, exposure and social reinforcement (Bandura, 1977; Keefe, Rumble, Scipio, Giordano and Perri, 2004).

9.4.2.4 Direct effects of pain acceptance on adjustment.

The finding that pain acceptance was strongly and positively associated with pain-related disability is consistent with a large volume of previous research that has demonstrated similar associations. This relationship has been demonstrated in both pain clinic (Esteve et al., 2007; McCracken, 1998; McCracken & Eccleston, 2003, 2006; Vowles et al., 2011; Vowles et al., 2014) and primary care samples (McCracken & Velleman, 2010). The current research provides support for pain acceptance as a resistance factor associated with improved physical function in a heterogeneous community-based sample of individuals with chronic pain.

A strong and positive association was noted between pain acceptance and QOL in the current research expanding the currently small body of evidence showing that pain acceptance is associated with improved QOL in community-based adults with chronic pain. Despite acknowledgement in multiple qualitative studies that pain acceptance is associated with improved

QOL (Bunzli et al., 2013; LaChappelle et al., 2008; Snelgrove & Liossi, 2013), relatively few studies have examined direct associations between acceptance and QOL in cross-sectional data from heterogeneous chronic pain samples. The apparent small number of studies that have explored these links have done so in varying subgroups of pain patients (for example, patients with haemophilia and neurological disorders) with robust positive links generally reported (Elander et al., 2009, 2013; Kratz Hirsh, Ehd & Jensen, 2013; Mason et al., 2008). This finding suggests the potential for improvements in QOL to occur through improving pain acceptance in pain rehabilitation.

That acceptance represents an important resistance factor associated with improved pain outcomes was supported by anecdotal evidence from Study Two, as all participants described acceptance as an important and positive aspect of their adjustment process. Participants described needing to make peace with what they could not achieve because of their pain, suggesting that acceptance was at least a partial feature of their coping armoury. Sally reported that acceptance of pain helped her re-engage with life.

> *Well you can be a victim …. but it's not going to change it, it's still going to be there… So you can wallow in self-pity or you can accept the fact that this is the way it's going to be. So I suppose it's almost like going through a grieving period, and you can go 'Right, I can sit around the house and not get on with my life' or 'I can get out there and enjoy myself and get on with it and to do that, well this is what I need to do.*

Similarly, Jane identified that acceptance of pain represented a turning point in her recovery as this was linked to her being able to recommence exercising. "*It was just focusing on my body and moving it. I don't know, just makes you feel like you're doing something. (I realized) I'm going to be in pain whether or not I do this, or not do this*".

Bettina reported that acceptance was linked to getting a diagnosis. She was also clear that acceptance did not mean giving up and was in fact linked to optimism.

> *I'll try and accept that I've got this banner now and I sit under it but I can … move beyond it … I'm just going to have to make positive, informed choices about how I will treat the condition and in so doing be able to take on more normal things again*.

Acceptance in general is defined as a 'willingness to experience difficult thoughts, feelings, body sensations, and memories without defense' (Thompson & McCracken, 2011). In the context of chronic pain, pain acceptance refers to acceptance of the internal experience of pain and a simultaneous willingness to engage in normal life activities despite pain (McCracken et al., 2004). Various mechanisms have been suggested for how pain acceptance may promote both reduced pain-related disability and improved QOL. According to McCracken, Carson, Eccleston and Keefe (2004), pain acceptance incorporates a continued engagement with valued life goals and is not a passive process of surrender. Thus, pain acceptance may facilitate coping that is focused on engagement in valued activities rather than on pain reduction (Lauwerier, van Damme, Goubert, Paemeleire, Devulder & Crombez, 2012). This may explain the strong links generally reported between pain acceptance and pain-related physical function.

9.4.2.5 Direct effects of values-based living on adjustment.

The desire of several participants in Study Two to play an active role in the lives of others was broadly interpreted as motivation to live in accordance with values. Thus, the inclusion of values-based living as a resistance predictor in Study Three was supported by anecdotal evidence as well as the existing literature. For example, Jacqui, one of the Study Two participants described this motivation as "*…the kick up the backside that I needed. I wasn't going to let pain… ruin a*

relationship with my Mum and my kids. They deserved better, so that was probably the incentive I suppose, to get up and have a go."

Values-based living demonstrated a strong and statistically significant correlation in the anticipated direction with pain-related disability. This is an important but not new finding, as values awareness has been previously reported to be strongly negatively associated with pain-related disability in pain clinic (McCracken & Yang, 2006; Vowles & McCracken, 2008; Vowles et al., 2011) and primary care samples (McCracken & Velleman, 2010). Further support for the protective effects of values on pain-related function can be found in research showing that the addition of values based interventions to other pain rehabilitation approaches reduced pain-related disability (Branstetter-Rost, Cushing & Douleh, 2009; McCracken & Keogh, 2009).

Values-based living also demonstrated a strong and positive correlation with QOL in Study Three. This finding adds to a limited body of research that has explored links between values-based living and QOL for those living with chronic pain. However, it is consistent with McCracken & Yang (2006) who also reported strong positive correlations between values and measures of health and vitality in 98 Singaporean health workers. The current research indicates that greater awareness of values and commitment to living in accordance with them confers QOL benefits for community-based individuals living with chronic pain. It suggests that targeting values-awareness in rehabilitation interventions might confer benefits not only for physical function but also for QOL. Further research is required to explore whether these findings generalise to other chronic pain samples.

9.4.3 Resistance factors, summary.

The hypotheses of the conceptual model that resistance factors would be directly related to adjustment were supported. Several unique contributions to the existing literature in relation to

resistance factors were noted. Firstly, the finding that pain self-efficacy offers protective effects for QOL adds to the currently small body of literature that has examined links between these two factors in community-based samples of adults with chronic pain. Secondly, the positive bivariate associations observed between pain acceptance, values and PA with QOL enhances current understanding of the links between resistance factors and positive adjustment outcomes. The next section examines the predictive utility of the combined risk and resistance models.

9.5 Risk-Resistance Models

9.5.1 Risk-resistance models predicting pain-related disability.

The hypothesis that the risk-resistance models would explain significant variation in adjustment was supported. As hypothesised, the resistance factors also explained additional variation in adjustment over and above that explained by the risk factors. In Study One, the resistance factors explained 13% of the variance in disability over that explained by the risk-only model. In Study Three, the resistance factors accounted for a significant 19% of the variance in pain-related disability over and above that accounted for by the risk factors. These findings are consistent with risk-resistance models in the developmental literature (Greenberg et al, 2001), in paediatric disability (Wallander & Varni, 1998, McLean et al., 2004) and in chronic pain (Wright et al., 2008). These results highlight that in treatment, there is a need to strengthen resistance factors at the same time that reduction of risk factors is addressed.

Considerable discordance exists in the large volume of literature that has examined multivariate predictors of pain-related disability using cross-sectional data. Multiple factors likely account for these differences including variations in predictors, measures and samples. Systematic reviews of longitudinal research examining predictors of neck and back pain demonstrated the highest level of evidence for the influence of affective (anxiety, depression) and cognitive

(catastrophising, fear-avoidance) risk factors in both the development and maintenance of pain-related disability (Linton, 2000). In a review of cognitive and social factors associated with pain-related disability in people with disabilities (Jensen et al., 2011), factors most strongly associated with pain-related disability were catastrophising, passive coping, perceived social support and solicitous partner responses.

Partly consistent with these findings, significant predictors in the Study One risk-resistance model predicting pain-related disability were pain severity, solicitous and punishing partner responses to pain and pain self-efficacy. When anxiety and depression were combined into a single variable and the model re-tested, this combined risk factor also became a significant predictor of disability. That NA was a significant predictor of disability in the multivariate context reflects other research in pain clinic samples that has identified depression or NA as a significant predictor of pain-related disability, even after adjusting for the effects of other robust predictors such as pain intensity and catastrophising (Borsbo et al., 2009; Nicholas et al., 2009).

It was of interest that despite the links observed between catastrophising and disability at the bivariate level, catastrophising was not a significant predictor of disability in Study One, after adjusting for the effects of pain severity and NA. This finding contrasts with previous research in both pain clinic and primary care samples that has emphasised the importance for physical function of catastrophic interpretations of chronic pain even after adjusting for the influence of depression (Arnow et al., 2011; Bergbom et al., 2011; Linton et al., 2011; Sullivan et al., 1998). The current results instead indicated that in this sample of relatively pain-disabled individuals, NA was a stronger determinant of disability than catastrophising.

Neither catastrophising nor fear-avoidance were significant predictors of disability in Study Three. This finding may have been influenced at least in part by the diagnostic profile of the sample

as it is possible that cognitive risk factors are strong predictors of disability in people with low back and neck pain, but are less strongly associated with disability in more diffuse pain syndromes such as fibromyalgia which had relatively high prevalence in Study Three. Some support for this idea can be found in a study by de Gier, Peters and Vlaeyen (2003) that found pain intensity but not fear of pain predicted duration of task tolerance in a sample of 81 women with fibromyalgia. The current results may also reflect Wideman and colleagues' (2013) suggestion that avoidance of movement represents only one of a range of different activity patterns and coping strategies that people use to manage chronic pain. Further research is needed to elucidate factors associated with disability in specific diagnostic groups of community-based individuals with chronic pain.

Although some research in community-based samples has demonstrated that catastrophising, not fear-avoidance, was associated with pain-related disability in multivariate models that included both predictors (Somers et al., 2009; Thompson et al., 2010) neither of these studies also included negative affect as a predictor. It is highly likely the degree of shared variance between these measures contributed to the current findings as moderate strength positive correlations were observed between these variables. It is possible that the predictive capacity of fear-avoidance and catastrophising in the multivariate context may have been stronger had these two constructs been combined into a single risk factor representative of pain-related fear. Such questions could be explored in subsequent research.

In Study Three, after controlling for the effects of age, only two variables were significant predictors of disability; pain severity and pain self-efficacy. To explore potential effects of collinearity, additional models were specified. Significant predictors in a second model explaining 55% of the variance in pain-related disability were pain severity, solicitous partner responses and pain acceptance. However, when all non-statistically significant predictors were removed, only pain severity and pain acceptance remained significant. This latter model explained 52% of the

variance in pain-related disability and regression co-efficients were all in the hypothesised directions. In this latter model, pain acceptance contributed 17% unique variance to pain-related disability, over and above that explained by pain severity. These findings suggest that in a sample of individuals less disabled by pain than typical pain clinic samples, perceived pain severity and either pain self-efficacy or pain acceptance are critical determinants of pain-related disability and therefore represent important therapeutic targets. The current findings in relation to pain self-efficacy are consistent with the results of Jackson and colleagues' (2014) meta-analysis in which the adjusted overall effect size for the relationship between self-efficacy and pain-related functional impairment was reported to be larger than the other reported meta-analytic effect sizes for relations between fear-avoidance beliefs and threat appraisals of pain and disability.

Despite that the psychological processes associated with pain self-efficacy and pain acceptance are quite different (Nicholas & Asghari, 2006), the current findings nonetheless highlight that in the current sample disability was determined by the extent to which individuals are able to engage with life activities despite the perceived intensity of their pain. Pain acceptance was a significant predictor of pain-related disability only in models where pain self-efficacy was not a predictor. This may again reflect issues of shared variance and partly concurs with results reported by Nicholas and Asghari (2006) who found that only the activities engagement subscale of the Chronic Pain Acceptance Questionnaire (McCracken et al., 2004) predicted disability after adjusting for the effects of pain self-efficacy. Nonetheless, the strong association between acceptance and pain-related disability in regression models that did not also include pain self-efficacy highlights the importance of pain acceptance as a protective factor for improved pain-related function.

In the community-based sample, values-based living did not explain any additional variance in the measure of disability after the effects of either pain self-efficacy or pain acceptance were taken into account. This finding contrasts with previous research in pain clinic (McCracken & Yang, 2006; Vowles et al., 2014) and primary care samples (McCracken & Velleman, 2010) that showed values was a significant predictor of pain-related disability even after adjusting for the effects of pain acceptance. The reasons for this discrepancy are unclear but may have related to sampling characteristics or to the different measure of values used in the current research.

Despite the weak bivariate links noted between partner responses and disability in both studies, solicitous partner responses to pain were a significant predictor of pain-related disability in the Study One risk-resistance model and a weak significant risk factor in a model predicting disability in Study Three. Previous research has identified a positive association between spousal responses to pain and functional outcomes (Newton-John, 2002, Raichle, Romano & Jensen, 2011, Romano et al., 1995, 1992). That solicitous responses accounted for a small amount of unique variance in the measure of pain-related disability in both pain clinic and community-based samples supports the inclusion of partners in pain rehabilitation.

The inclusion of both positive and negative social experiences in the current research adds to a small body of research that has examined the simultaneous effect of positive and negative social support in the chronic pain setting. Mostly this research has explored these effects referencing measures of mental health as the criterion variable (Luger, Cotter & Sherman, 2009, Mavandadi, Sorkum, Rook & Newsom, 2007, Revenson et al., 1991) with findings generally indicating that problematic support has a stronger negative effect on mental health than the benefits conferred by positive social support (Riemsma et al., 2000). Solicitous partner responses to pain but not social engagement was a significant predictor of disability in the multivariate models

predicting disability. This finding reflects results of Stephens, Druley and Zautra (2002) who compared the differential effects of negative support and avoidance coping to social support and active coping in 63 older adults recovering from knee joint replacement surgery. Both types of social support influenced post-operative functioning, but negative social support had a more powerful negative influence on pain-related interference than the benefits conferred from positive support. Further research regarding the simultaneous effects of positive and negative social support on pain adjustment outcomes in varying pain samples may further understanding of the ways in which negative and positive social experiences interact to influence adjustment to chronic pain.

That none of the social-ecological resistance factors were significant predictors of disability in the multivariate context is consistent with previous research that has also found social support did not contribute unique variance to measures of disability once the influence of other cognitive or coping factors were taken into account (Hermsen et al., 2014; López-Martínez et al., 2008). As noted above, these findings may relate at least in part to dual effects of social support in the chronic pain context as well as to a range of other individual factors shown to influence the impact of social support on physical function (Martire et al., 2011; Nezlek & Allen, 2006).

9.5.2 Risk-resistance models predicting quality of life.

Pain severity, catastrophising (in a positive association), NA, pain self-efficacy, pain acceptance, values-based living and emotional social support were all significant predictors of QOL in the risk-resistance model, after adjusting for age. These combined predictors accounted for a high 70% of the variance in QOL. The addition of the resistance factors accounted for 26% of the variance in QOL over and above that accounted for by the risk factors. This was a larger proportion of variance than seen in the regression model predicting pain-related disability and

demonstrates the importance of addressing resistance factors in pain treatment when aiming to improve QOL.

Much less research has investigated multivariate models predicting QOL in individuals with chronic pain compared to the research that has examined multivariate predictors of pain-related disability. Nonetheless, the current findings are consistent with previous research that has identified both pain severity and NA as significant predictors of QOL in community based samples of individuals living with chronic pain (Agar-Wilson & Jackson, 2012; Merlijn et al., 2006; Moore et al., 2010). The current results suggest that reduction of pain severity in heterogeneous groups of community based pain patients could be expected to provide improvements in QOL. The results also suggest that strengthening resistance factors, such as pain acceptance, self-efficacy and values-based living, in pain rehabilitation might confer additional treatment benefits over adjustment benefits obtained by reduction of risk factors.

In the multivariate context, catastrophising was a significant predictor of QOL but the regression co-efficient demonstrated a reversed sign. A positive regression weight for catastrophising was only observed in a model that contained neither pain acceptance nor pain self-efficacy. These results may indicate that catastrophising exerted a positive influence on QOL after adjusting for the effects of pain self-efficacy and pain acceptance, however, it is likely that the reversal of sign on this regression co-efficient may have reflected collinearity issues within the model (Ludlow & Klein, 2014). This issue is discussed in detail below. The links between catastrophising and QOL in the multivariate context were weaker than those observed between NA and QOL. This suggests it may be more important to address low mood rather than catastrophising when aiming to improve QOL for community-based individuals with chronic pain.

Both pain self-efficacy and pain acceptance were significant predictors of QOL in the multivariate model. These results are consistent with a relatively small body of research that has

examined links between pain self-efficacy, pain acceptance and QOL in multivariate models (Börsbo et al., 2010; Libby & Glenwick, 2010; Mason et al., 2008). The initial regression model suggests that pain self-efficacy and pain acceptance represent independent significant resistance factors associated with improved QOL for those with pain, however due to potential effects of multicollinearity within this model further research is needed to see if these results are replicated in other samples or using other statistical approaches better able to account for the high degree of shared variance between these measures.

Values-based living was a robust predictor of QOL in the multivariate context, even after adjusting for the effects of pain self-efficacy and pain acceptance. Very little research appears to have explored associations between values-based living and QOL for those living with chronic pain although McCracken and Yang (2008) reported strong positive correlations between these measures in a sample of 98 non-pain affected Singaporean health workers. The current findings support the idea that values-based interventions may be especially important for improving overall QOL in community-based individuals with chronic pain.

Emotional social support was also a significant resistance factor in the initial and final risk-resistance models predicting QOL. This finding was perhaps not surprising given that the QOLS assessed satisfaction with relationships but still highlights the influence of positive social support on overall QOL. It suggests that although social support may not provide a protective effect on pain-related function, it remains an important protective factor for QOL generally for those living with pain. None of the social-ecological risk factors were significant predictors of QOL in the multivariate context, a finding that contrasts somewhat with other research that has found negative social support had a more powerful negative influence on physical function and mental health than the benefits conferred from positive support (Luger, et al., 2009, Mavandadi et al., 2007a; Revenson et al., 1991; Stephens et al., 2002). The current results suggest instead that when

considering QOL, positive social experiences confer greater advantage than the influence of negative social interactions.

Neither PA nor optimism were significant predictors in the multivariate models predicting disability or QOL, reflecting the weaker bivariate associations of these constructs compared to other risk and resistance factors in the model. Although there is evidence in some research that optimism and PA are associated with improved physical function and QOL for those with chronic pain (Brenes, 2002; Cruz-Almeida et al., 2013; Seebach et al., 2012; Treharne et al., 2005; 2007; White et al., 2012) the current findings suggest that other resistance factors may represent more critical therapeutic targets.

Despite not violating recommended thresholds for multicollinearity, the reversal of signs of regression co-efficients in most of the regression models was suggestive of issues with collinearity (Mason & Perreault, 1991). These effects were most notable in models that contained pain self-efficacy as a predictor. This was not unexpected as the regression models contained a number of predictors with substantial shared variance. A high degree of shared variance was also noted between several model predictors and adjustment outcomes, most notably between pain self-efficacy and both pain-related disability and QOL. Tu and colleagues (2008) described statistical suppression effects in studies in which the predictor and outcome measures are continuous, when the association between two variables can be reversed, diminished, or enhanced when another variable is statistically controlled. It was unclear in the current research if the reversal of signs of regression co-efficients noted was attributable to a suppressor effect or due to collinearity. In a discussion of issues related to these effects in regression, Ludlow and Klein (2014) advised that is often no simple way to identify the variable causing the reversal effect. These authors stated that in the setting of multicollinearity, overall model prediction is generally reliable, but the accuracy of the regression coefficients and their standard errors can be affected and the chance of Type II

errors inflated (Mason & Perreault, 1991). That is, the chance that a significant predictor fails to be identified is increased (Mason & Perreault, 1991). In these situations, Ludlow and Klein (2014) recommend the best approach is to conclude the results were unexpected and uninterpretable, and occurred simply as a result of the collinear relationships existing within the predictor correlation matrix. Future research aiming to explore these relationships may benefit from alternative statistical approaches that establish discriminant validity of the measures.

9.6 Mediator effects

One of the aims of the current research was to explore the inter-relatedness of the risk and resistance factors in the model in order to further understand mechanisms through which pain severity, intrapersonal and social-ecological risk and resistance factors may influence pain adjustment outcomes. The hypothesis of the conceptual model that stress-processing variables, both risk and resistance, would mediate these relationships was supported in both single and parallel mediator models. Findings in relation to single mediator models are discussed first, followed by results of parallel mediator models.

9.6.1 Risk stress-processing factors as mediators in single models predicting disability and quality of life.

In Study One, pain severity, depression, anxiety and social engagement were all associated indirectly with pain-related disability via an association with either increased or decreased catastrophising. In Study Three pain severity, NA and PA were associated with pain-related disability via positive and negative associations with both catastrophising and fear-avoidance. Pain severity, NA and PA, optimism and social support were also associated indirectly with higher or lower perceived QOL through an association with catastrophising. In all models, each predictor

exerted its effects on disability and QOL only partly via the mediator, as the direct paths from predictor to outcome remained statistically significant.

The current findings are in line with previous research that has also demonstrated catastrophising and fear-avoidance as mediators of the chronic pain-disability relationship. For example, catastrophising mediated the relationship between pain severity and pain-related disability in adolescents with chronic musculoskeletal pain (Guite et al., 2011) and between an interaction term of pain and affective distress with disability in individuals with spinal cord injury (Ullrich et al., 2007). Fear-avoidance has also been shown to mediate the pain-disability relationship in injured workers (Kamper et al; 2012) and people with low back pain (Gay et al., 2015). The current findings partly concur with those of Lee and colleagues (2015). Their meta-analysis of 12 studies that explored mediators of the pain-disability relationship in adults with neck and back pain concluded that pain self-efficacy, psychological distress and fear-avoidance all mediated the pain-disability relationship but catastrophising did not.

Despite that only a relatively small amount of research has formally examined mediation of NA by catastrophising, strong bivariate links have previously been identified between these constructs (Börsbo et al., 2010; Sullivan & D'Eon, 1990). Fear-avoidance has been shown to mediate the relationship between NA and disability in 890 employees who had reported at least one day of back pain during the previous year (Gheldhof et al., 2006). Therefore the current finding that both fear-avoidance and catastrophising mediated this relationship in a community-based sample of pain-affected adults expands the literature and highlights that NA is not only directly associated with pain-related disability but is also associated indirectly via its effects on negative pain-related cognitions. Clearly, causality cannot be established from the cross-sectional data used but the direction of effects suggested by these current models would be worthy of exploration in subsequent research that can examine the temporal nature of the links.

Indirect effects of PA on disability via decreased catastrophising and fear-avoidance were also identified in the Study Three sample. Positive affect and optimism also influenced QOL through an association with decreased catastrophising. These findings suggest that positive psychological states may influence pain adjustment outcomes by decreasing negative or fearful pain-related cognitions. Although the existing body of research exploring formal mediation in these settings is currently very small, existing theories, such as Fredrikson's (2004) Broaden and Build model, that suggests positive emotions produce more broad-ranging thoughts and behaviours not geared to safety or survival, provide support for the identified paths.

That measures of social support were associated with pain related disability and QOL partly via an association with decreased catastrophising also expands the current literature. Despite that previous links have been identified between social experiences and catastrophising (Giardino, Jensen, Turner, Ehde & Cardenas, 2003; Sullivan et al., 2006; Sullivan et al., 2004; Tremblay & Sullivan, 2010), only one study in individuals with inflammatory bowel disease (Katz et al., 2016) was identified that showed catastrophising mediates relationships between social experiences and adjustment to health conditions. Although some theorists suggested that social support may reinforce expressions of pain and thus increase catastrophising (Savard & Crombez, 2006), more recently it has been noted that social support may be linked to decreased levels of catastrophising (Sullivan, 2012). The current findings support these associations. More research is needed to explore if these associations are replicated in other pain-affected samples.

9.6.2 Resistance stress-processing factors as mediators in single models predicting disability and quality of life.

The hypothesis of the conceptual model that resistance stress-processing factors would mediate relationships between condition parameters, intrapersonal and social-ecological factors and adjustment outcomes was also supported. In Study One, pain severity, anxiety, and depression

were all associated with pain-related disability indirectly partly via an association with decreased pain self-efficacy. By contrast, social engagement was indirectly related to lower levels of pain-related disability entirely through an association with increased pain self-efficacy. In Study Three, pain severity and NA and PA were associated with pain-related disability via positive and negative associations with three resistance stress-processing factors; pain self-efficacy, acceptance and values-based living.

Pain severity, NA and PA, optimism and social support were also all indirectly associated with higher or lower perceived QOL through an association with the three resistance stress-processing factors. All mediation models were statistically significant and explained a small to moderate amount of variance in pain-related disability and QOL. In all but three mediation models, each predictor exerted its effects on disability and QOL only partly via the mediator, as the direct paths from predictor to outcome remained statistically significant in most models. Notably, PA was associated with disability entirely via an association with increased pain self-efficacy. In two separate models, optimism was associated with QOL entirely by an association with increased pain self-efficacy and values-based living.

These findings are consistent with a number of studies that have identified pain self-efficacy as a mediator between risk factors and pain-related disability. For example, the meta-analysis by Lee and colleagues (2015) described above, reported pain self-efficacy was the most consistent mediator of the pain-disability relationship. This effect was demonstrated in Australian primary care patients (Costa et al., 2011) and in individuals with whiplash injuries (Soderlund & Asenlof, 2010). Considering research not included in this meta-analysis, pain self-efficacy has also been shown to mediate the relationship between pain severity and disability in pain clinic samples (Arnstein, 2000), individuals with chronic low back pain (Woby, Urmston & Watson, 2007) and in women with fibromyalgia (Puente et al., 2015). The current findings therefore add to the existing

body of literature supporting pain self-efficacy as a mediator of chronic pain-adjustment relationships and suggest that a consistent pathway through which pain influences disability is through the erosion of confidence to manage pain. As other theorists have noted, there is likely to be a highly reciprocal relationship between pain and self-efficacy (Jackson et al., 2014) which as evidenced by the bivariate associations in the current research, is strongly associated with physical function. As reduction of pain intensity is often difficult in patients requiring pain rehabilitation as physical treatment options are often exhausted, the current finding identifies pain self-efficacy as critical therapeutic target for clinicians working in these settings.

The current findings that the pain-disability relationship was also mediated by pain acceptance reflect previous research that has demonstrated that pain acceptance mediates this relationship in community-based adults with chronic pain (Fish et al., 2013) and between pain and HRQOL in individuals with haemophilia (Elander et al., 2009). Few studies were identified that examined values-based living as a mediator of relationships between pain and NA with pain adjustment outcomes. Much of the research that has examined links between pain, NA, acceptance and values has examined these associations placing perceived pain intensity and psychological distress instead as outcomes. For example, both poor pain acceptance and difficulty living according to values have both been shown to significantly predict NA (McCracken & Yang, 2006, Vowles et al., 2008) and moderate negative associations have been demonstrated between depression and anxiety with acceptance and values (Fish et al., 2013; McCracken & Vowles, 2008; McCracken & Yang, 2006; Vowles et al., 2008). Nonetheless, it is theoretically feasible that both pain and NA reduce acceptance and an individual's capacity to live according to values with resultant negative impact on disability and QOL. Clearly the current research is unable to establish causative links. Other research approaches such as multi-level modelling would be better placed to explore the temporal links between these constructs.

The current results also extend previous findings pertaining to inter-relations between PA, pain self-efficacy, acceptance and values-based living and adjustment outcomes. The finding that pain self-efficacy, acceptance and values mediated the PA-disability relationship is consistent with a small amount of previous research. For example, Wright and colleagues (2008) showed that pain self-efficacy mediated the relationship between resilience (a latent factor that incorporated measures of extraversion, vitality and PA) and pain-related physical function in 275 patients with osteoarthritis of the knee. Positive associations have also been noted between PA and other positive psychosocial constructs such as self-efficacy (Steptoe et al., 2011). Further research is needed to explore these associations and establish whether the current findings generalise to other pain samples.

The relationship between optimism and adjustment outcomes was mediated by the three resistance stress-processing factors. Positive associations have previously been noted between optimism and pain acceptance in chronic pain (Wright et al., 2011) and chronic illness samples (Edgar, Remmer, Rosberger & Fournier, 2000; King, Rowe, Kimble & Zerwic, 1998). Although no previous research was identified that specifically explored pain self-efficacy, acceptance or values as mediators of optimism-disability or optimism-QOL relationships, conceptually similar findings were identified in a sample of 161 older, community based individuals with knee osteoarthritis. In this study, mastery beliefs (defined as an internal sense of efficacy) mediated the relationship between optimism and well-being (Sherman & Cotter, 2013). Given that optimism is related to positive expectancies for the future and an enhanced ability to adapt (Goodin & Bulls, 2013), it is possible that higher optimism may promote improved adjustment outcomes by increasing acceptance, pain self-efficacy and values. More research is needed to particularly to explore these potential temporal links given the cross-sectional data used in the current research.

Jane, one of the Study Two participants provided some insights into how these processes may function as she described how optimism for her related to a sense of confidence to manage pain. She said that a sense of hope that her pain might improve promoted her capacity to stick to her management plan. *"I've still got my eyes open for, this might not be forever if I do my stretches and, and I've been doing like, relaxation tapes at night…."*.

Finally, the current results explored the social context of pain self-efficacy, acceptance and values-based living and found that social engagement influenced disability partly by promoting increased pain self-efficacy and experiences of social support were associated with QOL via an association with increased pain self-efficacy, acceptance and values. Although multiple studies have explored associations between social support and self-efficacy in general populations (Cutrona & Troutman, 1986; Holahan & Holahan, 1987; Karademas, 2006) with positive links typically identified, few studies appear to have explored these links in pain samples in recent years. No research was identified that examined mediation of the social support-QOL relationship by self-efficacy. However, anecdotal evidence from Study Two provided some insights into how these constructs might inter-relate. For example, Sally, described that her family promoted an ability to keep going, which may have partly reflected increased self-efficacy for managing pain: *"I think it keeps away the 'woe is me' attitude. Because we don't let one another get low. And if someone did go there, we would be told to 'Pull our head in, get on with it'*. Michael also recognised that his capacity to cope came partly from a learned attitude from his family. *"I think, I think so, it's just part of the family. It doesn't matter how bad the circumstances you just never give up"*. The current results promote understanding of how social processes might interrelate to promote self-efficacy and subsequently adjustment outcomes.

The current results are also consistent with previous findings in a pain clinic sample that showed that negative social experiences were associated with reduced pain acceptance

(McCracken, 2005). Links between social support and increased pain acceptance have also been identified in qualitative research (Kostova, Caiata-Zufferey & Schulz, 2014; LaChapelle et al., 2008). Study Two participants again provided anecdotal insight into these relationships. For example, Jacqui said that close family relationships were linked to her strongly valuing her role as a mother and daughter which in turn seemed to be linked to better function.

So that was probably the kick up the backside that I needed. I wasn't going to let pain… ruin a relationship with my Mum and my kids. They deserved better, so that was probably the incentive I suppose, to get up and have a go. (Jacqui, aged 45)

Clearly more research is needed to explore these links and their generalisability to other samples, especially given the identified complexities within the construct of social support in the context of chronic pain.

9.6.3 Parallel mediation models predicting disability.

In Study One when the simultaneous mediating effects of catastrophising were compared to those of pain self-efficacy, the relationships between pain severity, anxiety and depression and pain-related disability were mediated only by pain self-efficacy. The relationship between social engagement and pain related disability on the other hand was mediated by both self-efficacy and catastrophising, although the indirect effect via self-efficacy was significantly larger than that via catsstrophising. In the community-based sample in Study Three, when all five stress-processing factors were examined as simultaneous mediators of the relationships between pain severity and negative and PA and pain-related disability, only pain self-efficacy was a significant mediator.

Few studies have examined mediation in the context of chronic pain using parallel models however the current results support those of Costa and colleagues (2011) who compared fear-avoidance and self-efficacy as mediators of the relationship between pain severity and disability in

patients with chronic low back pain. Although both self-efficacy and fear-avoidance partially mediated this relationship at the time of pain onset, only improvements in self-efficacy mediated the relationship between changes in pain and changes in disability over a 12 month period. The current results are also consistent with those of Turner and colleagues (2006) who examined simultaneous mediators of treatment-related change in a cognitive-behavioural intervention for people with chronic jaw pain. In simple mediation models, a number of factors that included catastrophising, perceived ability to control pain and pain self-efficacy mediated treatment-related change. However, when analysed together in parallel models, of these mediators only self-efficacy was significant. The current findings suggest that pain self-efficacy represents a key mechanism through which pain severity and negative and PA are linked with pain-related disability.

9.6.4 Parallel mediation models predicting quality of life.

In parallel mediator models predicting QOL in Study Three, pain severity, PA, optimism and social support were associated with QOL indirectly via pain self-efficacy, pain acceptance and the degree to which they were able to live according to their values but not through an association with catastrophising. The indirect effects via pain self-efficacy and values-based living were significantly larger than those via pain acceptance. The relationship between NA and QOL was mediated by all four tested stress-processing factors but indirect effects were larger via pain self-efficacy and values than they were via pain acceptance. No previous research appears to have explored mediation of relationships between risk or resistance factors and QOL via simultaneous mediators. Therefore, the current findings provide initial support for pain self-efficacy and values-based living as comparatively important mechanisms through which pain severity, PA, optimism and social support are associated with QOL in those with chronic pain. More research is needed to explore similar models in other samples.

Taken together, results of all the mediation analyses suggest that pain severity, intrapersonal and social-ecological factors affect pain-related disability through a range of different pain appraisal and coping mechanisms. However, of these, indirect effects via pain self-efficacy appears to be strongest. These same predictors were associated with QOL through a range of different pain appraisal and coping mechanisms but of these, indirect effects via pain self-efficacy and values-based living were the strongest. Although it is unclear the extent to which these findings are generalizable beyond the included sample, the current results suggest that self-efficacy and values are key mechanisms through which the effects of various risk and resistance factors are associated with adjustment outcomes. This highlights the relative importance of resistance factors for both positive and negative adjustment outcomes.

Some interpretive difficulties were encountered due to the effects of collinearity within the parallel mediator models that arose due to some variables being highly correlated. This issue was most notable for the relationship between pain self-efficacy and pain acceptance and between pain self-efficacy and pain-related disability. Collinearity issues were evidenced by reversal of co-efficients of the indirect effects of risk and resistance factors on pain-related disability when mediated by catastrophising, fear-avoidance and values-based living. Reversal of some regression co-efficients of the paths from the mediators to both pain-related disability and QOL were also noted. In most cases where co-efficients were reversed, the indirect effects were small and not statistically significant. However these issues mean that the current models are best considered as exploratory and requiring further testing and refinement. Nonetheless, they offer insights into the importance of the resistance stress-processing factors in the conceptual model, both as direct protective influences and as mechanisms through which other factors appear to influence outcomes. In future research, mediation analyses could be undertaken using statistical approaches better able to minimise shared variance, such as structural equations modelling.

Some further interpretative caveats apply to the mediation models in the current research. The intention of these models was to broaden current conceptualisations of pain adjustment processes, not to decouple constructs whose theoretical links may have important implications for pain rehabilitation. These issues were highlighted by McCracken (2013) who noted that while integration of constructs from varying paradigms offers pain research the opportunity to broaden understanding of adjustment processes it relies on explicit understanding of the underlying assumptions of the involved constructs. For example, addressing values in pain rehabilitation without also addressing pain acceptance could potentially lead to worsened adjustment outcomes as some research has suggested that treatment-related changes in acceptance may promote a later capacity to use values to guide behaviour (Vowles & McCracken, 2008). Further, some research has noted that excessive task persistence in the setting where this is difficult can contribute to low mood (Brandtstädter & Renner, 1990; Crombez, Eccleston, Van Damme, Vlaeyen & Karoly, 2012).

9.7 Moderator effects

A final aim of the current research was to explore whether the relationships between risk factors and adjustment outcomes was moderated by the resistance factors in the model. Moderation analyses determine whether the strength of the relationship between a predictor and an outcome varies according to the level of the moderator. Significant moderation effects have implications for treatment as they identify which individuals might be particularly at risk. The moderator hypothesis was partially supported but only in Study Three.

9.7.1 Positive affect.

It was expected that PA would buffer risk-adjustment relationships in the model as previous research has shown it acted as a moderator in conceptually similar relationships. For example,

multi-level modelling showed that the weekly association between pain and NA was attenuated when levels of PA were high in 124 patients with fibromyalgia and osteoarthritis (Zautra, Johnson & Davis et al., 2005) and may reduce generalisability of fear of pain from painful to non- painful stimuli (Meulders et al, 2014). The idea that PA may buffer the effect of a stressor on adjustment is also consistent with Fredrikson's Broaden and Build theory of positive emotions (Fredrikson, 1998) in which positive affective states are seen as significant resources for well-being.

Despite the previous data supporting this contention, PA did not moderate any risk-adjustment relationships in Study Three. It is possible these findings reflect characteristics specific to the current sample. Approximately one third of participants in Study Three reported a diagnosis of fibromyalgia, a condition reported to be associated with relative deficits in PA (Davis, Zautra & Reich, 2001; Zautra, Hamilton & Burke, 1999). Participants in Study Three reported a mid-range mean score for PA on the short form of the PANAS. Normative data for this scale is not available which makes interpretation of this finding difficult.

As Finan & Garland (2015) pointed out, it is also possible that moderating effects of PA on risk-adjustment relationships may vary according to the temporal dynamics of measurement of PA as some affirmative moderating results that have been noted in multi-level modelling have not been replicated in cross-sectional research. Exploration of potential moderating effects of PA on risk-adjustment relationships in other heterogeneous or diagnosis specific samples of individuals with chronic pain represents an area for further research.

9.7.2 Optimism.

The idea that optimism may moderate or buffer risk-adjustment relationships is consistent with two major theories of stress and coping (Hobfoll, 1989; Lazarus & Folkman, 1984) that propose individual differences may influence stressor appraisals and the extent to which an event

is perceived as stressful. The current finding in Study Three that optimism did not moderate risk-adjustment relationships contrasts with other research that has established optimism as a moderator of risk-adjustment relationships in college students (Hirsch et al., 2007), in chronic disease samples (Hurt et al., 2014) and in those with chronic pain (Cannella et al., 2007). Other research has also identified a mediating role of optimism on the relationship between pain and QOL in Chinese individuals with lung cancer (Wong & Fielding, 2007). Further research is required to determine interactions between optimism and risk-adjustment relationships for those with chronic pain.

9.7.3 Instrumental and emotional social support.

The finding that social support did not moderate the risk-outcome relationships in the current research contrasts with research in general populations that has established social support as a moderator of risk-adjustment relationships (Cohen & Wills, 1985; Cohen & Hoberman, 1983; Thoits, 1995). Very little research appears to have examined a moderating effect of social support on risk-adjustment relationships for those with chronic pain, however, the current findings contrast with those of Stark-Taylor and colleagues (2013) who found a happy marriage dampened the negative relationship between changes in pain and changes in disability in women with osteoarthritis and / or fibromyalgia. The current findings also contrast with research that has found social support attenuated the negative relationship between functional impairment and depression (Benka et al., 2014), pain and negative mood (Feldman et al., 1999) and negative social experiences and depression (Riemsma et al., 2000).

The current findings are consistent though with other research that has failed to identify significant moderating effects of social support on the relationship between pain and depression (Pjanic et al., 2014) and on the relationship between disability and depression (Doeglas et al., 2004). It is possible the mixed findings in relation to a potential buffering effect of social support

on risk-adjustment relationships for those with chronic pain relate to differing effects of social support in the context of chronic pain and its capacity to act as a social resource as well as a reinforcer of pain behaviours. More research is needed to clarify these effects in other samples of individuals with chronic pain.

9.7.4 Pain self-efficacy.

The conceptual model proposed moderator effects where the relationship between the risk factors and adjustment varies with, or is moderated by, levels of the resistance factors. This idea is consistent with Rose and colleagues (2004) who suggested that resistance factors can act as moderators that specify the conditions under which a risk factor exerts a negative influence. Therefore, pain self-efficacy, pain acceptance and values-based living were all tested as potential moderators of risk-adjustment relationships.

No significant interaction effects of pain self-efficacy were identified in either sample. This finding contrasts with that of Lowe and colleagues (2008) who found that acceptance coping was associated with lowered depression only in individuals reporting high self-efficacy beliefs. The lack of significant effect in the current research also contrasts with research from non-chronic pain fields that has demonstrated a buffering effect of self-efficacy on the relationship between stressor appraisals and QOL (Prati et al., 2010). The reasons pain self-efficacy did not moderate risk adjustment relationships in the current research are unclear as it is theoretically feasible that risk-disability or risk-QOL relationships may vary in strength according to level of pain self-efficacy. It is possible that sampling characteristics may have influenced these findings, for example the low overall level of pain self-efficacy reported in the pain clinic sample. It is also possible that measurement factors may have influenced the current results. For example, Peng, Schaubroeck and Xie, (2015) showed that intra-individual variations in self-efficacy as opposed to a single measure

buffered the negative relationship between job demands and psychological distress. Subsequent research may further understanding of potential moderating effects of pain self-efficacy in the context of chronic pain.

9.7.5 Pain acceptance.

The current research found that overall, those with high pain acceptance had the lowest levels of pain-related disability, however the negative relationships between both pain severity and NA with pain-related disability were stronger for those reporting high acceptance compared to those reporting low acceptance. This finding contrasts with previous research demonstrating that pain acceptance attenuated the relationship between pain and physical HRQOL in men with haemophilia (Elander et al., 2009) and the relationship between catastrophising and physical task performance in individuals with chronic low back pain (Richardson et al., 2010). The current finding indicates that for individuals who are relatively disabled and low in acceptance, increases in pain and negative affect do not negatively impact physical function to the same extent as they do for less disabled individuals. This suggests that in the setting of high disability and low acceptance, the impact of incremental increases of pain or negative affect on disability levels is lessened, possibly because of the already high level of disability in these individuals. One extension of this finding might be that interventions addressing, for example negative affect, would be expected to yield less benefit for more disabled individuals with low acceptance however it is possible even small changes in reported negative affect in relatively disabled individuals may translate into clinically meaningful change in disability. As pain acceptance as not available to examine as a moderator in the Study One sample, it is unclear whether these effects are specific to better functioning groups. This represents an opportunity for subsequent research.

It is possible that the noted moderation effects of pain acceptance may vary according to what aspects of pain acceptance are assessed. The Chronic Pain Acceptance Questionnaire (McCracken et al., 2004) consists of two factors assessed in separate subscales that relate to distinct aspects of the process of pain acceptance; the willingness to accept pain and the extent to which a person is engaged in life activities regardless of pain (McCracken et al., 2004). These potential effects were not explored in the current research due to the large number of effects proposed by the model but pose interesting questions for subsequent research.

9.7.6 Values-based living.

A similar finding was noted in relation to the moderating effects of values-based living on the negative relationship between NA and disability. As with pain acceptance, the current results indicated that people reporting low levels of values awareness were more disabled overall, however the strength of the relationship between NA and disability was strongest for people reporting high levels of values-awareness. Implications of this finding are similar to that described above. That is, in the setting of high disability and low values, the impact of incremental increases of pain or negative affect on disability levels is lessened, possibly because of the already high level of disability in these individuals. No other research appears to have explored whether values-based living moderates risk-adjustment relationships for those with chronic pain. Further research could explore differential moderating effects of values by different aspects of this construct, for example values awareness compared to values living. It is also important to note that interpretive caveats apply to the findings in relation to moderation of risk-disability relationships as although the identified interactions were statistically significant, the overall explained variance attributable to the interactions was very small.

9.8 Strengths, Limitations and Implications for Further Research

9.8.1 Strengths and limitations.

The current research investigated the direct, mediating and moderating effects of a range of psychosocial factors on pain-related disability and QOL, using cross sectional data obtained from two different samples of individuals living with chronic pain. A major strength of the research was the use of a theoretical framework that facilitated inclusion of a range of risk and resistance predictors, drawn from varying theoretical paradigms and ecological levels. Predictors included stress-processing factors (individual), intrapersonal factors such as NA, PA and optimism (individual), partner and family reactions to pain (family) and degree of social engagement and social support (friends, family and community). By investigating this inclusive theory-driven model, this research contributes to the research that has investigated risk-resistance models as predictors of pain-related disability as well as to the much smaller body of research that has examined predictors of QOL for those with chronic pain.

In order to account more comprehensively for the variations in adjustment for those living with chronic pain, there is a need for research to occur within a broad framework that considers cognitive, affective and social factors (Yeung et al., 2012). Therefore, consideration of the inter-relatedness of the risk and resistance factors by examining mediator and moderator effects and incorporation of a range of predictors from varying theoretical paradigms represents a particular strength of the research. This is because each paradigm alone represents only a subset of variables that may influence outcomes (Jensen, 2011).

Other study strengths include the large sample sizes and the consideration given to the complexity of the effects of various risk and resistance factors on chronic pain adjustment. Despite variations in the measures used in Study One and Study Three, the use of the same theoretical and

analytical approach facilitated some comparison of risk and resistance factors associated with pain-related disability across these different samples. That the profile of significant predictors varied across samples has clinical implications, suggesting that interventions likely to be helpful in pain clinic settings may not be equally applicable in less disabled groups of pain patients. This represents a further strength of the current research.

The identified significant mediation and moderation effects provided particular insights into the complex ways that risk and resistance factors interact to influence outcomes. Both risk and resistance stress-processing factors were identified as mechanisms through which intrapersonal and social factors indirectly influence adjustment. It was noteworthy that the moderation effects of the resistance factors on the risk factors strengthened rather than attenuated these negative relationships, highlighting that the simple relationships between one predictor and another can become substantially more complicated in the setting of other factors.

Several limitations of this study should be mentioned. This study used only cross-sectional measures and so conclusions cannot be drawn about causality or the changing nature of the relationships between risk and resistance factors and adjustment over time. That is, the findings of both Studies One and Three are not static but instead are likely to change across time. This is a point that has repeatedly been raised as a criticism of chronic pain research. Recent trends toward use of techniques such as daily diary recording and latent growth curve modelling better address this research shortfall by capturing daily variations in pain related experiences and temporal changes in adjustment (Keefe et al., 2004). A further challenge in this field is the myriad of measures currently available to measure risk and resistance predictors and criterion variables as well as the dual use of predictor variables (such as depression or pain severity) as outcome measures (Meredith et al., 2008).

Many of the constructs in the research were closely inter-related and were expected to demonstrate shared variance. This issue has also been previously highlighted in the literature (Campbell, Fisher & Dunn, 2014). Therefore careful consideration was given to the most appropriate analytical approach. A regression-based approach was adopted because the research was exploratory in nature, included a large number of variables, and aimed to test multiple direct and indirect effects. Hayes' (2013) PROCESS macro for SPSS also uses a regression-based approach and use of this macro in the current research offered the opportunity to test relevant indirect effects in unique ways. For example, PROCESS allows simultaneous testing of multiple mediators and facilitates comparison of these indirect effects. This was not possible with other statistical approaches. Additionally, there is considerable debate regarding the most accurate approach to test moderation effects in alternative statistical approaches such as structural equations modelling rendering this approach for the current model challenging (Jose, 2013).

Difficulties interpreting the results because of multicollinearity issues are acknowledged. These issues were anticipated due to the relatedness of the constructs in the research, however inclusion of the range of measures in this study was considered important in order to consider the influence of each of the factors in the context of the others. To reduce issues with multicollinearity, consideration was given to the idea of using exploratory factor analysis to investigate discriminant validity of the measures. The resultant factor scores representing the obtained latent variables could have then been used as the independent variables in the regression analyses. However, this approach was rejected on several grounds. Firstly, this approach did not reflect the overall aims of the research which were to explore direct, indirect and interactive associations of a number of established constructs with pain adjustment outcomes. Secondly, issues have been identified with factor scores in regression analyses that suggested their use in the current research may be problematic. Factor scores are linear combinations of the observed variables that take into account

the shared as well as error variance and are sensitive to the factor extraction and rotation method used (DiStefano, Zhu & Mindrila, 2009). According to DiStefano and colleagues (2009) this leads to the problem of indeterminancy, or the fact that there are an infinite number of possible solutions that could account for the relationships between the test items and factors producing problems with interpretation of results. Subsequent research that aimed to test only subsets of the relationships proposed by the current model may benefit from use of statistical approaches that establish discriminant validity of the measures prior to model testing and reduce measurement error, such as structural equations modelling. Issues related to the large number of tests undertaken in the current research are also acknowledged as this may have inflated the Type 1 error rate. This issue was partly addressed by using bootstrapped confidence intervals to test significance of indirect effects in place of alpha levels. This increased confidence in the reliability of the estimates.

Use of pre-collected data limited the choice of predictors available for the first study. Several other demographic variables such as compensability or education level that may have influenced the adjustment outcome in Study One were not able to be included in the models because these data were not reliably recorded in the clinical dataset. Due to the small number of Study Three participants who reported compensable pain conditions and the small effect size noted of this risk factor on adjustment outcomes, the effect of compensability on adjustment was not analysed in Study Three. However, as evidence exists to demonstrate compensability is a risk factor for poorer adjustment in chronic pain (Harris et al., 2005; Rohling et al., 1995), this represents an area for further research. Location or type of pain was not included as a predictor in any of the models as the aim of the research was to explore the hypothesised effects in heterogeneous samples of individuals with chronic pain. Some research has identified distinct behavioural and psychosocial profiles in different pain syndromes (Turk, Okifuji, Sinclair, & Starz, 1998; Turk &

Rudy, 1990) suggesting that pain patients can be grouped by psychosocial variables as well as by pathology. As such the current results may not be able to be extended to subgroups of pain patients. However, limiting chronic pain research to only a particular type of pain or diagnosis limits the generalisability of results to groups of patients seen in pain clinics or general practice, where a mix of diagnoses and clinical presentations is the norm.

The use of self-report measures in the current research may have been a source of bias as it has been noted that pain patients may under-report subjective activity levels compared to objective measures (Jensen & Karoly, 1991, Kremer, Block & Gaylor, 1981). Recent research that has used behavioural observations or daily diary approaches that link behaviours to concurrent cognitive and affective experiences may allow improved understanding of some of the mechanisms underlying the interference by pain on daily activities and satisfaction with life. However, the argument that self-report measures lack objectivity must be considered in light of the fact that the current research was primarily interested in the feelings, thoughts and social experiences of people living with chronic pain and self-report measures represent an important method of gaining insight into these subjective experiences.

9.8.2 Implications for future research.

There are several important considerations for future research. More qualitative research of specifically resilient samples of individuals living with chronic pain, using a larger sample size, may shed more light on processes associated with positive adjustment and the relationships between risk and resistance factors and pain adjustment. It is possible other risk or resistance factors not included in the current research may improve the predictive capacity of future risk-resistance models. The use of research methods that better capture the lived experience of chronic pain and temporal variations in risk and resistance factors, such as multi-level modelling, may

further understanding of adjustment processes. This research highlighted that adjustment processes appear to be complex. Alternative analyses of indirect effects (for example mediated moderation or moderated mediation) may help elucidate the complex relationship between psychosocial predictors and adjustment to chronic pain.

Finally, although more recent developments in cognitive-behavioural approaches to chronic pain rehabilitation, such as acceptance and values-driven approaches (McCracken & Morley, 2014), emphasise positive pain adjustment processes, there remains an ongoing focus in pain research and treatment on the importance of reducing fear-based appraisals of pain (Finan & Garland, 2015). The current research demonstrated instead that resistance factors such as pain self-efficacy, pain acceptance and values-living may represent key influences of pain adjustment processes, by acting both directly and as mechanisms through which the effects of other risk and resistance factors are expressed. Of the examined risk factors, pain severity and NA emerged as the strongest influences of both pain-related disability and QOL. Despite that more novel resistance factors such as PA and optimism were not significant adjustment predictors in the multivariate setting, their moderate strength bivariate associations with QOL suggests these factors may promote an ability to live well with chronic pain in community-based individuals. As the effect sizes associated with current psychological interventions for chronic pain remain modest (Morley, Williams & Eccleston, 2013) it is possible that the integration of novel resistance factors into rehabilitation interventions may yield adjustment benefits for those with chronic pain. The inter-relationships between risk and resistance factors identify targets for possible points of intervention as well as opportunities for further research.

9.9 Conclusion

Wallander and Varni's (1998; Wallander et al., 1989) generic risk and resistance model provided a comprehensive, theory-driven approach for integrating a range of risk and resistance factors identified as relevant to adjustment processes in chronic pain. The models tested in the current research explained a moderate to large proportion of variance in the selected outcomes, which demonstrates the utility of a risk-resistance approach for predicting chronic pain adjustment outcomes. This research adds to existing literature not just by exploring the direct effects of a range of intra and inter-individual adjustment predictors within a multivariate model, but also by investigating mediation and moderation effects.

As such the current research represents some of the first in the field to integrate cognitive, affective and social factors into a comprehensive framework predicting adjustment to chronic pain. Self-efficacy was strongly associated with disability and mediated the relationships between most examined risk and resistance factors with disability and QOL. Moderation analyses provided insights into the ways in which resistance factors can influence the relationship between pain severity, NA and disability.

The current research makes several valuable contributions to the chronic pain literature: (a) Despite its small sample size, the qualitative component of the research appears to be the first investigation of adjustment processes specifically in a group of individuals that judged to be coping well with pain; (b) the qualitative research identified a protective factor not previously described in the chronic pain literature, the effects of caring for others. Broadly, this was interpreted as a protective effect of being motivated to behave in ways that were aligned with values; (c) the current research highlights the usefulness of Wallander's risk and resistance model in identifying the direct, moderating and mediating effects of psychosocial factors on adjustment to chronic pain, using cross-sectional data and (d) Wallander's model provides a theory driven approach for integrating

a range of risk, but also importantly resistance factors, as currently there is less knowledge about protective factors that may foster better outcomes; (e) it demonstrates the importance of investigating indirect relations among the predictors as some of these effects appear to be complex; it thus indicates that future research is needed to clarify these processes, (f) that further research is also required to investigate whether alternative or additional variables might improve the predictive capacity of similar models and g) the conceptual model furthers understanding in relation to potential therapeutic targets for clinicians working with clients with chronic pain.

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Appendix A – Ethics Approval and Research Agreement

Ethics approval to access the de-identified PMU patient database of assessment clinical outcomes was obtained from Barwon Health Human Research Ethics Committee (BHHREC) (acting as the primary HREC in this study) in January 2011. Ethics approval was obtained from Monash University (acting as the secondary HREC) in May 2011. Copies of the research ethics approval letters are included below. An intellectual property agreement was signed between Barwon Health and Monash University in April 2011. This was drafted with assistance of Monash University legal advisers. A copy of this agreement follows.

Study One

Office for Research

OFFICE FOR RESEARCH
P.O. Box 291
Geelong Victoria 3220
Telephone: 03 5226 7920
Facsimile: 03 5226 7296
e-mail: ferc@BarwonHealth.org.au

Barwon Health

Correspondence: Jo Sheedy
17 Lupton Street
GEELONG WEST VIC 3218

ETHICS COMMITTEE APPROVAL STATEMENT
LOW RISK

HREC Project Number	10/133
Site	Barwon Health
Date Approved	18/01/2011
Principal Investigator	Jo Sheedy
Title:	Resilience Factors in Chronic Pain
Co investigators	Penne Schmidt
Student names	

Thankyou for submitting the above for our consideration. Your project is approved and you may commence. Approval is granted on the basis that this is a low risk project, as defined by the "National Statement on Ethical Conduct in Human Research" sections 2.1.6 to 2.1.7.

Your obligations under this approval include notifying the Committee of any intent to deviate from the approved protocol and of the occurrence of any untoward events.

Please note that your final report is due on completion and in the interim progress reports are due annually on the anniversary of your approval.

Should you require any further information concerning the Committee's approval of your research or have any concerns regarding the reporting requirements please contact the Office for Research on 5226 7920.

In all future correspondence regarding your study please quote the HREC Project Number and full title of your research project.

On behalf of the Committee, best wishes for your project.

Yours sincerely

Research Review Committee

Dr Mary Lou Chatterton
Manager, Research Office
Barwon Health
Telephone: (03) 5245 8231
Fax: (03) 5200 3023
Email: mchatt@barwonhealth.org.au

MONASH University

Monash University Human Research Ethics Committee (MUHREC)
Research Office

Human Ethics Certificate of Approval

Date:	**11 May 2011**
Project Number:	**2011000660**
Project Title:	**Risk and resistance factors in adjustment to chronic pain**
Chief Investigator:	**Dr Louise McLean**
Approved:	**From: 11 May 2011 To: 11 May 2016**

Terms of approval

1. The Chief investigator is responsible for ensuring that permission letters are obtained, if relevant, and a copy forwarded to MUHREC before any data collection can occur at the specified organisation. **Failure to provide permission letters to MUHREC before data collection commences is in breach of the National Statement on Ethical Conduct in Human Research and the Australian Code for the Responsible Conduct of Research.**
2. Approval is only valid whilst you hold a position at Monash University.
3. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval and to ensure the project is conducted as approved by MUHREC.
4. You should notify MUHREC immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.
5. **Complaints:** The researchers are required to inform MUHREC promptly of any complaints made about the project, whether the complaint was made directly to a member of the research team or to the primary HREC.
6. **Amendments to the approved project (including changes in personnel):** Requires the submission of a Request for Amendment form to MUHREC and must not begin without written approval from MUHREC. Substantial variations may require a new application.
7. **Future correspondence:** Please quote the project number and project title above in any further correspondence.
8. **Annual reports:** Continued approval of this project is dependent on the submission of an Annual Report. This is determined by the date of your letter of approval.
9. **Final report:** A Final Report should be provided at the conclusion of the project. MUHREC should be notified if the project is discontinued before the expected date of completion.
10. **Monitoring:** Projects may be subject to an audit or any other form of monitoring by MUHREC at any time.
11. **Retention and storage of data:** The Chief Investigator is responsible for the storage and retention of original data pertaining to a project for a minimum period of five years.

Ben Canny

Professor Ben Canny
Chair, MUHREC

cc: Ms Jo Sheedy, Dr Penelope Schmidt, Ms Tania Gibbie

Postal – Monash University, Vic 3800, Australia
Building 3E, Room 111, Clayton Campus, Wellington Road, Clayton
Telephone +61 3 9905 5490 Facsimile +61 3 9905 3831
Email muhrec@adm.monash.edu.au www.monash.edu/research/ethics/human/index/html
ABN 12 377 614 012 CRICOS Provider #00008C

RESEARCH AGREEMENT

This AGREEMENT is made on the 15th day of April 2011

BETWEEN

Barwon Health Pain Management Unit ("PMU")
Of The Geelong Hospital, Bellarine St., Geelong VIC. 3220.

And

Jo Sheedy ("the student")
Of 17 Lupton Street, Geelong West, VIC. 3218.

Recitals

A. The student is enrolled in the Masters of Counselling Psychology/ PhD program ("the program") at Monash University, Clayton Campus ("the University").
B. The student is being supervised by Dr. Louise McLean staff member of the University.
C. The agreement is based on proposed research ("proposed research") set out in Appendix A of this agreement.
D. PMU agrees to make available to the student raw data containing information about chronic pain clients and their thoughts and behavior in relation to their chronic pain.
E. PMU and the student agree to participate in the project ("the project") which includes publications on the terms and conditions herein.

THE PARTIES AGREE

Terms

1. The commencement date of this agreement is the date when both parties have signed this agreement and if not on the same date then whichever is the later.

2. The end date of this agreement is the earlier of the date on which the terms of this agreement are completed or two years from the commencement date.

3. The agreement can be extended by written agreement of the parties or terminated early in accordance with the provisions of this agreement.

4. The parties acknowledge that in carrying out the project the student acts independently of, and is not an employee of the University and as such has no authority to act for or bind the University in any manner whatsoever other than as contemplated by this agreement.

5. The parties acknowledge that prior to the project commencement the student will be required to obtain ethics approval from the University and from Barwon Health. The student will use her best endeavours to obtain the requisite ethics approval.

6. Notwithstanding anything in this agreement, the student will own copyright in her thesis, the proposed research or other works the student produces for the purposes of assessment towards her degree program.
7. If either party contemplates carrying out additional research outside of the student's program using PMU data, Barwon Health must be notified and give approval before further research is undertaken.

Project

1. The parties agree that in exchange for access by PMU to its raw data the student will complete a research study examining risk and protective factors in adjustment to chronic pain based on the research proposal in Appendix A, a copy of the research study will be provided to PMU upon completion of the student's program.

2. The student will use her best endeavours to publish her analysed data in peer reviewed journals in reasonable time and acknowledge PMU's contribution in each and every journal paper or conference paper as the case may be.

3. PMU agrees that no other individual will be given permission to undertake a replication of the project during the time that it is being completed by the student and not before the submission of her thesis required in order to complete the program at the University, as this would place the originality of her thesis in jeopardy.

4. The parties agree that the student hereby licenses PMU to use, reproduce, modify or adapt the student data for research, educational and reporting purposes only after the completion of her program.

5. The parties agree that PMU hereby licenses the student to use and publish, for the purpose of the student's assessable work towards her degree program the data arising as a result of the project to the extent that it is owned by PMU.

Publications

1. The student shall prepare and publish the results of her project in a reputable journal within a reasonable time. The student will give notice to PMU of any proposed publication/presentation of the results at least 30 days before submission. Within that time PMU may do any one or more of the following:
    a. provide comments on the proposed publication, in which case the student must consider such comments but will not be bound to follow them;
    b. request that the student remove specified Confidential Information (other than the results of the project) from the Publication, and in this event the student will remove such specified Confidential Information as is reasonably required to protect any Intellectual Property belonging to PMU;
    c. if the student has not received any comments from PMU on the proposed publication within 30 days of giving notice, the student may proceed to publication.

2. PMU acknowledges that the student will publish her data and analysis as part of her thesis program.

Authorship

1. Authorship shall be decided in accordance with National Health and Medical Research Council (NHMRC) authorship policy and the mminimum requirement for authorship in accordance with the "Vancouver Protocol" and the *Copyright Act 1968 (Commonwealth).*

2. The parties agree that lead and second authorship will be reserved for the student and her University supervisor.
3. The parties agree that the student and her University supervisor reserve the right to decide about the final version of any publication(s) for submission and reserve the right to select the most suitable Journal(s).
4. PMU staff may be considered as authors in accordance with the authorship principles and applicable legislation.

Intellectual Property

In this Agreement:

(a) "Background IP" means Intellectual Property not created under the Project which is contributed by either party for the purpose of carrying out the terms of this agreement and includes proposed research.
(b) "Intellectual Property" means all intellectual property protectable by statute.
(c) "PMU data" is raw data belonging to PMU comprising of information related to chronic pain patients and their behavior, perception and response to the chronic pain.
(d) "student data" means data created by the student and includes data set(s) arranged, collated, organized and formatted by the student, including any commentary and so constituting "intellectual property" in the nature of copyright in the analysed data set, created in the process of completing the research proposal or project.

1. It is acknowledged at all times that PMU retains full ownership of the PMU data to be used as the basis of the project.

2. The parties agree that the student will retain ownership of the student data.

3. The student data may not be used by PMU as the basis for publication without prior written permission of the student.

4. Ownership of background IP will remain with the contributing party.

5. Nothing in this agreement shall be construed as amounting to assignment of intellectual property rights and neither party may assign, charge or otherwise deal with any of its rights or interest in this agreement.

Confidentiality

1. Each party acknowledges that all documents, data and information disclosed by the other party is "Confidential Information" and shall be used only for the purposes of this agreement. Each party shall keep the confidential information confidential and may disclose it only to its officers and employees who have a need to know for the purposes of this agreement. Before disclosure, each party disclosing confidential information shall direct that its officers and employees keep the information confidential.

2. Confidential Information does not include information which is in the public domain at the time of disclosure; is published or otherwise becomes part of the public domain through no fault of PMU or the student; is received from a third party without an obligation of non-disclosure; is independently created by the student; or is required to be disclosed by law.

3. The parties agree that the data utilized for the project shall be treated as confidential and will only be used for the purpose of fulfilling the terms of this agreement.

4. The parties agree that psychological outcomes data will at all times be de-identified and will be accessible only to the student and her University Supervisor, Dr. Louise McLean. The parties shall take precautions to ensure that any additional personal information arising from the project or proposed research is collected, stored, used and disclosed in accordance with Federal and State privacy legislation.

5. The obligations of confidentiality are continuing and shall not cease on termination of this agreement.

Warranties and Indemnification

The parties warrant that in carrying out their respective obligations under this agreement they will not violate the copyright or any other intellectual property right of any third party. The parties agree to indemnify each other from and against any and all liability, losses, actions, proceedings, claims, demands, damages and costs (including legal costs) arising out of any third party claim of breach of intellectual property rights or privacy rights, or disclosure of any confidential information, arising out of this agreement.

Termination

1. Either party may on 14 days written notice terminate this agreement for breach of this Agreement, or if the program is discontinued for reasons beyond the student's reasonable control or for any other reason as agreed between the parties.

2. In the event of termination of this agreement and upon written request the student will return to PMU all PMU data.

IN WITNESS WHEREOF the parties have signed this Agreement on the day and year first hereinbefore written.

SIGNED for and on behalf of)

Barwon Health Pain Management Unit by)

(Name) .....)

(Title) .....)

in the presence of:)

.....)

SIGNED by)

Jo Sheedy)

……)

in the presence of:
……..)

Study Two

MONASH University

Monash University Human Research Ethics Committee (MUHREC)
Research Office

Human Ethics Certificate of Approval

Date:	**4 July 2013**
Project Number:	**CF13/1274 - 2013000630**
Project Title:	**Living with chronic pain**
Chief Investigator:	**Dr Angelika Anderson**
Approved:	**From 4 July 2013 to 4 July 2018**

Terms of approval

1. The Chief investigator is responsible for ensuring that permission letters are obtained, if relevant, and a copy forwarded to MUHREC before any data collection can occur at the specified organisation. **Failure to provide permission letters to MUHREC before data collection commences is in breach of the National Statement on Ethical Conduct in Human Research and the Australian Code for the Responsible Conduct of Research.**
2. Approval is only valid whilst you hold a position at Monash University.
3. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval and to ensure the project is conducted as approved by MUHREC.
4. You should notify MUHREC immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.
5. The Explanatory Statement must be on Monash University letterhead and the Monash University complaints clause must contain your project number.
6. **Amendments to the approved project (including changes in personnel):** Requires the submission of a Request for Amendment form to MUHREC and must not begin without written approval from MUHREC. Substantial variations may require a new application.
7. **Future correspondence:** Please quote the project number and project title above in any further correspondence.
8. **Annual reports:** Continued approval of this project is dependent on the submission of an Annual Report. This is determined by the date of your letter of approval.
9. **Final report:** A Final Report should be provided at the conclusion of the project. MUHREC should be notified if the project is discontinued before the expected date of completion.
10. **Monitoring:** Projects may be subject to an audit or any other form of monitoring by MUHREC at any time.
11. **Retention and storage of data:** The Chief Investigator is responsible for the storage and retention of original data pertaining to a project for a minimum period of five years.

Professor Nip Thomson
Chair, MUHREC

cc: Ms Jo Sheedy; Dr Jocelynne Gordon

Postal – Monash University, Vic 3800, Australia
Building 3E, Room 111, Clayton Campus, Wellington Road, Clayton
Telephone +61 3 9905 5490 Facsimile +61 3 9905 3831
Email muhrec@monash.edu http://www.monash.edu.au/researchoffice/human/
ABN 12 377 614 012 CRICOS Provider #00008C

Study Three

ETHICS COMMITTEE CERTIFICATE OF EXEMPTION
Negligible Risk Research

Barwon Health

7 November 2014

Barwon Health Reference	**14/134**
Project Title	**Living with Chronic Pain**
Principal Researcher	**Jo Sheedy**
Research Team	**Dr Louise McLean, Dr Kate Jacobs**

The Barwon Health Research Office advises that the above project is exempt from ethical review as outlined in the National Statement on Ethical Conduct in Human Research Section 5.

Conduct of the project is subject to compliance with the conditions set out and any additional conditions specified by the Barwon Health Research Governance Committee.

The documents reviewed and approved are:

Application for Exemption of Ethics Committee Review
Research Protocol
Explanatory Statement
Background Information Questionnaire
Flyer

In order to comply with the National Statement on Ethical Conduct in Human Research (2007), Guidelines for Good Clinical Research Practice and local research policies and guidelines, you are required to notify the Barwon Health Office for Research of:

- The actual start date of the project at Barwon Health;
- Any amendments to the project after these have been approved by the reviewing HREC;
- Any adverse events or unexpected developments in the project with ethical implications;
- Your inability to continue as Principal Investigator and any other change in research personnel involved in the project at Barwon Health;
- Any decision taken to end the project prior/after the expected date of completion, and;
- To provide a comprehensive written annual/final report/s on the anniversary of project approval advising of the progress of the project and a final report advising of completion.

Templates for reporting changes to the project and annual/final reports may be obtained from the Department of Health website - http://www.health.vic.gov.au/clinicaltrials/application-instructions.htm

Special Conditions

The site/s to which this approval pertains includes:

- *Barwon Health*

Additional Conditions

Barwon Health reserves the right to conduct an audit of this project at any time.

Please quote the Barwon Health reference number and title in all future correspondence.

The approval of this project will be ratified at the upcoming Human Research Ethics Committee (HREC) meeting.

On behalf of the Barwon Health Office for Research, best wishes for your research project.

Yours sincerely

Dr Giuliana Fuscaldo
Chair – Research Review Committee
Manager, Office for Research
Barwon Health

All correspondence to: The Office for Research, Barwon Health – Post. PO Box 281 Geelong 3220
Phone: (03) 4215 3372
Email: HREC@barwonhealth.org.au

Study Three

MONASH University

Monash University Human Research Ethics Committee (MUHREC)
Research Office

Human Ethics Certificate of Approval

This is to certify that the project below was considered by the Monash University Human Research Ethics Committee. The Committee was satisfied that the proposal meets the requirements of the *National Statement on Ethical Conduct in Human Research* and has granted approval.

Project Number:	CF14/2290 - 2014001213		
Project Title:	Living with chronic pain		
Chief Investigator:	Dr Louise McLean		
Approved:	**From:** 9 September 2014	**To:**	9 September 2019

Terms of approval - ***Failure to comply with the terms below is in breach of your approval and the Australian Code for the Responsible Conduct of Research.***

1. The Chief investigator is responsible for ensuring that permission letters are obtained, if relevant, before any data collection can occur at the specified organisation.
2. Approval is only valid whilst you hold a position at Monash University.
3. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval and to ensure the project is conducted as approved by MUHREC.
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11. **Retention and storage of data:** The Chief Investigator is responsible for the storage and retention of original data pertaining to a project for a minimum period of five years.

Professor Nip Thomson
Chair, MUHREC

cc: Dr Kate Jacobs, Ms Jo Sheedy

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Appendix B – Study One Questionnaire

Disability Questionnaire (Roland & Morris, 1983)

When your pain hurts, you may 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 have pain. When you read them, you may find that some stand out because they describe you **today**. When you read a sentence that describes you today, put a tick ☑ in the box beside it. If the sentence does not describe you, then leave the box blank and go on to the next one. Remember; only tick the sentence if you are sure that it describes you **today.**

1. I stay at home most of the time because of my pain	☐
2. I change position frequently to try and get my pain 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 up stairs	☐
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 am in pain almost all the time.	☐
14. I find it difficult to turn over in bed because of my pain	☐
15. My appetite is not very good because of my pain	☐
16. I have trouble putting on my socks (or stockings) because of my pain	☐
17. I only walk short distances because of my pain	☐
18. I sleep less well because of my pain	☐
19. Because of my pain, I get dressed with help from someone else	☐
20. I sit down for most of the day because of my pain	☐
21. I avoid heavy jobs around the house because of my pain	☐
22. Because of my pain, I am more irritable and bad tempered with people than usual	☐
23. Because of pain, I go up stairs more slowly than usual	☐
24. I stay in bed most of the time because of my pain	☐

Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995)

Please read each statement and circle a number 0, 1, 2, or 3, which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement. The rating scale is as follows:

0 = Did not apply to me at all
1 = Applied to me to some degree, or some of the time
2 = Applied to me to a considerable degree, or a good bit of the time
3 = Applied to me very much, or most of the time.

1. I found it hard to wind down……	0	1	2	3
2. I was aware of dryness of my mouth……	0	1	2	3
3. I couldn't seem to experience any positive feelings at all……	0	1	2	3
4. I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)…….	0	1	2	3
5. I found it difficult to work up the initiative to do things……	0	1	2	3
6. I tended to over-react to situations……	0	1	2	3
7. I experienced trembling (e.g., in the hands)..……	0	1	2	3
8. I felt that I was using a lot of nervous energy……	0	1	2	3
9. I was worried about situations in which I might panic and make a fool of myself….	0	1	2	3
10. I felt I had nothing to look forward to……	0	1	2	3
11. I found myself agitated……	0	1	2	3
12. I found it difficult to relax……...……	0	1	2	3
13. I felt downhearted and blue……	0	1	2	3
14. I was intolerant of anything that kept me from getting on with what I was doing….	0	1	2	3
15. I felt close to panic……	0	1	2	3
16. I was unable to become enthusiastic about anything……	0	1	2	3
17. I felt I wasn't worth much as a person……	0	1	2	3
18. I felt that I was rather touchy……	0	1	2	3
19. I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)……..	0	1	2	3
20. I felt scared without any good reason……	0	1	2	3
21. I felt life was meaningless……	0	1	2	3

Pain Responses Self Statements (PRSS; Flor, Behle & Birbaumer, 1993)

Most of the time we have an internal conversation with ourselves. We encourage ourselves, for example, to do certain things. We blame ourselves if we have made a mistake and we reward ourselves for our accomplishments. When we are in pain we also say certain things to ourselves that are different from what we say when we are feeling good. Below are listed typical thoughts of people in pain. Please read each of the statements and then mark how often you have this thought when your pain is severe. Please circle the appropriate number on the scale ranging from:
0 = almost never to **5 =almost always**

	Almost never					**Almost always**
1. If I stay calm and relaxed, things will be better……	0	1	2	3	4	5
2. I cannot stand this pain any longer……	0	1	2	3	4	5
3. I can do something about my pain ……	0	1	2	3	4	5
4. No matter what I do, my pain doesn't change ……	0	1	2	3	4	5
5. I need to relax……..	0	1	2	3	4	5
6. I'll manage ……	0	1	2	3	4	5
7. I need to take some pain medication ……..	0	1	2	3	4	5
8. I will soon be better again……...	0	1	2	3	4	5
9. This will never end …….	0	1	2	3	4	5
10. I am a hopeless case……..	0	1	2	3	4	5
11. There are worse things than my pain……	0	1	2	3	4	5
12. I'll cope with it……	0	1	2	3	4	5
13. When will it get worse again?…….	0	1	2	3	4	5
14. This pain is killing me……..	0	1	2	3	4	5
15. I can't go on anymore……..	0	1	2	3	4	5
16. This pain is driving me crazy……..	0	1	2	3	4	5
17. Distraction helps best……..	0	1	2	3	4	5
18. I can help myself……..	0	1	2	3	4	5

West-Haven Yale Multiphasic Pain Inventory ***(MPI; Kerns, Turk & Rudy, 1985)***

SECTION 1

In this section we are interested in knowing how you describe your pain and how it affects your life.
Circle a number on the scale beside each question to indicate how that question applies to you. Please answer all 28 questions.

1.	Rate the level of your pain at the present moment.	0	1	2	3	4	5	6
		No Pain				*Very Intense Pain*		
2.	In general, how much does your pain interfere with your day-to-day activities?	0	1	2	3	4	5	6
		No Interference				*Extreme Interference*		
3.	Since the time your pain began, how much has you pain changed your ability to work?	0	1	2	3	4	5	6
	(Tick here ☐ if you have retired for reasons other than pain)	*No change*				*Extreme change*		
4.	How much has your pain changed the amount of satisfaction or enjoyment you get from taking part in social and recreational activities?	0	1	2	3	4	5	6
		No change				*Extreme change*		
5.	How supportive or helpful is your spouse (significant other) to you in relation to your pain?	0	1	2	3	4	5	6
		Not at all supportive				*Very supportive*		
6.	Rate your overall mood during the past week.	0	1	2	3	4	5	6
		Extremely low				*Extremely high*		
7.	How much has your pain interfered with your ability to get enough sleep?	0	1	2	3	4	5	6
		No interference				*Extreme interference*		
8.	On the average, how severe has your pain been during the last week?	0	1	2	3	4	5	6
		Not at all severe				*Extremely severe*		
9.	How able are you to predict when your pain will start, get better, or get worse?	0	1	2	3	4	5	6
		Not at all				*Very much*		
10.	How much has your pain changed your ability to take part in recreational and other social activities?	0	1	2	3	4	5	6
		No change				*Extreme change*		
11.	How much do you limit your activities in order to keep your pain from getting worse?	0	1	2	3	4	5	6
		Not at all				*Very much*		
12.	How much has your pain changed the amount of satisfaction or enjoyment you get from family related activities?	0	1	2	3	4	5	6
		Not change				*Extreme change*		
13.	How worried is your spouse (significant other) about you because of your pain?	0	1	2	3	4	5	6
		Not at all worried				*Extremely worried*		
14.	During the past week how much control do you feel that you have had over your life?	0	1	2	3	4	5	6
		No control				*Extreme control*		

15.	On an average day, how much does your pain vary (increase or decrease)?	0	1	2	3	4	5	6
		Remains the same						*Changes a lot*
16.	How much suffering do you experience because of your pain?	0	1	2	3	4	5	6
		No suffering						*Extreme suffering*
17.	How often are you able to do something that helps to reduce your pain?	0	1	2	3	4	5	6
		Never						*Very often*
18.	How much has your pain changed your relationship with your spouse, family, or significant other?	0	1	2	3	4	5	6
		No change						*Extreme* change
19.	How much has your pain changed the amount of satisfaction or enjoyment you get from work?	0	1	2	3	4	5	6
	(Tick here ☐ if you are not presently working).	*No change*						*Extreme change*

SECTION 1 contd

In this section we are interested in knowing how you describe your pain and how it affects your life.
Circle a number on the scale beside each question to indicate how that question applies to you. Please answer all 28 questions.

20.	How attentive is your spouse (significant other) to you because of your pain?	0	1	2	3	4	5	6
		Not at all attentive						*Extremely attentive*
21.	During the past week how much do you feel that you've been able to deal with your problems?	0	1	2	3	4	5	6
		Not at all						*Extremely well*
22.	How much control do you feel that you have over your pain?	0	1	2	3	4	5	6
		No control at all						*A great deal of control*
23.	How much has your pain changed your ability to do household chores?	0	1	2	3	4	5	6
		No change						*Extreme change*
24.	During the past week how successful were you in coping with stressful situations in your life?	0	1	2	3	4	5	6
		Not at all successful						*Extremely successful*
25.	How much has your pain interfered with your ability to plan activities?	0	1	2	3	4	5	6
		No change						*Extreme change*
26.	During the past week how irritable have you been?	0	1	2	3	4	5	6
		Not at all irritable						*Extremely irritable*
27.	How much has your pain changed or interfered your friendships with people other than your family?	0	1	2	3	4	5	6
		No change						*Extreme change*
28.	During the past week how tense or anxious have you been?	0	1	2	3	4	5	6
		Not at all tense or anxious						*Extremely tense & anxious*

SECTION 2

In this section, we are interested in knowing how your spouse (or significant other) responds to you when he or she knows that you are in pain. Please circle a number on the scale beside each question to indicate how often your spouse (or significant other) responds to you in that way when you are in pain. Please answer all 14 questions.

1.	Ignores me.	0	1	2	3	4	5	6
		Never						*Very often*
2.	Asks me what he/she can do to help.	0	1	2	3	4	5	6
		Never						*Very often*
3.	Reads to me.	0	1	2	3	4	5	6
		Never						*Very often*
4.	Gets irritated with me.	0	1	2	3	4	5	6
		Never						*Very often*
5.	Takes over my jobs or duties.	0	1	2	3	4	5	6
		Never						*Very often*
6.	Talks to me about something else to take my mind off the pain.	0	1	2	3	4	5	6
		Never						*Very often*
7.	Gets frustrated with me.	0	1	2	3	4	5	6
		Never						*Very often*
8.	Tries to get me to rest.	0	1	2	3	4	5	6
		Never						*Very often*
9.	Tries to involve me in some activity.	0	1	2	3	4	5	6
		Never						*Very often*
10.	Gets angry with me.	0	1	2	3	4	5	6
		Never						*Very often*
11.	Gets my pain medication.	0	1	2	3	4	5	6
		Never						*Very often*
12.	Encourages me to work on a hobby.	0	1	2	3	4	5	6
		Never						*Very often*
13.	Gets me something to eat or drink.	0	1	2	3	4	5	6
		Never						*Very often*
14.	Turns on the TV to take my mind off my pain.	0	1	2	3	4	5	6
		Never						*Very often*

SECTION 3

Listed below are 18 daily activities. Circle a number on the scale beside each question to indicate how often you do that activity. Please answer all 18 questions.

1.	Wash dishes.	0	1	2	3	4	5	6
		Never						*Very often*
2.	Mow the lawn (☐ tick here, if you do not have a lawn to mow).	0	1	2	3	4	5	6
		Never						*Very often*
3.	Go out to eat.	0	1	2	3	4	5	6
		Never						*Very often*
4.	Play cards or other games.	0	1	2	3	4	5	6
		Never						*Very often*
5.	Go grocery shopping.	0	1	2	3	4	5	6
		Never						*Very often*

6.	Work in garden (☐ tick here, if you do not have a garden).	0	1	2	3	4	5	6
		Never						*Very often*
7.	Go to a movie.	0	1	2	3	4	5	6
		Never						*Very often*
8.	Visit friends.	0	1	2	3	4	5	6
		Never						*Very often*
9.	Help with the house cleaning.	0	1	2	3	4	5	6
		Never						*Very often*
10.	Work on the car (☐ tick here, if you do not have a car).	0	1	2	3	4	5	6
		Never						*Very often*
11.	Take a ride in a car or bus.	0	1	2	3	4	5	6
		Never						*Very often*
12.	Visit relatives (☐ tick here, if you do not have relatives within 160 kms).	0	1	2	3	4	5	6
		Never						*Very often*
13.	Prepare a meal.	0	1	2	3	4	5	6
		Never						*Very often*
14.	Wash the car (☐ tick here if you don't have a car).	0	1	2	3	4	5	6
		Never						*Very often*
15.	Take a trip.	0	1	2	3	4	5	6
		Never						*Very often*
16.	Go to a park or beach.	0	1	2	3	4	5	6
		Never						*Very often*
17.	Do the laundry.	0	1	2	3	4	5	6
		Never						*Very often*
18.	Work on a needed household repair.	0	1	2	3	4	5	6
		Never						*Very often*

Pain Self Efficacy Questionnaire (PSEQ, Nicholas, 1988)

Please rate how confident you are that you can do the following things at present, despite the pain. To indicate your answer circle one of the numbers on the scale beside each item, where **0 = not at all confident** and **6 = completely confident**.

		Not at all confident						**Completely confident**
1.	I can enjoy things, despite the pain.	0	1	2	3	4	5	6
2.	I can do most of the household chores (e.g., tidying up, washing dishes, etc) despite the pain.	0	1	2	3	4	5	6
3.	I can socialise with my friends or family members as often as I used to do, despite the pain.	0	1	2	3	4	5	6
4.	I can cope with my pain in most situations.	0	1	2	3	4	5	6
5.	I can do some form of work, despite the pain (“work” includes housework, paid and unpaid work).	0	1	2	3	4	5	6
6.	I can still do many of the things I enjoy doing, such as hobbies or leisure activity, despite the pain.	0	1	2	3	4	5	6
7.	I can cope with my pain without medication.	0	1	2	3	4	5	6
8.	I can still accomplish most of my goals in life, despite the pain.	0	1	2	3	4	5	6
9.	I can live a normal life style, despite the pain.	0	1	2	3	4	5	6
10.	I can gradually become more active, despite the pain.	0	1	2	3	4	5	6

Appendix C – Missing Data Analysis

Each scale or subscale was examined for missing data. Missing data patterns were assessed in SPSS using inspection of frequency tables. Little's Missing Completely at Random (MCAR) test (Little, 1988) was used to assess if the data was missing at random. The MCAR test (Little, 1988) is a single global statistic that uses all the available data to assess missing data patterns. For this test, the null hypothesis is that the data are missing completely at random; if the *p* value is less than 0.05, the null is rejected. Separate variance *t* tests were also used to identify variables whose pattern of missing values may have influenced other quantitative variables. The *t* test was computed using an indicator variable that specifies whether a variable is present or missing for an individual case. Differences were examined between means of the quantitative variables when the indicator variable was present or missing. Significant differences in means suggest that the data is unlikely to be missing at random.

The proportion of missing data for all scales except the Multidimensional Pain Inventory (Kerns, Turk & Rudy, 1995) subscales assessing partner responses to pain, ranged between zero (age and gender) and 10% (anxiety). Overall 96.92% of data points were complete. However, approximately 25% of responses to the subscales assessing partner behaviours in response to participants' pain were missing. See Table 1. These subscales ask participants to rate the frequency with which their partner engages in particular behaviours in response to their pain (for example, 'ignores me' and 'tries to get me to rest'). The original version of the MPI (Kerns et al., 1995) instructs participants who are not living with a partner to respond to these items by referencing the person with whom they have their closest relationship. However, the pain clinic questionnaires did not include this statement. Thus, it was likely that participants not partnered at the time of assessment did not respond to these items. Graphs One, Two and Three below summarise patterns of missing data.

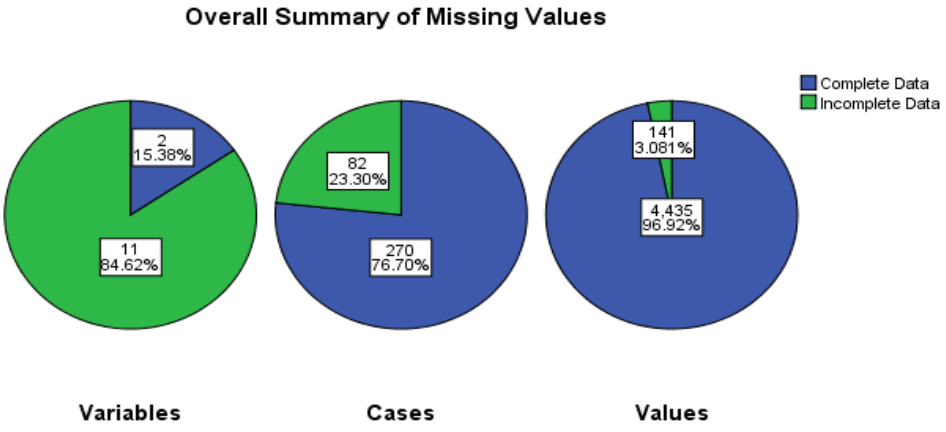

Figure C1 *Missing data patterns.*

Missing data analyses were conducted in two stages. Stage one included only scales or subscales not pertaining to partner responses to pain. Separate variance t-tests of only these variables revealed no significant differences between means of the quantitative variables when the indicator variable was present or missing. This suggests that the data were likely missing at random. The significance value of Little's (1977) MCAR test for these variables was more than .05, indicating that these data were missing completely at random (χ 2 = 233.91, *df* = 225, *p* = .33).

The second stage of the missing data analyses involved analysing missing data within the two MPI (Kerns et al., 1995) subscales pertaining to partner responses to pain. Descriptive statistics were obtained to assess proportions of missing data. Little's MCAR test for these data was highly significant, indicating that these data were not missing at random (χ 2 = 55.55, *df* = 7, *p* = .00).

To explore group differences between those with complete versus incomplete partner data, the dataset was stratified into two groups; complete (265 participants) and incomplete (87 participants). When Pearson Chi Square was used to examine proportional gender differences across the two groups no significant differences were found (χ 2= 1.33, *p* = 0.25). Group differences

for other variables were then examined. Levene's test for equality of variances was only significant for group differences in DASS depression score. For this predictor, the *t* value for equal variances not assumed is reported. Only one statistically significant difference between groups was noted; those responding to partner questions about pain were significantly more socially active than non-responders ($t = 2.72$, $p = 0.01$).

Management of missing data

A number of different options exist to manage missing data. These include case deletion, replacement of missing values with the mean and data imputation (Graham, 2009). Deletion of cases with missing partner responses was considered. However, due to the substantial loss of data resulting from this approach, alternative strategies were sought. Graham (2009), argues that despite historical beliefs to the contrary, imputation may be at least as appropriate as case deletion and may produce comparatively less biased results.

Thus, these missing data were replaced using the Expectation-Maximisation (EM) algorithm. The EM algorithm uses the means, variances, and co-variances of the available data to calculate replacement values and is as an accurate and robust procedure for replacing values that are missing at random (Graham, 2009; Tabachnik & Fidell, 2013). Multiple imputation was also considered to replace missing values but because SPSS does not pool results of resultant data sheets, the series of data sheets produced would have substantially complicated the analysis. Therefore, EM was used instead. To enable comparison, two data sets were created - an imputed one and one that contained only cases with complete partner responses. Descriptive data for these two data sets were compared. Minimal differences were noted between measures assessing partner responses. To ensure that the data imputation did not affect regression estimates, the regression analyses were examined in both datasets, the one with deleted cases (N = 265) and the fully imputed data (N = 352). Results did not differ, thus only results from the imputed dataset are reported.

Table C1

Descriptive Statistics of Original and Imputed Data

	N Original data	Missing *Number*	Missing *%*	*M* Original data	*M* Imputed data	*SD* Original data	*SD* Imputed data
Age	343	9	2.6	51.62	51.62	13.66	13.49
Catastrophising	349	3	0.9	3.15	3.15	1.11	1.11
Anxiety	316	36	10.2	10.80	11.03	10.08	10.23
Depression	326	26	7.4	17.87	17.96	12.60	12.63
Pain self-efficacy	344	8	2.3	21.47	21.78	14.12	14.49
Pain severity	352	0	0.0	4.47	4.48	.97	.96
Interference	348	4	1.1	4.51	4.52	1.13	1.13
Social engagement	347	5	1.4	1.78	1.79	1.18	1.16
Punishing partner response	265	87	24.7	2.01	2.02	1.64	1.49
Solicitous partner response	265	87	24.7	3.50	3.39	1.57	1.50

Appendix D – Study One Data Screening and Variable Distribution

Assessment of normality

Each variable was assessed for normality and outliers in SPSS 20.0 (IBM Corp., 2012). Histograms with a normality plot, box plots and detrended normal quantile-quantile (Q-Q) plots were examined for all variables. Skewness and kurtosis values were assessed. Recommended thresholds of these values are +/- 1.5 (Tabachnik & Fidell, 2013). Kolmogorov-Smirnov and Shapiro-Wilk statistics were also used to assess significance of skewness and kurtosis values, with a significant result indicative of non-normal distributions. However, these test statistics are commonly significant in large sample sizes (>200), thus it is suggested they are not the best indicator of sample distribution (Pallant, 2010; Tabachnik & Fidell, 2013). Frequency histograms depict visually how well the sample fits a normal distribution. Box plots also show the distribution of a sample. The box represents 50% of cases and the whiskers extend out to represent the largest and smallest values. Additional small circles outside the whiskers represent outliers in the sample (Pallant, 2010). The detrended normal Q-Q plot shows the differences between the observed and expected values of a normal distribution. If the distribution is normal, the points should cluster in a horizontal band around zero with no pattern.

The above tests indicated that most variables had a substantively normal distribution. Skewness and kurtosis values for all variables were all within the recommended level of +/- 1.5 (Tabachnik & Fidell, 2013). These values can be seen in Table D1. A number of high outliers were noted in the distribution of anxiety subscale of the Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995). Consideration was given to replacing these values with the mean, however a trial of this approach replacing all values above 19.0 did not alter the distribution substantially. Because the differences between the mean and the five per cent trimmed mean were small (See Table D1) a decision was made to retain these high outliers in the dataset.

Table D1

Skewness and Kurtosis Values of Study One Distributions

	Skewness	*Kurtosis*
Age	.12	-.26
Disability	-.37	-.73
Pain severity	-.38	-.26
Anxiety	1.07	.53
Depression	.43	-.96
Catastrophising	-.54	-.18
Punishing partner response	.60	-.21
Solicitous partner responses	-.26	-.49
Pain self-efficacy	.57	-.52
Social engagement	.50	-.32

Table D2

Means of Variables and Five Per Cent Trimmed Means

Variable	Mean	5% Trimmed mean
Age	51.62	51.55
Disability	14.50	14.64
Pain severity	4.48	4.51
Anxiety	5.51	5.08
Depression	8.98	8.81
Catastrophising	3.15	3.20
Solicitous Responses	3.93	3.42
Punishing Responses	2.02	1.95
Pain self-efficacy	21.66	21.12
Social Engagement	1.79	1.75

Assessing for multivariate outliers

Mahalanobis distances were calculated to assess for multivariate outliers in the risk-resistance regression model. Four multivariate outliers were identified and can be seen below in Table D3. A decision was made to retain these multivariate outliers in the data set because these values were clustered together and did not greatly exceed critical values. To ensure the outliers on Mahalanobis distances did not influence results, the regression and indirect effects analyses were examined in a dataset that had these values deleted. Results were unchanged.

Table D3

Multivariate Outliers Risk-Resistance Models Predicting Pain-Related Disability – Mahalanobis Distances

DV	Critical value	Number of values observed over critical value	Values observed over critical value
Pain-related disability	26.13	4	27.04, 29.15, 30.36, 38.86

Note. DV = Dependent variable

Multivariate outliers were also checked for each of the regression analyses of indirect effects completed using PROCESS (Hayes, 2013). Four independent variables were entered in each of these analyses. Predictors in separate equations were the independent variables, pain severity, anxiety, depression and social engagement and the two mediators, catastrophising and pain self-efficacy. The critical value for Mahalanobis distance for these models was 16.27 as indicated by the Chi square critical values table for three predictors at the level of .001 (Tabachnik & Fidell, 2013). A single case was identified in two models and two cases were identified in the other two models. These values can be seen in Table D4. They were retained in the analyses because of the

small number of cases which were close to critical values. To ensure these outliers did not influence results, the analyses were repeated in a dataset that had these cases deleted, with the same results noted.

Table D4

Multivariate Outliers Parallel Mediator Models Analyses Predicting Pain-Related Disability–Mahalanobis Distances

IV	Critical value	Number of values observed over critical value	Values observed over critical value
Pain severity	16.27	2	18.75, 18.29
Anxiety	16.27	2	18.90, 16.77
Depression	16.27	1	22.88
Social Engagement	16.27	1	18.92

The critical value for Mahalanobis distance for the models testing moderation effects was 13.82 for two predictors respectively at the level of .001 (Tabachnik & Fidell, 2013). A single case was identified in two of the models predicting pain-related disability, these values can be seen below in Table D5. Again, these multivariate outliers were retained in the analyses because of the very small number of cases identified which were close to critical values. The analyses were repeated for the cases that had identified a significant moderation effect in a dataset that had these cases deleted, with the same results noted

Table D5

Multivariate Outliers Moderation Analyses Predicting Pain-Related Disability – Mahalanobis Distances

IV	Moderator	Critical value	Cases over critical value	Value of case over critical value
Catastrophising	Self-Efficacy	13.82	1	15.49
Depression	Social Engagement	13.82	1	13.98

Appendix E –Study Two: Semi Structured Interview Schedule

Introductory Script

Thank you for agreeing to be part of this research project. As you may be aware, the goal of the research is to find out about the process of living with chronic pain on a day to day basis. Chronic pain, as you may know, is pain that lasts for more than three months. In this interview, you are the expert on pain because you are the person living with it. There are no right or wrong answers. I simply would like to find out what it is like to walk in your shoes.

Qualitative questions

1. Tell me, in your own words, what is it like to live with chronic pain?

2. What does 'coping with pain' mean to you?

Probes: What makes living with the pain easier for you? How do these things make it easier for you to manage?

3. Describe the process you went through in order to adapt to the living with pain.

Probes: If you don't feel you have made adaptations to living with pain, has there been anything that may have stopped this process?

4. Do any of the things we have just talked about influence how you see yourself?

5. If your pain is bad, how do you keep going mentally?

6. If your pain is bad, how do you keep going physically?

Probes: Are the things that help you keep going different according to whether your pain is 'good' or 'bad' at that time?

7. Tell me about a time when you felt you managed your pain the best.

8. Is there anything else you would like to tell me that we haven't discussed yet?

Copies of the Roland Morris Disability Questionnaire (Roland & Morris, 1983) and the Depression Anxiety and Stress Scale (Lovibond & Lovibond, 1995) can be seen in Appendix B.

Appendix F – Study Three Questionnaires

Thank you for participating in this research. The following questions ask about your background information. PLEASE COMPLETE BOTH SIDES OF EACH PAGE.

Please record your age _____

Are you? Male ☐ Female ☐

Are you? Single ☐ Defacto / married ☐ Separated / divorced ☐

Is English your first language?
Yes ☐ No ☐ If no, please state first language _____

Are you currently working?
Not working ☐ Studying ☐ Working part-time ☐
Working full-time ☐ Carer ☐ Stay at home parent ☐
Please detail number of years of education completed after Year 9 (for example, completion of Year 11 would be 2 years) _____

Is your annual family income: Less than $30,0000 ☐ Between $30,000 and $70,000 ☐
Between $70,000 and $120,000 ☐ More than $120,000 ☐

Pain diagnosis (if you have one)
_____
_____
Have you experienced pain on most days of the week for more than six months?
☐ Yes ☐ No

Approximately, how long have you been living with chronic pain?
_____ Years OR _____ Months

Is your pain condition currently or has your pain condition ever been compensable under:
TAC Yes ☐ No ☐
WorkCover Yes ☐ No ☐

Do you require pain relieving medications?
☐ Yes ☐ No
If yes, do you need them:
☐ Every day ☐ Sometimes ☐ Rarely

Please list any other health conditions you may have _____
_____
_____
_____

Over the past six months, have any health conditions other than your pain condition caused you to stop doing your regular activities for more than a week in or have required a stay in hospital?

Yes ☐ No ☐

Did you see this survey at: A Community Health Centre ☐ If yes, please list which centre:

Anglesea ☐ Belmont ☐ Corio ☐ Newcomb ☐ Torquay ☐

Or at: Hydrotherapy pool at McKellar centre ☐

A medical, physiotherapy or other type of health practice ☐ A community group ☐

An exercise facility ☐ Online newsletter or other online link ☐

Brief Pain Inventory (Pain Intensity Items Only) Copyright 1991 Charles S. Cleeland, PhD. Pain Research Group. All rights reserved.

Please complete the following questions.

Throughout our lives, most of us have had pain from time to time (such as minor headaches, sprains, and toothaches). Have you had pain other than these everyday kinds of pain today?

Yes ☐ No ☐

Please rate your pain by circling the one number that best describes your pain at its worst in the last 24 hours.

0 1 2 3 4 5 6 7 8 9 10

No pain Pain as bad as you can imagine

Please rate your pain by circling the one number that best describes your pain at its least in the last 24 hours.

0 1 2 3 4 5 6 7 8 9 10

No pain Pain as bad as you can imagine

Please rate your pain by circling the one number that best describes your pain on the average.

0 1 2 3 4 5 6 7 8 9 10

No pain Pain as bad as you can imagine

Please rate your pain by circling the one number that tells how much pain you have right now.

0 1 2 3 4 5 6 7 8 9 10

No pain Pain as bad as you can imagine

Pain Disability Index (Tait et al., 1990).

The rating scales below are designed to measure the degree to which aspects of your life are disrupted by chronic pain. In other words, we would like to know how much pain is preventing you from doing what you would normally do. Respond to each category indicating the overall impact of pain in your life, not just when pain is at its worst. Please circle the number on the scale that describes the level of disability you typically experience. A score of 0 means no disability at all, and a score of 10 signifies that all of the activities in this category have been totally disrupted or prevented by your pain.

Family/Home Responsibilities: This category refers to activities of the home or family. It includes chores performed around the house (e.g. yard work) and errands or favours for other family members (e.g. driving the children to school).

No Disability 0__ 1__ 2__ 3__ 4__ 5__ 6__ 7 __ 8__ 9__ 10__ Worst Disability

Recreation: This category includes hobbies, sports, and other similar leisure time activities.

No Disability 0__ 1__ 2__ 3__ 4__ 5__ 6__ 7 __ 8__ 9__ 10__ Worst Disability

Social Activity: This category refers to activities which involve participation with friends other than family members. It includes parties, theatre, dining out, and other social functions.

No Disability 0___ 1___ 2___ 3___ 4___ 5___ 6___ 7 ___ 8___ 9___ 10 Worst Disability

Occupation: This category refers to activities that are part of or directly related to one's job. This includes non-paying jobs as well, such as that of home duties or volunteer.

No Disability 0___ 1___ 2___ 3___ 4___ 5___ 6___ 7 ___ 8___ 9___ 10 Worst Disability

Sexual Behavior: This category refers to the frequency and quality of one's sex life.

No Disability 0___ 1___ 2___ 3___ 4___ 5___ 6___ 7 ___ 8___ 9___ 10 Worst Disability

Self-Care: This category includes activities, which involve personal maintenance and independent daily living (e.g. taking a shower, driving, getting dressed, etc.)

No Disability 0___ 1___ 2___ 3___ 4___ 5___ 6___ 7 ___ 8___ 9___ 10 Worst Disability

Life-Support Activities: This category refers to basic life supporting behaviours such as eating, sleeping and breathing.

No Disability 0___ 1___ 2___ 3___ 4___ 5___ 6___ 7 ___ 8___ 9___ 10 Worst Disability

Quality of Life Scale (Flanagan, 1978; Burckhardt et al., 1989)

Please read each item and circle the number that best describes how satisfied you are at this time. Please answer each item even if you do not currently participate in an activity or have a relationship. You can be satisfied or dissatisfied with not doing the activity or not having the relationship.

	Delighted	Pleased	Mostly Satisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
Material comforts home, food, conveniences, financial security.	7	6	5	4	3	2	1
Health - being physically fit and vigorous.	7	6	5	4	3	2	1
Relationships with parents, siblings & other relatives- communicating, visiting, helping . . .	7	6	5	4	3	2	1
Having and rearing children	7	6	5	4	3	2	1
Close relationships with spouse or significant other	7	6	5	4	3	2	1
Close friends	7	6	5	4	3	2	1
Helping and encouraging others, volunteering, giving advice . . .	7	6	5	4	3	2	1
Participating in organisations and public affairs	7	6	5	4	3	2	1
Learning- attending school, improving understanding, getting additional knowledge . .	7	6	5	4	3	2	1
Understanding yourself - knowing your assets and limitations - knowing what life is about . .	7	6	5	4	3	2	1
Work - job or in home . . .	7	6	5	4	3	2	1
Expressing yourself creatively	7	6	5	4	3	2	1
Socializing - meeting other people, doing things, parties, etc	7	6	5	4	3	2	1
Reading, listening to music, or observing entertainment	7	6	5	4	3	2	1
Participating in active recreation	7	6	5	4	3	2	1
Independence, doing for yourself	7	6	5	4	3	2	1

Pain Catastrophising Scale (Sullivan et al., 1995)

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery. We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

When I'm in pain…..	Not at all	To a slight degree	To a moderate degree	To a great degree	All the time
I worry all the time about whether the pain will end.	1	2	3	4	5
I feel I can't go on.	1	2	3	4	5
It's terrible and I think it's never going to get any better.	1	2	3	4	5
It's awful and I feel that it overwhelms me.	1	2	3	4	5
I feel I can't stand it anymore.	1	2	3	4	5
I become afraid that the pain will get worse.	1	2	3	4	5
I keep thinking of other painful events.	1	2	3	4	5
I anxiously want the pain to go away.	1	2	3	4	5
I can't seem to keep it out of my mind.	1	2	3	4	5
I keep thinking about how much it hurts.	1	2	3	4	5
I keep thinking about how badly I want the pain to stop.	1	2	3	4	5
There's nothing I can do to reduce the intensity of the pain	1	2	3	4	5
I wonder whether something serious may happen.	1	2	3	4	5

Tampa Scale for Kinesiophobia (Kori et al., 1990)

Please indicate the degree to which you agree or disagree with the following statements.

	Strongly disagree	Disagree	Agree	Strongly agree
People aren't taking my medical condition seriously enough.	1	2	3	4
My body is telling me I have something dangerously wrong.	1	2	3	4
My condition has put my body at risk for the rest of my life.	1	2	3	4
I wouldn't have this much pain if there weren't something potentially dangerous going on in my body.	1	2	3	4
Pain always means I have injured my body.	1	2	3	4
If I were to try to overcome it, my pain would increase.	1	2	3	4
Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening.	1	2	3	4
Pain lets me know when to stop exercising so that I don't injure myself.	1	2	3	4
I'm afraid I might injure myself if I exercise.	1	2	3	4
I can't do all the things normal people do because it's too easy for me to get injured.	1	2	3	4
No-one should have to exercise when she / he is in pain.	1	2	3	4

Positive and Negative Affect Scale, Short Form (Watson et al., 1988)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. Indicate to what extent you have felt this way over the past week.

	Very slightly or not at all	A little	Moderately	Quite a bit	Always
Upset	1	2	3	4	5
Hostile	1	2	3	4	5
Alert	1	2	3	4	5
Ashamed	1	2	3	4	5
Inspired	1	2	3	4	5
Nervous	1	2	3	4	5
Determined	1	2	3	4	5
Attentive	1	2	3	4	5
Afraid	1	2	3	4	5
Active	1	2	3	4	5

Illness Invalidation Inventory (Kool et al., 2010).

We are interested in how others react to people who have health problems or an illness. Each of the sections below refers to different people in your life. We would like you to rate how often during the past year each person or category of people reacted toward you in the way described. After each statement, circle the number between 1 (never) and 5 (very often) to indicate how often they reacted toward you that way.

The questionnaire has five sections, and you will rate the same reactions a number of times, but referring to different people. If a particular section does not apply to you, you may skip that part of the questionnaire and go on to the next section. Remember, rate the items with respect to how others reacted toward you as a person who has health problems or an illness.

Section 1: Spouse or partner

If you are single (not married, a widow/widower, or without a steady partner) then skip Section 1 and go directly to Section 2.

My spouse or partner……	**Never**	**Seldom**	**Some-times**	**Often**	**Very often**
….finds it odd that I can do much more on some days than on other days.	1	2	3	4	5
….thinks I should be tougher.	1	2	3	4	5
….takes me seriously.	1	2	3	4	5
….gives me unhelpful advice.	1	2	3	4	5
….understands the consequences of my health problems or illness.	1	2	3	4	5
….makes me feel like I am an exaggerator.	1	2	3	4	5
….thinks I can work more than I do	1	2	3	4	5
….gives me the chance to talk about what is on my mind.	1	2	3	4	5

Section 2: Family For example, children, parents, brothers, sisters, uncles, aunts, grandparents, in-laws.

My family……	**Never**	**Seldom**	**Some-times**	**Often**	**Very often**
….finds it odd that I can do much more on some days than on other days.	1	2	3	4	5
….thinks I should be tougher.	1	2	3	4	5
….takes me seriously.	1	2	3	4	5
….gives me unhelpful advice.	1	2	3	4	5
….understands the consequences of my health problems or illness.	1	2	3	4	5
….makes me feel like I am an exaggerator.	1	2	3	4	5
….thinks I can work more than I do.	1	2	3	4	5
….gives me the chance to talk about what is on my mind.	1	2	3	4	5

Section 3: Medical professionals
For example, your GP, medical specialist, physiotherapist, and other medical professionals. (Do not include your employer's company doctor).

Medical professionals	**Never**	**Seldom**	**Some-times**	**Often**	**Very often**
….find it odd that I can do much more on some days than on other days.	1	2	3	4	5
….think I should be tougher.	1	2	3	4	5
….take me seriously.	1	2	3	4	5
….give me unhelpful advice.	1	2	3	4	5
….understand the consequences of my health problems or illness.	1	2	3	4	5
….make me feel like I am an exaggerator.	1	2	3	4	5
….think I can work more than I do.	1	2	3	4	5
….give me the chance to talk about what is on my mind.	1	2	3	4	5

Section 4: Work environment For example, your co-workers and boss.

If you did not have paid or unpaid employment in the past year, then skip this Section and go directly to Section 5.

People at work…….	**Never**	**Seldom**	**Some-times**	**Often**	**Very often**
….find it odd that I can do much more on some days than on other days.	1	2	3	4	5
….think I should be tougher.	1	2	3	4	5
….take me seriously.	1	2	3	4	5
….give me unhelpful advice.	1	2	3	4	5
….understand the consequences of my health problems or illness.	1	2	3	4	5
….make me feel like I am an exaggerator.	1	2	3	4	5
….think I can work more than I do.	1	2	3	4	5
….give me the chance to talk about what is on my mind.	1	2	3	4	5

Section 5 : Social services For example, your employer's company doctor, vocational rehabilitation staff, unemployment and other government agencies, organisations for care at home, general government workers and health insurance companies.

If you did not have any interactions with these providers, you may skip this Section.

People in social services…….	**Never**	**Seldom**	**Someti mes**	**Often**	**Very often**
….find it odd that I can do much more on some days than on other days.	1	2	3	4	5
….think I should be tougher.	1	2	3	4	5
….take me seriously.	1	2	3	4	5
….give me unhelpful advice.	1	2	3	4	5
….understand the consequences of my health problems or illness.	1	2	3	4	5
….make me feel like I am an exaggerator.	1	2	3	4	5
….think I can work more than I do.	1	2	3	4	5
….give me the chance to talk about what is on my mind.	1	2	3	4	5

Chronic Pain Acceptance Questionnaire (McCracken et al., 2004)

Directions: Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following rating scale to make your choices. For instance, if you believe a statement is 'Always True,' you would circle 6 in the line next to that statement.

	Never true	Very rarely true	Seldom true	Some-times true	Often true	Almost always true	Always true
I am getting on with the business of living no matter what my level of pain is.	0	1	2	3	4	5	6
Although things have changed, I am living a normal life despite my chronic pain.	0	1	2	3	4	5	6
I lead a full life even though I have chronic pain.	0	1	2	3	4	5	6
Keeping my pain level under control takes first priority whenever I'm doing something.	0	1	2	3	4	5	6
Before I can make any serious plans, I have to get some control over my pain.	0	1	2	3	4	5	6
When my pain increases, I can still take care of my responsibilities.	0	1	2	3	4	5	6
I avoid putting myself in situations where my pain might increase.	0	1	2	3	4	5	6
My worries and fears about what pain will do to me are true.	0	1	2	3	4	5	6

Engaged Living Scale (Trompetter et al., 2013)

Instructions: Using the scale provided, decide how much you either disagree or agree with each of the following statements. Circle the number from 1 to 5 that best indicates how you feel.

	Completely Disagree	Disagree	Neutral	Agree	Completely Agree
I have values that give my life more meaning.	1	2	3	4	5
I know what motivates me in life.	1	2	3	4	5
I believe that I've found important values to live according to.	1	2	3	4	5
I know exactly what I want to do with my life.	1	2	3	4	5
I make choices based on my values, even if it is stressful.	1	2	3	4	5
I know how I want to live my life.	1	2	3	4	5
I know what I want to do with my life.	1	2	3	4	5
I believe that my values are really reflected in my behaviour.	1	2	3	4	5
I believe that how I behave fits in with my personal wants and desires.	1	2	3	4	5
My emotions don't hold me back from doing what's important to me.	1	2	3	4	5
I live the way I always intended to live.	1	2	3	4	5
I am satisfied with how I live my life.	1	2	3	4	5
Nothing can stop me from doing something that's important to me.	1	2	3	4	5
I believe that I am living life to the full right now.	1	2	3	4	5
I make time for the things that I consider important.	1	2	3	4	5
I feel that I am living a full life.	1	2	3	4	5

Life Orientation test – Revised (LOT-R, Scheier et al., 1994)

Please be as honest and accurate as you can throughout. Try not to let your response to one statement influence your responses to other statements. There are no "correct" or "incorrect" answers. Answer according to your own feelings, rather than how you think "most people" would answer.

	Strongly disagree	Disagree	Neutral	Agree	Strongly agree
In uncertain times, I usually expect the best.	0	1	2	3	4
It's easy for me to relax.	0	1	2	3	4
If something can go wrong for me, it will.	0	1	2	3	4
I'm always optimistic about my future.	0	1	2	3	4
I enjoy my friends a lot.	0	1	2	3	4
It's important for me to keep busy.	0	1	2	3	4
I hardly ever expect things to go my way.	0	1	2	3	4
I don't get upset too easily.	0	1	2	3	4
I rarely count on good things happening to me.	0	1	2	3	4
Overall, I expect more good things to happen to me than bad.	0	1	2	3	4

Medical Outcomes Study Social Support Survey (Sherbourne & Stewart, 1991)

People sometimes look to others for companionship, assistance, or other types of support. How often is each of the following kinds of support available to you if you need it? Circle one number on each line.

	None of the time	A little of the time	Some of the time	Most of the time	All of the time
To help you if you were confined to bed?	1	2	3	4	5
To take you to the doctor if you need it?	1	2	3	4	5
To prepare your meals if you are unable to do it yourself?	1	2	3	4	5
To help with daily chores if you were sick?	1	2	3	4	5
To have a good time with?	1	2	3	4	5
To turn to for suggestions about how to deal with a personal problem?	1	2	3	4	5
Who understands your problems?	1	2	3	4	5
To love and make you feel wanted?	1	2	3	4	5

Study Three questionnaires that were also used in Study One can be found in Appendix B.

Appendix G – Study Three Data Screening and Variable Distribution

Missing data

Table G1

Means, Standard Deviations of Study Variables Comparing Males to Females

	% missing	Mean (SD)		*t* (p)
		Male (N=59)	Female (N=222)	
Age	.00	56.34 (14.57)	48.51 (13.76)	3.71***
Years Education	.35	4.67 (3.71)	5.16 (3.06)	-.94
Years Pain Duration	.00	11.77 (12.65)	12.30 (10.57)	-.29
Pain-Related Disability	2.83	43.38 (15.76)	41.92 (14.98)	.64
Quality of Life	1.61	62.02 (18.79)	64.33 (17.73)	-.36
Pain Severity	2.82	5.40 (1.63)	5.39 (1.77)	.04
Negative Affect	.64	12.76 (5.49)	12.13 (4.46)	.82
Catastrophising	1.20	37.55 (14.29)	35.15 (11.80)	1.68
Fear of movement	.29	28.51 (5.40)	25.54 (5.84)	3.69***
Solicitous partner responses	1.77	3.07 (1.11)	3.23 (1.03)	-.98
Punishing partner responses	1.86	1.77 (1.12)	1.85 (1.12)	-.50
Lack of understanding - medical	.12	2.20 (.86)	2.39 (.95)	-1.50
Lack of understanding - family	3.42	2.55 (1.00)	2.83 (1.12)	-1.86
Discounting - medical	3.18	2.09 (.88)	2.25 (.93)	-1.27
Discounting - family	3.60	2.27 (1.10)	2.62 (.93)	-2.28*
Pain Self-Efficacy	1.20	30.10 (11.86)	31.32 (11.34)	-.73
Pain Acceptance	.18	19.78 (8.38)	21.22 (7.55)	-1.20
Values awareness – Values Living	.25	35.19 (6.84)	37.61 (6.29)	-2.46*
Positive affect	2.82	15.10 (4.42)	15.25 (3.65)	-.24
Optimism	.29	5.60 (3.04)	6.25 (2.67)	-1.51
Emotional social support	.08	14.00 (4.59)	13.24 (4.14)	1.16
Instrumental social support	.27*	14.23 (5.36)	12.67 (4.93)	2.03*

Note 1 ***$p < .001$, **$p < .01$, *$p < .05$

Note 2. * in the column reporting proportion of missing data indicates a statistically significant value of Little's Missing Completely at Random test suggesting data were not missing at random.

Assessment of Normality

Each variable was assessed for normality and outliers in SPSS 20.0 (IBM Corp., 2012). Histograms with a normality plot, box plots and detrended normal quantile-quantile (Q-Q) plots were examined. Skewness and kurtosis values were assessed. Recommended thresholds of these values are +/- 1.5 (Tabachnik & Fidell, 2013). Kolmogorov-Smirnov and Shapiro-Wilk statistics were also used to assess significance of skewness and kurtosis values. These methods of assessing normality are discussed in more detail in Appendix C.

Results indicated that most variables had a substantively normal distribution. Skewness and kurtosis values for all variables except years of education and pain duration were all well within the recommended level of +/- 1.5 (Tabachnik & Fidell, 2013; Table G1). One extreme outlier was identified in both years of education (27 years, variable mean 5.06 years) and pain duration (69 years, variable mean 12.19 years). These values were both replaced with the variable mean, as is suggested by Tabachnik and Fidell (2013). This achieved skewness and kurtosis values for both variables that were below the recommended thresholds of +/- 1.5 (Tabachnik & Fidell, 2013). Because the differences between the mean and the five per cent trimmed mean were small (See Table G2) a decision was made to retain these high outliers in the dataset.

Table G2

Skewness and Kurtosis Values of Study Three Distributions

	Skewness	*Kurtosis*
Age	.01	-.70
Years Education	.61	.19
Pain Duration	1.36	1.49
Pain-Related Disability	-.59	-.38
Quality of Life	-.08	-.51
Pain severity	-.28	-.04
Negative Affect	.49	-.47
Catastrophising	.38	-.70
Fear-avoidance	.04	.04
Punishing partner response	.79	.32
Solicitous partner responses	-.18	-.63
Lack understanding – Medical	.38	-.34
Lack understanding – Family	.12	-.93
Discounting - Medical	.62	-.08
Discounting - Family	.45	-.76
Pain self-efficacy	.32	-.66
Pain acceptance	.02	-.22
Positive Affect	-.26	-.52
Optimism	-.10	-.34
Values Awareness	-.45	.70
Instrumental Social Support	-.25	-1.07
Emotional Social Support	-.24	-.86

Table G3

Means of variables and five per cent trimmed means

Variable	Mean	5% Trimmed mean
Age	50.15	50.22
Years Education	5.06	4.09
Pain Duration	11.98	11.00
Pain-Related Disability	42.23	42.80
Quality of Life	63.84	63.93
Pain severity	5.39	5.43
Negative Affect	12.26	12.09
Catastrophising	38.86	34.50
Fear-avoidance	26.16	26.12
Punishing partner response	1.84	1.78
Solicitous partner responses	3.20	3.21
Lack understanding - Medical	2.77	2.76
Lack understanding - Family	2.35	2.32
Discounting - Medical	2.54	2.50
Discounting - Family	2.22	2.17
Pain self-efficacy	31.06	30.79
Pain acceptance	20.92	20.91
Positive Affect	15.22	15.27
Optimism	6.11	6.11
Values Awareness	37.10	37.27
Instrumental Social Support	12.99	13.10
Emotional Social Support	13.40	13.49

Assessing for multivariate outliers

As explained in Section 7.7.4.2 in Chapter Seven, Mahalanobis distances were calculated to assess for multivariate outliers in the risk-resistance regression models. Only a single multivariate outlier was identified in model predicting disability, however, its presence did impact results, so it was removed from the analysis. The value of this outlier can be seen below in Table G4.

Table G4

Multivariate Outliers Risk-Resistance Models Predicting Pain-Related Disability and QOL – Mahalanobis Distances

DV	Critical value	Number of values observed over critical value	Values observed over critical value
Pain-related disability	29.59	1	35.41
Quality of life	31.26	-	-

Note. DV = Dependent variable

Multivariate outliers were also checked for each of the regression analyses of indirect effects completed using PROCESS (Hayes, 2013). Four independent variables were entered in each of these analyses. Predictors were age, years education, the five mediators, catastrophising, fear-avoidance, pain self-efficacy, pain acceptance and values-based living and then each of the predictors. For pain-related disability these were pain severity and negative and positive affect. For models predicting QOL, predictors were age, the four mediators, catastrophising, pain self-efficacy, pain acceptance and values-based living and then the predictors which were pain severity and negative and positive affect, optimism and emotioanl and instrumental social support.

The critical value for Mahalanobis distance for the models testing mediation effects that predicted pain-related disability was 26.13 and for models predicting QOL was 18.55 as indicated by the Chi square critical values table for eight predictors at the level of .001 (Tabachnik & Fidell, 2013). A single case was identified in three models predicting pain-related disability, a single case was also identified in five models predicting QOL. See Table G5. Again, these multivariate outliers were retained in the analyses because of the very small number of cases identified which were close

to critical values. To ensure these outliers did not influence results, the analyses were repeated in a dataset that had these cases deleted, with the same results noted.

Table G5

Multivariate Outliers Parallel Mediator Models Analyses Predicting Pain-Related Disability–Mahalanobis Distances

IV	Critical value	Number of values observed over critical value	Values observed over critical value
Pain severity	26.13	1	26.59
Negative Affect	26.13	1	28.40
Positive Affect	26.13	1	32.71

Table G6

Multivariate Outliers Parallel Mediator Models Analyses Predicting Quality of Life–Mahalanobis Distances

IV	Critical value	Number of values observed over critical value	Values observed over critical value
Pain severity	18.55	1	20.08
Negative Affect	18.55	1	20.08
Positive Affect	18.55	1	20.35
Optimism	18.55	1	20.78
Emotional Support	18.55	1	21.14
Instrumental Support	18.55	1	23.36

The critical value for Mahalanobis distance for the models testing moderation effects that predicted pain-related disability was 18.47 and for models predicting QOL was 16.27 as indicated by the Chi square critical vales table for four and three predictors respectively at the level of .001 (Tabachnik & Fidell, 2013). A single case was identified in five of the models predicting pain-related disability and a single case was identified in one model that predicted QOL. These cases are listed for each analysis in Tables G7 and G8. Again, these multivariate outliers were retained in the analyses because of the very small number of cases identified which were close to critical values. The analyses were repeated for the cases that had identified a significant moderation effect in a dataset that had these cases deleted, with the same results noted.

Table G7

Multivariate Outliers Moderation Analyses Predicting Pain-Related Disability – Mahalanobis Distances

IV	Moderator	Critical value	Cases over critical value	Value of case over critical value
Pain	Acceptance	18.47	1	19.03
Catastrophising	Acceptance	18.47	1	19.52
Negative Affect	Acceptance	18.47	1	19.80
Catastrophising	Instrumental support	18.47	1	18.91
Negative Affect	Instrumental support	18.47	1	19.01

Table G8

Multivariate Outliers Moderation Analyses Predicting Quality of Life – Mahalanobis Distances

IV	Moderator	Critical value	Cases observed over critical value	Values of case over critical value
Negative affect	Emotional Support	16.27	1	16.57

Appendix H – Living Well with Chronic Pain

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Living well with chronic pain

Joanne Sheedy[a], Louise McLean[a], Kate Jacobs[a] and Lou Sanderson[b]

[a]Monash University Education Faculty, Clayton, VIC, Australia; [b]Kardinia Health, Belmont, VIC, Australia

ABSTRACT

Objective: The aim of this study was to explore factors associated with resilience in chronic pain.

Methods: A sequential mixed methods design was used. Six individuals reporting low levels of psychological distress on the Depression, Anxiety and Stress Scale and low levels of pain-related disability on the Roland Morris Disability Questionnaire were interviewed about their experiences of living with chronic pain. Participants were recruited from a general medical practice in Victoria, Australia and were interviewed between May and September 2013. Potential participants were identified by treating health professionals as individuals who appeared to be coping well with chronic pain.

Results: Factors associated with resilience in chronic pain included social support, confidence for physical activity, optimism and positive thinking. Caring for others was also identified as a novel protective factor. Values driven behaviour may have motivated some participants to optimise their pain management via active coping approaches.

Conclusions and implications for practice: This study highlights protective factors that may contribute to resilience in chronic pain. Improved understanding of such factors may help with development of interventions to promote better adjustment to chronic pain conditions.

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Resilience; chronic; pain; coping; primary; care

Introduction

Chronic pain is a heterogeneous disorder with high prevalence in Western societies (Blyth et al., 2001). It is associated with a number of negative health sequelae such as psychological distress and high levels of disability (Bair, Robinson, Katon, & Kroenke, 2003; Raftery et al., 2011). A number of mechanisms have been suggested to explain how chronic pain exerts its negative effects on health. Increased physiological burden as a result of the stress associated with living with pain has been implicated (Sturgeon & Zautra, 2010) and elevated rates of anxiety and depression are frequently reported (Asmundson, Jacobson, Allerdings, & Norton, 1996; Banks & Kerns, 1996; Dersh, Gatchel, Mayer, Polatin, & Temple, 2006). Avoidant coping behaviours often increase disability (Holmes & Stevenson, 1990) and cognitive factors such as fear of movement have been associated with poorer pain outcomes (Arnow et al., 2011; Somers et al., 2009; Thompson, Urmston, Oldham, & Woby, 2010; Vlaeyen et al., 1995; Vlaeyen & Linton, 2000).

CONTACT Joanne Sheedy joanne.sheedy@monash.edu
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The negative factors contributing to poor pain outcomes are well recognised (Dersh et al., 2006), however, research attention has only recently focused on the protective role of positive psychological factors. The positive psychology literature suggests that personal strengths and positive qualities can provide adjustment benefits for varying life situations (Steptoe, Dockray, & Wardle, 2009; Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000). There is also evidence to show that some positive factors, such as optimism, pain acceptance, positive affect and self-efficacy may reduce the negative impact of chronic pain (Hanssen et al., 2013; McCracken & Zhao-O'Brien, 2010; Vancleef & Peters, 2011; Wright et al., 2011; Zautra, Johnson, & Davis, 2005). Improved understanding of protective factors may help develop interventions to promote improved chronic pain adjustment outcomes.

It is likely that protective factors may be most evident in those who are functioning well despite chronic pain, however the concept of resilience in chronic pain is under-researched. Most studies investigating resilience have not used a well-functioning sample (Lynch, Sloane, Sinclair, & Bassett, 2013; Newton-John, Mason, & Hunter, 2014; Ong, Zautra, & Carrington Reid, 2010; Ramírez-Maestre, Esteve, & López, 2012; Sofaer-Bennett, Moore, Lamberty, & O'Dwyer, 2007; West, Buettner, Stewart, Foster, & Usher, 2012; Wright, Zautra, & Going, 2008). This study, therefore, aimed to explore the experience of resilience and its associated processes in a group of individuals thought to be functioning well, or displaying resilience, in living with chronic pain.

Methodology

Research design

The research design was a sequential mixed methods design in which qualitative and quantitative data were used to address different aspects of the research aims (Creswell, 2009). Before proceeding to interview, validated measures of anxiety, depression and stress pain-related disability were completed. This quantitative aspect of the research provided a measure of the participants' physical and psychological function and was important because of the specific nature of the sample sought for the research. A qualitative approach then used semi-structured individual interviews to explore the experience of living with chronic pain. In the interviews, there was a particular focus on protective factors that may have promoted pain coping.

Ethical considerations

Ethics approval for the study was granted by the Monash University Human Research Ethics Committee.

Measures

The Depression, Anxiety and Stress Scale (DASS) (Lovibond & Lovibond, 1995) was used to quantify symptoms of anxiety and depression. The original 42 item DASS has been demonstrated to have good reliability and validity (Lovibond & Lovibond, 1995). The shortened 21 item version of the DASS used in this study divides the items evenly into three subscales assessing anxiety, depression and stress symptoms. Respondents are

asked to indicate how frequently they have experienced that symptom in the past week. Items are scored between zero (*never*) and three (*almost always*). The subscales are scored by summing the item responses; higher scores reflect higher levels of anxiety, stress or depression. The DASS 21 has good reported reliability, with Cronbach's alphas reported as .94 for the depression subscale, .87 for the anxiety subscale and .91 for the stress subscale (Antony, Bieling, Cox, Enns, & Swinson, 1998).

Participants also completed a Roland Morris Disability Questionnaire (RMDQ) (Roland & Morris, 1983) to measure their pain-related disability. The original RMDQ was developed for assessing the functional impact of back pain. The modified RMDQ used in this study asks respondents to relate the items to their pain, regardless of its site. It consists of 24 items, which are answered by checking if the item applies as true on that day. The questionnaire is scored by summing the number of items checked as true. Higher scores indicate greater disability. The RMDQ is reported to be valid and reliable, Cronbach's alpha is reported as .92 (Roland & Morris, 1983).

Procedure

Participants were recruited through a multidisciplinary general practice that incorporated general medical practitioners (GPs), nurses, physiotherapists, exercise physiologists, dieticians and podiatrists. Inclusion criteria included a diagnosis of non-malignant chronic pain and age over 18. Following ethics approval, the Clinical Director of the practice completed a clinic database search, using the terms 'chronic pain' or 'fibromyalgia' which identified 90 patients. Guidelines developed by the researchers were provided to clinicians to then identify well-functioning patients from this initial list. Guidelines suggested suitable participants might be individuals who had not required referral for specialist pain management and who appeared to be coping well both physically and psychologically with pain. The practice posted letters to these individuals inviting them to contact the researchers by telephone, email or post if they wished to participate in the research. Upon receipt of contact, the first author telephoned the person, explained the study and obtained verbal consent. Potential participants were then further screened at the time of the interview and written consent was obtained. Participants with scores of 'severe elevation' in any domain of the DASS and/or those who endorsed more than half of the disability items on the RMDQ were interviewed but these data were not included in the current study.

In order to ensure adequate follow up of individuals who may have reported elevated levels of psychological distress or disability, participants whose scores on the DASS fell in the severely or extremely severely elevated range in any domain, or were above 18 checked items on the RMDQ (Roland & Morris, 1983) were referred back to their GP. If such a referral was deemed necessary, written consent for this was obtained at the conclusion of the interview process. Of those screened, three individuals were referred back to their GP for review.

Semi-structured interview

Semi-structured interviews were used to explore participant's experiences of living with chronic pain. The interview schedule was developed by the researchers and was based

on the research aims and literature review. Questions were open-ended and addressed coping with pain, the process of adjusting to life with pain and factors that enhanced coping. Examples of questions included: 'Do you feel that you went through a process to learn to adapt to the living with the pain? If so, what was this process like?' The first author conducted all interviews, which lasted between 45 and 70 minutes. With consent, all interviews were audio recorded and later transcribed.

Participants

Ten individuals were screened for participation in the research. Of these, six individuals (one male, five females) aged between 33 and 65 years old met eligibility criteria and participated in the current study. Table 1 presents participant demographics.

Data analyses

Quantitative analyses

Descriptive statistics of questionnaire data were analysed with the Statistical Package for the Social Sciences Version 20.0 (IBM Corp., 2011).

Qualitative analyses

Thematic analysis was used to analyse the data using the six-stage process outlined by Braun and Clark (2006). Initially data were coded descriptively by content and then

Table 1. Participant demographic details.

Demographic	Number or mean (SD)
Gender	
Male	1
Female	5
Age	
Mean (SD)	47.00 years (10.56)
Range	33–65 years
Education level	
Year 11	2
Diploma	2
Bachelor Degree	2
Marital status	
Married/defacto	4
Single	2
Employment status	
Unemployed	2
Working part-time	1
Working full time	1
Carer/parent	1
Retired	1
Pain duration	
Mean (SD)	5.58 years (3.01)
Range	(1.5–10 years)
Diagnoses	
Fibromyalgia	3
Low back pain and fibromyalgia	2
Osteoarthritis and fibromyalgia	1

categorised into possible themes. The content from one interview was allowed to inform subsequent ones. Themes common across multiple interviews were identified and highlighted. Definitions of each theme were recorded by the first author to promote coding consistency. A pragmatic approach to the analysis was adopted in which the subjective nature of the data analysis process was acknowledged (Mertens, 2014). Consideration was given to the broader meanings in the data and these were referenced within the context of the literature.

Following the interview, participants were provided with an electronic version of their transcript to confirm accuracy and to ensure participants consented to use of the entire transcript in the analyses. The first two authors cross-checked themes and the data on which they were based from the original transcripts. They also re-examined the initial analysis and agreed on the superordinate and subordinate themes which were then mapped.

Results

Questionnaire profiles

Only one participant reported anxiety, depression and stress symptom scores within the range classified as 'normal' together with a low disability score. The remaining participants reported mild to moderate elevations in at least one of the anxiety, depression or stress subscales, and endorsed half or less of the disability items. Table 2 shows participant adjustment profiles.

Two main themes were identified – resilience processes and negative pain-related experiences. Resilience processes refers to positive factors reported in the current research that appeared to be linked to improved pain coping and better function. Resilience processes were further coded into eight subcategories. These were stoicism, confidence and motivation to manage pain, having a management plan, physical activity, cognitive strategies, support from health professionals and getting a diagnosis. Negative pain-related experiences were those experiences that appeared to make life with chronic pain more difficult. These factors were further coded into four subcategories. These were pain-related losses, negative social experiences and negative impacts on mood and sense of self. Participant quotes are presented using pseudonyms.

Resilience processes

Stoicism. All participants said they did not want to 'give in' to pain and most felt this was a part of their personality that had pre-dated their pain.

Table 2. Medication, measures of depression, anxiety, stress and disability.

Pseudonym	Medication	Depression	Anxiety	Stress	Disability
Sally	Paracetamol, AD	Normal	Mild elevation	Normal	2/24
Bettina	Low dose AD	Normal	Moderate elevation	Mild elevation	2/24
Jane	–	Normal	Moderate elevation	Normal	6/24
Jacqui	Low dose AD	Moderate elevation	Mild elevation	Normal	4/24
Kim	Paracetamol, AD, AI	Moderate elevation	Moderate elevation	Normal	12/24
Michael	Oral opioid	Normal	Normal	Normal	4/24

Note: AD = anti-depressant and AI = anti-inflammatory.

> I don't know if it's determination or just not willing to give in … it's almost like … something's trying to stop me doing stuff so I'm trying to fight against that. It's not like I'm super strong. It's just I just don't want this to get me. (Jane, aged 51)

Confidence to manage pain. Michael, Jacqui and Sally described their resilience as arising from their families or earlier life experiences and said they had an innate sense of confidence that they could cope depsite their pain. 'Yeah, I mean we've always been that sort of a family where you pick yourself up and get on with it "what are you whinging about?" … and that's … what keeps me going' (Jacqui, 45 years). Kim, Bettina and Sally recognised that recovery was up to them. 'I mean essentially everything is up to you at the end of the day, how you steer your life after something has happened' (Kim, aged 33).

Motivation to manage pain – working and caring for others. Sally and Jane both reported they were motivated to cope with pain because they needed to work. Sally also needed to take care of her elderly parents.

> If I don't do what's right (for me) I can't … fulfil my role in my family unit. Because I still have to earn an income and I have a father with dementia. And a mother that is caring for him so I have to be able to function to support them. (Sally, aged 44)

Caring responsibilities for others fostered a sense of resilience. Kim recognised that playing a supportive role in her young niece's life motivated her to keep active. Jacqui said she wanted to preserve her relationship with her family.

> *So that was probably the kick up the backside that I needed. I wasn't going to let pain … ruin a relationship with my Mum and my kids. They deserved better, so that was probably the incentive I suppose to get up and have a go.* (Jacqui, aged 45)

Having a management plan. All participants said a specific management plan was important. For many, this meant keeping physically active, accessing professional treatment, getting adequate sleep and optimising medications. There was emphasis on discovering an effective approach and then committing to adherence.

Physical activity. Most participants believed that exercise was essential to health, even when in pain, and reported that working with GPs, physiotherapists and exercise physiologists helped them slowly become more active.

> Being more confident to take on physical exercise (helped me) … my GP did help. She encouraged me, 'Don't be scared of that. If you do it and it's too hard, don't push yourself, but if you try and you get some benefit from it, then that … would be better for you.' Again, it was just hearing it from someone that I trusted that I thought, of course, I should be doing that sort of thing. (Bettina, aged 44)

Cognitive strategies. All participants reported a range of cognitive strategies they used in order to cope with chronic pain. Pain acceptance was mentioned frequently.

> Well you can be a victim … but it's not going to change it, it's still going to be there … So you can wallow in self-pity or you can accept the fact that this is the way it's going to be. So I suppose it's almost like going through a grieving period, and you can go 'Right, I can sit around the house and not get on with my life' or 'I can get out there and enjoy myself and get on with it and to do that, well this is what I need to do. (Sally, aged 44)

Bettina reported that acceptance was linked to getting a diagnosis. She was also clear that acceptance did not mean giving up and was in fact linked to optimism.

> I'll try and accept that I've got this banner now and I sit under it but I can … move beyond it … I'm just going to have to make positive, informed choices about how I will treat the condition and in so doing be able to take on more normal things again. (Bettina, aged 44)

Other reported strategies included positive thinking. Kim described making an effort to 'be in the moment', while Bettina, Kim and Jane mentioned optimism and hope. Participants reported that making mental comparisons to those worse off than them aided coping.

Support from health professionals. Several participants reported that health professionals who took their symptoms seriously fostered their ability to cope. 'I think that's been … a good part of the coping mechanism, is that you've got someone you can rely on' (Michael, aged 65).

Getting a diagnosis. Five participants said getting a diagnosis legitimised treatment, helped plan management and promoted acceptance. For some, a diagnosis represented a turning point in recovery.

> It wasn't until they finally diagnosed fibromyalgia and I think it was then more the fact that they could say to me 'This is what you have got' and then you go 'Right I am not an idiot, I am not a hypochondriac, there is something wrong with me, I can get on and I can deal with this. (Sally, aged 44)

Positive social experiences. Four participants said that both instrumental and emotional support fostered coping, although Jacqui said she didn't want to accept help if it was motivated by pity. Feeling understood was reported to be important.

> It's just having them show a little bit of understanding. We don't have to have a big conversation about it, but if a friend says … it would probably be better for you if we drove rather than catch the tram or the train, just that one little thing … showing me that they understand. (Kim, aged 33)

Negative pain-related experiences

Pain-related losses. Pain-related losses included physical, social, financial and work-related losses and were reported by all participants.

> It's not being able to … do the physical things the way that you have done them in the past, you try to do something and you just get stopped by a wall of pain. So you don't do it, and then you don't do it again. (Michael, aged 65).

Negative social experiences. Pain was reported to impact negatively on relationships, especially with partners. Other negative social experiences included lack of understanding from others and decreased social connectedness. Invalidation was frequently described and was associated with the invisibility of chronic pain. 'Because it's an invisible illness, and there's that constant feeling that … people don't think your pain is genuine, or they think it's all in your head' (Kim, aged 33).

Negative impacts on sense of self. Several participants reported negative effects of pain on their sense of self, confidence and self-esteem. Other participants described earlier, preferred versions of themselves that pre-dated their pain. Sally and Jane reported that their sense of self was unchanged by pain. 'No, I think I've just, I've just proved probably that I'm tough, tougher than I thought I was' (Jane, aged 51).

Negative effects of pain on mood. All participants described the negative pain-related emotional experiences. These included anger, anxiety and intolerance. 'I can lose my temper quite easily, where I was never short-tempered' (Michael, aged 65).

Discussion

This research aimed to explore protective factors associated with living well with chronic pain. Despite seeking a well-functioning or resilient sample for the research, most participants reported elevated levels of psychological distress and pain-related disability. Because health professionals identified the initial list of possible participants, this suggests some individuals may have masked their pain-related distress during consultations.

Caring for others and a desire to preserve relationships appeared to increase motivation to cope with pain and represent novel themes not widely described. Innate confidence for managing pain was also described. Other identified themes were consistent with previously reported findings of chronic pain qualitative research (Bunzli, Watkins, Smith, Schutze, & O'Sullivan, 2013; Snelgrove & Liossi, 2013). Positive themes included acceptance, stoicism, keeping positive and social support. Negative themes included pain-related losses, effects of pain on sense of self, mood and negative social interactions. These latter themes are also consistent with the small number of qualitative studies that have specifically examined resilience in chronic pain (Evans & de Souza, 2008; West et al., 2012).

Caring responsibilities to others appeared to motivate some participants to cope with pain. The health and mortality benefits of caring for others are well recognised in the general health literature (Momtaz, Rahimah & Hamid, 2014; Okun, Yeung, & Brown, 2013; Poulin, 2014) but have rarely been mentioned in chronic pain research. In fact, some qualitative pain research has emphasised the negative aspects of caring responsibilities, including isolation and the conflict between caring and wage earning (Zheng et al., 2013). In this study, caring for others appeared to represent a previously undocumented potential source of resilience for those with chronic pain.

Motivation may be an important factor influencing resilience. Jensen, Nielsen and Kerns' (2003) motivational theory of chronic pain suggested readiness to engage in adaptive pain self-management is influenced not only by confidence to adopt such behaviours but also by beliefs about the importance of them. This latter factor may in turn be influenced by values. Participants in this sample expressed a belief in the importance of coping. They continued to engage in the values-based activities and relationships that mattered to them. This is consistent with research that has highlighted that values awareness confers benefits for pain adjustment (Vowles & McCracken, 2008).

In addition to being motivated by values, coping in this sample appeared to be influenced by learning as several participants described their resilient approach to pain as arising from earlier life experiences (Folkman & Moskowitz, 2004). Participants also

attributed their active coping approach to dispositional traits or simply how they were as a person. This description is consistent with findings from a qualitative study of 13 injured workers who described pain coping as an intrinsic and life-defining attitude that arises through modelling by parents and significant others (Carroll, Rothe, & Ozegovic, 2013). Teaching patients that coping skills can be learned or modified may help some people live better with chronic pain.

All participants endorsed a belief in the importance of physical activity with most working with health professionals to become more active. Physical activity was reported to be linked to improved mood and social connectedness. These observations reflect much previous research demonstrating the positive influence of confidence for physical activity on functional impairment in chronic pain (Vlaeyen et al., 1995; Vlaeyen & Linton, 2000).

Most participants in this research did not report a strong negative impact of pain on their sense of self. This finding is interesting as identity reconstruction has been identified as a major task for those living with chronic pain (Risdon, Eccleston, Crombez & McCracken, 2003). The current findings are in contrast to previous qualitative research that has reported profound effects of chronic pain on sense of self (Smith & Osborn, 2007; Soklaridis, Cartmill, & Cassidy, 2011). These differences may reflect the well-functioning nature of the current sample, but may also be explained by the self-enmeshment theory of pain (Morley, Davies, & Barton, 2005). This theory suggests that emotional adjustment to chronic pain is partly determined by the extent to which aspects of the self are enmeshed with pain (Sutherland & Morley, 2008). It proposes that continued engagement in valued life activities despite pain may be a defining characteristic of those whose sense of self is not negatively impact upon by pain.

A frequent focus of discussion concerned the negative impact of pain on social experiences and interactions. This is consistent with previous research (Silva, Sampaio, Mancini, Luz, & Alcântara, 2011; Sofaer-Bennett et al., 2007; Soklaridis et al., 2011). Invalidation was frequently mentioned in this sample and was experienced in relationships with partners, friends and health professionals. Invalidation has previously been described as a significant negative consequence of chronic pain (Upshur, Bacigalupe, & Luckmann, 2010).

By contrast, validating communication by health-care providers has been associated with more positive affect and significantly less worry (Linton, Boersma, Vangronsveld, & Fruzzetti, 2012). High quality health-care relationships characterised by open interactions and symptom validation have also been associated with improved outcomes in pain rehabilitation (Oosterhof, Dekker, Sloots, Bartels, & Dekker, 2014). Consistent with this, participants reported supportive relationships with health-care providers, particularly their GP, significantly fostered their coping efforts. These findings taken together suggest that communication skills training for health-care professionals regarding chronic pain may provide adjustment benefits for patients.

Strengths and limitations

The high predominance of fibromyalgia in this sample may have contributed to findings that were particular to that disorder. The sample was recruited from a single clinic and this may have biased results towards a specific demographic. The small sample size of the current study is a limitation, however, the use of validated measures to purposely select

a well-functioning sample represents a strength of the study. Importantly, this is one of the first studies to explore adjustment processes in this specific sample of individuals with chronic pain. For this reason, it may offer unique insights into positive adjustment processes in chronic pain.

Implications for mental health professionals

The current research highlights several positive factors that mental health professionals could incorporate into interventions to improve chronic pain outcomes. Consistent with other research demonstrating benefits of values-based interventions for those living with chronic pain (Vowles & McCracken, 2008), the current results suggest that encouraging those with chronic pain to define their values and live in accordance with them may confer adjustment benefits. Continued engagement in important relationships may help reduce the negative impact of pain on an individual's sense of self. In addition, empowering individuals to value the supportive roles they may play in the lives of others may improve adjustment outcomes.

An important finding of the current research relates to the communication style of health professionals. Specifically, participants reported that a validating communication style and empathic support from treating health professionals significantly fostered their ability to cope with chronic pain. It is important that health professionals understand the potential impact of their communication style on the coping style of individuals with chronic pain.

Conclusions

This study highlights some important factors that may contribute to resilience in chronic pain. Caring for others, optimism and physical activity all appeared to foster resilience. Active pain coping also appeared to be associated with resilience, although the reasons that some people engage in active coping while others do not remain unclear. The drivers of these behaviours are likely multifactorial and may include trait differences and learning histories. Motivation to continue to engage in valued life activities may be an important protective factor that influences adjustment via coping.

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Appendix I – Results of Moderation Analyses

Table I1

Summary of Moderation Analyses Predicting Pain-Related Disability (10,000 Bootstrap Samples)

Predictor (X)	Coefficient (SE) Interaction term [95% BC CIs]	Model summary R^2	△ R_2 due to interaction
Moderator: Pain Self-Efficacy			
Pain Intensity	1.15 (.95) [-.71; 3.02]	.61, *F*(5, 275) = 87.70***	.00
Catastrophising	.04 (.14) [-.23; .30]	.56, *F*(5, 275) = 68.78***	.00
Fear-avoidance	.09 (.28) [-.45; .63]	.55, *F*(5, 275) = 67.96***	.00
Negative Affect	-.08 (.35) [-.76; .60]	.56, *F*(5, 275) = 70.30***	.00
Moderator: Pain Acceptance			
Pain Intensity	.13 (.05) [.04; .22]#	.54, *F*(5, 275) = 64.13***	.01
Catastrophising	.01 (.01) .00; .02]	.42, *F*(5, 275) = 39.35***	.01
Fear-avoidance	.01 (.01) [-.01; .04]	.41, *F*(5, 275) = 38.01***	.00
Negative Affect	.05 (.02) [.01; .08]#	.44, *F*(5, 275) = 43.25***	.01
Moderator: Values-Based Living			
Pain Intensity	.05 (.04) [-.02; .12]	.44, *F*(5, 275) = 42.47***	.00
Catastrophising	.01 (.01) [.00; .02]	.32, *F*(5, 275) = 26.28***	.01
Fear-avoidance	.00 (.01) [-.02; .03]	.30, *F*(5, 275) = 23.79***	.00
Negative Affect	.03 (.01) [.01; .06]#	.32, *F*(5, 275) = 25.49***	.01
Moderator: Positive Affect			
Pain Intensity	.13 (.10) [-.07; .33]	.42, *F*(5, 275) = 40.08***	.00
Catastrophising	.04 (.02) [.00; .07]	.31, *F*(5, 275) = 24.43***	.01
Fear-avoidance	.04 (.03) [-.03; .10]	.26, *F*(5, 275) = 19.38***	.00
Negative Affect	.06 (.04) [-.03; .04]	.30, *F*(5, 275) = 23.51***	.00
Moderator: Optimism			
Pain Intensity	.20 (.16) [-.10; .51]	.39, *F*(5, 275) = 35.53***	.00
Catastrophising	.00 (.02) [-.04; .05]	.24, *F*(5, 275) = 17.18***	.00
Fear-avoidance	.01 (.05) [-.10; .11]	.19, *F*(5, 275) = 12.88***	.00
Negative Affect	.03 (.06) [-.09; .15]	.25, *F*(5, 275) = 17.86***	.00
Moderator: Emotional Social Support			
Pain Intensity	.17 (.10) [-.02; .36]	.38, *F*(5, 275) = 33.77***	.01
Catastrophising	.00 (.01) [-.03; .03]	.25, *F*(5, 275) = 18.48***	.00
Fear-avoidance	.01 (.03) [-.05; .08]	.20, *F*(5, 275) = 14.02***	.00
Negative Affect	.03 (.04) [-.05; .10]	.25, *F*(5, 275) = 18.34***	.00
Moderator: Instrumental Social Support			
Pain Intensity	.15 (.08) [-.01; .31]	.37, *F*(5, 275) = 32.43***	.01
Catastrophising	.00 (.01) [-.02; .03]	.24, *F*(5, 275) = 17.22***	.00
Fear-avoidance	-.01 (.03) [-.06; .05]	.18, *F*(5, 275) = 12.21***	.00
Negative Affect	.05 (.03) [-.02; .11]	.25, *F*(5, 275) = 18.37***	.01

Note 1. Unstandardized *b* coefficients reported, effects adjusted for covariates age and years education.
Note 2. #Indicates significant indirect effect based on bias-corrected confidence intervals
CI indicates confidence interval; M, moderating variable. ***p<.001, **p<.01, *p<.05

Table I2

Summary of Moderation Analyses Predicting Quality of Life (10,000 Bootstrap Samples)

Predictor (X)	Coefficient (SE) Interaction term [95% BC CIs]	Model summary R^2	△ R_2 due to interaction
Moderator: Pain Self-Efficacy			
Pain Intensity	.05 (.03) [-.01; 12]	.57, *F*(3, 277) = 122.05***	.00
Catastrophising	.01 (.01) [.00; .02]	.55, *F*(3, 277) = 113.78***	.00
Fear-avoidance	.00 (.01) [-.02; 02]	.54, *F*(3, 277) = 110.38***	.00
Negative Affect	.01 (.01) [-.02; .03]	.61, *F*(3, 277) = 141.63***	.00
Moderator: Pain Acceptance			
Pain Intensity	-.01 (.06) [-.12; 10]	.50, *F*(3, 277) = 69.50***	.00
Catastrophising	.01 (.01) [-.01; .02]	.45, *F*(3, 277) = 57.34***	.00
Fear-avoidance	.01 (.02) [-.02; 04]	.45, *F*(3, 277) = 56.29***	.00
Negative Affect	-.01 (.02) [-.05; .03]	.52, *F*(3, 277) = 75.22***	.00
Moderator: Values-Based Living			
Pain Intensity	-.10 (.07) [-.24; .05]	.46, *F*(3, 277) = 58.50***	.00
Catastrophising	.00 (.01) [-.02; .02]	.40, *F*(3, 277) = 45.65***	.00
Fear-avoidance	.00 (.02) [-.04; .04]	.37, *F*(3, 277) = 40.47***	.00
Negative Affect	-.03 (.03) [-.07; .02]	.45, *F*(3, 277) = 57.05***	.00
Moderator: Positive Affect			
Pain Intensity	.00 (.12) [-.24; .24]	.40, *F*(3, 277) = 46.82***	.00
Catastrophising	-.02 (.02) [-.05; .02]	.37, *F*(3, 277) = 39.72***	.00
Fear-avoidance	-.07 (.04) [-.15; .00]	.32, *F*(3, 277) = 33.06***	.01
Negative Affect	.04 (.05) [-.05; .13]	.44, *F*(3, 277) = 54.85***	.00
Moderator: Optimism			
Pain Intensity	-.04 (.19) [-.41; .34]	.36, *F*(3, 277) = 38.79***	.00
Catastrophising	.00 (.03) [-.05; .05]	.29, *F*(3, 277) = 27.55***	.00
Fear-avoidance	-.01 (.06) [-.13; .11]	.23, *F*(3, 277) = 20.24***	.00
Negative Affect	-.01 (.07) [-.13; .12]	.38, *F*(3, 277) = 41.68***	.00
Moderator: Emotional Social Support			
Pain Intensity	-.05 (.11) [-.27; .16]	.43, *F*(3, 277) = 51.83***	.00
Catastrophising	-.02 (.02) [-.05; .01]	.40, *F*(3, 277) = 45.51***	.00
Fear-avoidance	-.04 (.04) [-.12; .02]	.35, *F*(3, 277) = 36.64***	.00
Negative Affect	-.05 (.04) [-.12; .03]	.45, *F*(3, 277) = 55.68***	.00
Moderator: Instrumental Social Support			
Pain Intensity	-.09 (.10) [-.29; .10]	.33, *F*(3, 277) = 34.52***	.00
Catastrophising	.01 (.01) [-.02; .03]	.30, *F*(3, 277) = 29.89***	.00
Fear-avoidance	.04 (.03) [-.02; .10]	.55, *F*(3, 277) = 67.96***	.00
Negative Affect	.00 (.03) [-.07; .07]	.39, *F*(3, 277) = 44.73***	.00

Note 1. Unstandardized b coefficients reported, effects adjusted for age.
Note 2. #Indicates significant indirect effect based on bias-corrected confidence intervals, CI indicates confidence interval; M, moderating variable.