RELATIONSHIPS BETWEEN PAIN-RELATED AND COGNITIVE VARIABLES AND DISABILITY IN WOMEN WITH FIBROMYALGIA AND OCCUPATIONAL OVERUSE SYNDROME

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Abstract

There is an expanding body of evidence indicating that cognitive factors contribute to the prediction of disability resulting from chronic pain. Indeed, some research amongst chronic low back pain sufferers has demonstrated that pain-related fear is actually more disabling than pain itself (Crombez, Vlaeyen, Heuts, & Lysens, 1999). The present study utilised a sample comprising individuals with fibromyalgia syndrome (FMS) and occupational overuse syndrome (OOS), and an asymptomatic comparison group. There were three broad aims: The first aim was to evaluate between group differences in pain-related and cognitive variables; and the second, was to assess the relevance of the fear component of the model proposed by Vlaeyen, Kole-Snijders, Boeren, and van Eek (1995) for this sample. The third aim, related to the second, was to explore the relationship between pain and disability, considering the effects of fear of movement, catastrophising, and vigilance for pain. Symptomatic individuals (n = 68) completed measures of pain, fear of movement, vigilance for pain, disability, catastrophising, and cognitive errors. The comparison group (n = 24) completed only the last two measures. Medical assessment, including tender point examination, was also carried out on all participants. There were no differences between individuals with OOS and FMS in relation to current pain, catastrophising, vigilance, or fear of movement. However, significant differences were revealed between participants with FMS and the comparison group in terms of somatic cognitive errors, but not general cognitive errors. Multiple regression using data from symptomatic participants demonstrated that although pain had the largest impact on disability, fear of movement and vigilance also contributed uniquely to this outcome. However, further analysis revealed that catastrophising moderated the relationship between pain and disability. Catastrophising did not impact on disability reporting amongst women experiencing high levels of pain, but amongst women with low levels of pain high catastrophising was associated with more disability reporting than low catastrophising. Furthermore, the association between pain and catastrophising rendered the contribution of fear of movement to disability non significant. Theoretical and methodological implications of these results are discussed.

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Chapter 1

Introduction

"Pleasure is nothing else but the intermission of pain" John Seldon (1584 - 1654)

1.1 Overview

Most human beings experience a number of pains during their lifetime, such as toothache, headaches, abdominal pain, and labour pains. Some pains are minor or transitory, and the origin and meaning of the pain readily apparent. Other pains persist and become chronic, and the suffering engendered may be compounded by diagnostic uncertainty.

This thesis is concerned with chronic pain, which lasts longer than three to six months, or persists after healing has taken place (Carette, 1996; Osterweis, Kleinman, & Mechanic, 1987). Chapter 1 provides a general overview of chronic pain, before concentrating on specific chronic pain syndromes. The aim is to define pain and to review some relatively recent developments in our understanding of the pathophysiology of the experience. This outline will provide context for exploration of psychological variables and chronic pain in Chapter 2.

The second chapter reviews the theoretical basis and current status of the literature on the cognitive variables which together with pain and disability, are the focus of this thesis: Fear of movement/reinjury, cognitive distortion, catastrophising, and vigilance for pain. Chapter 3 delineates two chronic pain syndromes, fibromyalgia syndrome (FMS) and occupational overuse syndrome (OOS). These disorders are often described as discreet entities, but appear to have substantial areas of overlap (Carette, 1996). FMS and OOS merit greater consideration. For example, it is not clear whether conclusions from existing research evaluating cognitive variables can be generalised to these syndromes. At the commencement of this thesis it was not apparent that there was published data on the relevance of fear of movement/reinjury for sufferers with FMS and OOS. Chapter 4 provides a detailed description of the aims and justifications of the present research.

1.2 Definitions of Pain

The International Association for the Study of Pain (IASP) defines pain as "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey & Bogduk, 1994, p. 210). An appended note highlights that pain is a subjective experience, and that the definition avoids associating pain with a stimulus.

Alternative definitions vary in their emphasis on emotional and cognitive aspects of the pain experience. For example, Price (1999) proposes that pain is :

> a somatic perception containing (1) a bodily sensation with qualities like those reported during tissue-damaging stimulation, (2) an experienced threat associated with this sensation, and (3) a feeling of unpleasantness or other negative emotion based on this experienced threat. (p. 1).

The second definition is similar to the one published by the IASP. A key difference is the focus on the "threat" value of pain and associated negative affect. It is clear from both these definitions that pain and nociception are not synonyms. Nociception involves the transmission of signals related to noxious stimuli to the central nervous system (CNS). The work of Beecher with wounded soldiers, described by Melzack and Wall (1965), provided evidence that nociception need not necessarily be accompanied by perception of pain. However, it is important to note also that some of the central nervous system changes which will be outlined in this chapter still occur without the conscious experience of pain (J.P. Chambers, personal communication, November 19, 2000).

1.3 Why do we Feel Pain? Adaptive Value Versus Costs

Pain perception has an adaptive value. It serves to warn the organism and prevent further injury. Additionally, inflammatory changes after injury may encourage rest so that healing can occur (Melzack & Wall, 1988). In comparison, chronic pain has no apparent evolutionary advantage. Phantom pain after amputation of a limb seems only to compound the difficulties already facing the amputee, with the unpleasant experience of pain. Relentless pain is probably amongst the most distressing of human experiences (Tyrer, 1992).

In addition to the individual morbidity associated with chronic pain, there are also substantial costs to society in the form of earnings related compensation, and expenses associated with medical treatment. These costs are considerable as there are many potential

claimants. According to Wall (1999), 60% of the population have taken more than one week off work with back pain at some time during their life. The prevalence of chronic pain is reflected in the New Zealand context by ongoing costs faced by The Accident Rehabilitation and Compensation Insurance Corporation (ACC) for chronic pain and musculoskeletal pain in the year 1999. These were in excess of 1.5 million dollars for entitlement claims (D. Ohlsson, personal communication December 1, 2000).

Now that pain has been defined and the attendant personal and societal costs considered, the rest of this introduction will focus on the evolving understanding of pain perception.

1.4 Pathophysiology of Pain

Descartes (1664) used a bell ringing analogy to explain a pain pathway. This analogy assumed a direct connection between the receptor in the skin and the brain (see Figure 1). Additionally, a linear relationship was implied between the stimulus intensity and pain perception. Notions of nerve cell specialisation and a direct relationship between stimulus intensity and the experience of pain formed the basis of specificity theory. Pattern theory provided an alternative explanation of pain, with the patterning of nerve cell inputs and central summation as the central components. Melzack and Wall (1965) reviewed the central tenets and the evidence to support these theories. They proposed that neither theory alone provided an adequate explanation of pain, but they integrated aspects of specialisation with input control and central summation in an alternative model known as gate control theory.

1.4.1 The gate control theory of pain

The gate control theory of pain has been modified since publication, but the basic principles have been shown to be remarkably robust. In the original statement of the theory Melzack and Wall (1965) proposed firstly that the substantia gelatinosa in the dorsal horn serves as a gate, controlling and modifying afferent signals before the first central transmission cells are reached. Inhibition in the substantia gelatinosa was thought to be increased by large-diameter fibre activity and decreased by small-diameter fibre activity. Secondly, Melzack and Wall indicated that summation of inputs into the dorsal horn serve to trigger central control, activating processes which modify gating. Finally, they outlined the role of input into transmission neurones in the activation of processes involved with perception of, and response to pain. The output of the transmission cells was described not merely as a function of the number of inputs, but dependent on the number, frequency, and specialised function of the cells involved. Melzack and Wall also suggested that there

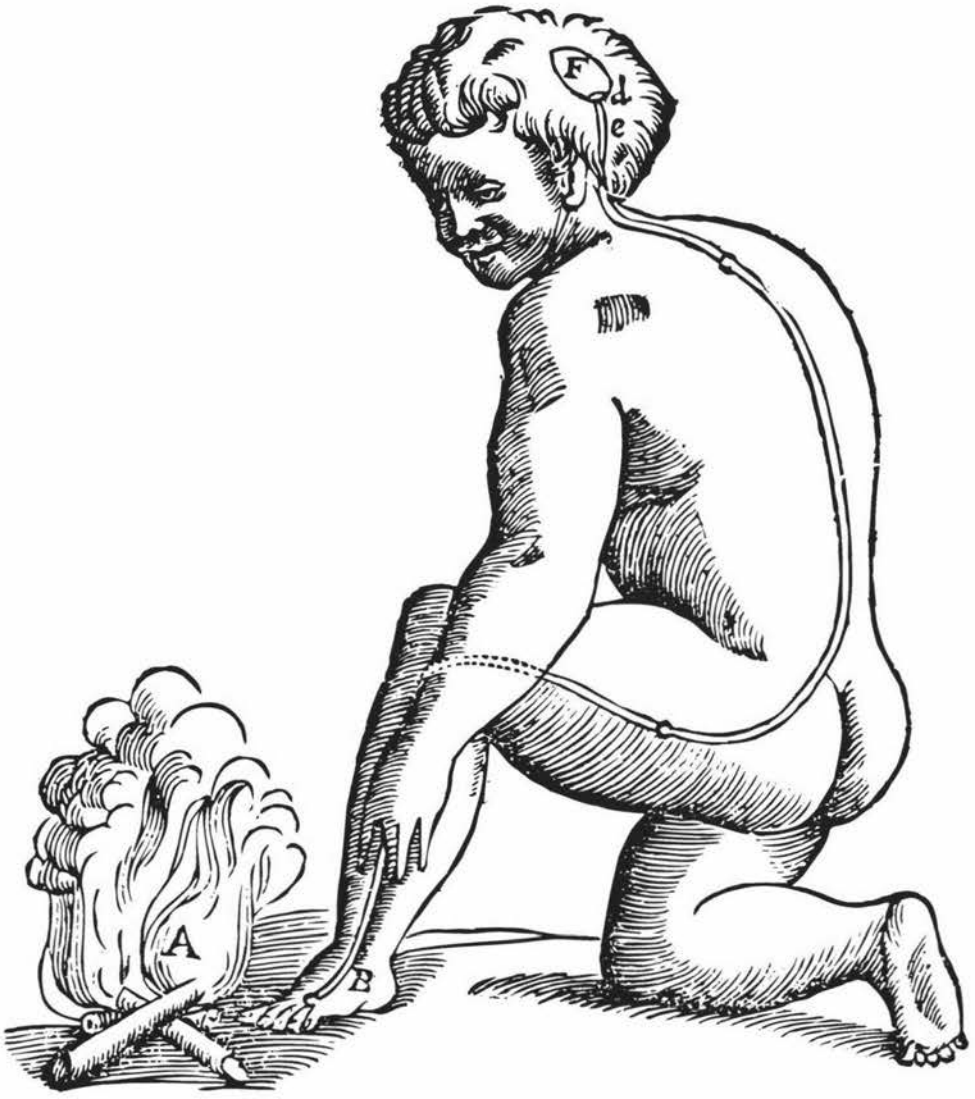

Figure 1. An early drawing of a pain pathway taken from Descartes (1664/1972) "Traite de l'homme".

would be a critical mass of inputs involving integration before the transmission cells would fire.

1.4.2 Involvement of higher centres

Gate control theory included the first clear statement of the idea that there was a specific system for pain modulation, although evidence for descending control of nociception was sparse at that time (Fields & Basbaum, 1994). It is now clear that pain modulation involves amplification and reduction of signals within the CNS and the mechanisms involved at the cellular level have been largely clarified. There are a number of reviews of this research available (e.g., Fields & Basbaum, 1994). However, more work is needed to

explain some of the phenomena in relation to the organism as a whole. In the original statement of the theory Melzack and Wall (1965) proposed that memory, experience, attention, and emotion were part of the gate control system, but the precise role of these variables was not fully elaborated. Research into the mechanisms of pain modulation continues and includes exploration of the nociceptive flexion reflex, placebo analgesia, and hypnotic analgesia. Two experimental research papers involving the nociceptive flexion reflex and placebo analgesia will be briefly summarised to highlight the role of higher centres, and specifically cognitive variables, in pain modulation.

Willer, Boureau, and Albe-Fessard (1979) investigated pain sensation and the nociceptive flexion reflex of the biceps femoris muscle with human participants in four different conditions. These were a mental task, stress, noxious stimulation of the contralateral ulna nerve, and noxious stimulation of the sural nerve. (The latter elicits the flexion reflex). Willer et al. (1979) reported some parallel findings; pain sensation and the nociceptive reflex were both reduced by the attentional demands of a mental task, but increased by expectation of pain. However, the remainder of the results showed a dissociation between pain modulation and the nociceptive reflex (Willer et al., 1979). Ulnar nerve stimulation decreased the sensation of pain from the sural nerve, without altering the flexion reflex. A dissociation was also present between the fading of pain and the nociceptive reflex from sural nerve stimulation. Willer et al. hypothesised, in relation to the first of the non parallel findings, that information from the ulnar nerve activates descending control of the sural inputs without altering the reflex. The differences in fading may also indicate alternative descending pathways to the dorsal horn. In the context of the requirements of the organism the withdrawal reflex has a useful function, but once the alarm has been raised it is doubtful that increments in stimulus intensity are beneficial (Willer et al., 1979).

In an intricately designed study, Amanzio and Benedetti (1999) evaluated expectation versus conditioning in relation to placebo analgesia. They found that endogenous opioid systems were implicated in placebo responses induced by expectation, while placebo responses elicited by conditioning were apparently mediated by other subsystems. The placebo response resulting from expectation was reversible by naloxone (an opioid antagonist) indicating activation of endogenous opioids in the process. It was notable that conditioning with ketorolac (a non steroidal anti-inflammatory) produced a placebo response that was naloxone insensitive, while ketorolac conditioning and expectation elicited a placebo response which was partially naloxone reversible.

Amanzio and Benedetti (1999) suggested that expectation and conditioning are both involved in placebo analgesia and that a balance between them is important in the activation

of opioid or other subsystems. They concluded that expectation interacts with neurochemical systems to produce analgesia, that past experience is relevant to placebo responding, and finally, that improved knowledge of mechanisms linking mental processes and pain will help plan appropriate therapeutic interventions.

These findings support the involvement of psychological factors in pain modulation. It is possible that psychological variables may also be implicated in the alterations in the CNS which accompany chronic pain. These physiological changes will be briefly outlined in the section which follows.

1.4.3 Nervous system plasticity

Gate control theory, by demonstrating the plasticity of the pain transmission system, also laid the foundation for explanation of the persistence of pain following healing. It is now established that the nervous system is not fixed or 'hard wired', but able to alter in response to experience, development, or injury (McQuay & Dickenson, 1990). The term plasticity is used in this context to denote a change in the structure, or the function of nervous system pathways, or both.

Alteration in sensitivity within the nervous system is a key component of either inflammatory or neuropathic pain. The latter features the same pattern of signals as nociceptive pain, but in this instance they arise from nerve damage (Merskey & Bogduk, 1994). A change in sensitivity is apparent when there is either an increase or a reduction in the intensity of the stimulus necessary to produce pain. A lowering of the stimulus intensity needed to produce pain means that pain occurs in response to what would normally be a non-painful stimulus (allodynia; Woolf & Chong, 1993). An augmented response to a normally painful stimulus (hyperalgesia) also can occur (Merskey & Bogduk, 1994). Additionally, sensitivity can increase in tissues remote from the original injury and this is termed secondary hyperalgesia (Woolf & Chong, 1993).

1.4.4 Central sensitisation

While the alterations described above may be apparent in the periphery, research has provided evidence that they reflect CNS change. Central sensitisation refers to a situation of increased excitability of neurones in the dorsal horn, featuring a decrease in response threshold, increased responding to suprathreshold stimuli, and expansion of receptive fields (Coderre, Katz, Vaccarino, & Melzack, 1993; Li, Simone, & Larson, 1999). "Wind up" is a related phenomenon which results from increasing neuronal activity with repeated C

fibre stimulation. It has been demonstrated in various contexts using animal models (e.g., Dickenson & Sullivan, 1987; Li et al., 1999). Li et al. (1999) induced wind up using electrical stimulation, to see whether features of central sensitisation could be produced. Li et al. concluded that wind up and central sensitisation appear to share some common mechanisms, in particular the increase in receptive fields believed to contribute to hyeralgesia, although not all aspects of central sensitisation were produced in the experiment. Early work on the phenomenon of central sensitisation provided a rationale for the use of pre-emptive analgesia before surgery to prevent the barrage of inputs hypothesised to be responsible for central change. McQuay, Carroll, and Moore (1988) demonstrated that participants receiving premedication with an opioid, or a local anaesthetic block, or both, required less analgesia post-operatively. Similarly, Bach, Noreng, and Tjellden (1988) showed that phantom limb pain post amputation was reduced for participants who received an epidural block before surgery, and who were pain free at the time of surgery. This reduction was maintained one year after operation (Bach et al., 1988). However, more research is needed to evaluate the role of pre-emptive analgesia for routine use, and to determine the most appropriate pharmacological interventions (Woolf & Chong, 1993).

1.5 The Multifactorial Nature of Pain

Chronic pain is a complex phenomenon arising from alterations in pain pathways. As outlined by Dray, Urban, and Dickenson (1994) these range from increases in neuronal excitability, to changes in cell phenotype, and to the actual neural structure. The physiological changes do not occur in isolation. Indeed, the pathophysiological processes of chronic pain are interwoven with the thoughts and emotions of the individual feeling pain.

> The psychological evidence strongly supports the view of pain as a perceptual experience whose quality and intensity are influenced by the unique past history of the individual, by the meaning he gives to the pain-producing situation and by his 'state of mind' at the moment. We believe that all these factors play a role in determining the actual patterns of nerve impulses that ascend from the body to the brain and travel within the brain itself. In this way pain becomes a function of the whole individual, including his present thoughts and fears as well as his hopes for the future. (Melzack & Wall, 1988, p. 32).

Ideas about psychological components of pain perception will be pursued in more detail in the next chapter. The brief overview of a large and complex area of research in this introduction has been attempted in order to provide context for the literature review outlining the role of cognitions in chronic pain in Chapter 2.

Chapter 2

Cognitions and Chronic Pain

2.1 Cognitions, Coping, and Disability : An Overview

One of the important developments attributable to the gate control theory summarised in Chapter 1 has been the recognition of the complex nature of pain perception. Wall (1999) commented on the futility of attempts to segregate sensation and perception, as sensory and cognitive factors interact contributing to the overall experience of pain. Gate control theory legitimises perspectives such as the biopsychosocial model outlined by Keefe and France (1999), which emphasises the dynamic nature of processes involved in pain perception, and the reciprocal influences of biological, psychological, and social mechanisms. Whilst all these factors are acknowledged as playing a role, the focus of this thesis is on particular cognitive variables and pain, and how these variables impact on disability.

A number of authors have commented on the lack of a linear relationship between pain and disability, or between impairments, pain, and disability, (Vlaeyen, Kole-Snijders, Boeren, & van Eek 1995; Vlaeyen, Kole-Snijders, Rotteveel, Ruesink & Heuts, 1995; Waddell, Newton, Henderson, Somerville, & Main; 1993). Many studies, including those listed, have shown that cognitive variables, such as pain related beliefs, coping strategies, and ways of thinking are vital in the process of adjustment in chronic pain (Flor & Turk, 1988; Jensen & Karoly, 1992; Jensen, Turner, Romano, & Lawler, 1994; Turner, Jensen, & Romano, 2000). Thus, as Moss-Morris and Petrie (1997) indicated, there is increasing evidence that when there is a mismatch between symptomatic presentation and the extent of disability, examination of cognitions may improve our understanding of both chronic pain and other medical conditions. Indeed, some researchers have demonstrated that pain beliefs are more important than pain itself in prediction of disability (Vlaeyen, Kole-Snijders, Boeren, et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). The next section will briefly define coping in order to contextualise the subsequent literature review of beliefs, cognitions, and chronic pain. The study variables fear of movement, vigilance and awareness (or attention to pain), cognitive errors, and catastrophising are all discussed in this chapter together with sufficient background information to provide a

sense of how these variables fit into some of the existing cognitive models.

2.2 Coping

The construct of coping is frequently referred to in the literature on cognitions and chronic pain. Therefore, brief consideration will be given to how coping has been described and categorised. Lazarus (1993) defined the process of coping as ".. ongoing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person" and adds that the attempt is unrelated to outcome (p. 237).

According to Lazarus and Folkman (1991) coping was traditionally considered as a trait or style implying relative stability. However, here is now recognition that while some aspects of coping may be stable, there are also alterations in the processes of coping in response to the changing demands of a situation. Folkman and Lazarus (1991) described a number of ways of coping, but suggested that most could probably be subsumed under the broad categories of emotion-focused or problem-focused coping. Lazarus (1993) designated problem-focused coping as an active approach involving evaluation of the problem and an attempt to improve the situation. In comparison, he indicated that emotion-focused coping involves reappraisal and management of troublesome emotions, without necessarily altering the situation. In the chronic pain literature, coping approaches tend to be dichotomised as active/passive or avoidant/confrontational. Examples of active coping with chronic pain provided by Waddell (1998) included exercise and "ignoring" pain, whilst withdrawal, rest, and use of analgesia are amongst passive strategies (p. 197). Some of the issues raised by the different emphases used in cataloguing coping will be discussed in subsequent sections of this thesis.

Lazarus (1993) proposed that the meaning of a threat in a stressful situation is important in relation to coping, although he acknowledged that meanings may change over time, or between situations. In terms of coping with chronic pain, the extent to which pain is seen as threatening will depend on beliefs, expectations, and the meaning ascribed to pain. As Price (1999) discussed, individuals are likely to make different appraisals of the meaning of pain. One person may label abdominal pain as "indigestion", while another has concerns about cancer. In the first example pain may be evaluated as non-threatening, while in the second pain is appraised as a danger to health and wellbeing, (Melzack & Wall, 1988; Price, 1999). Sometimes a person experiencing pain may not be concerned about a threat to survival, but may fear that pain is indicative of damage to body tissues, and that movement and activity will exacerbate the damage. Consequently, particular activities

may be avoided. Avoidance of activities perceived to initiate or worsen pain may be seen as an appropriate problem solving strategy by the individual. However, it is now recognised that particular beliefs and behaviours can impact on rehabilitation. Vlaeyen, Kole-Snijders, Boeren, et al. (1995) proposed that pain related fear, specifically fear of movement/reinjury, precipitates avoidance and has important consequences in terms of chronic pain behaviours and outcomes. Some aspects of the theory they outline underpin a major part of this thesis. Consequently, development of their model, which includes pain related fear and avoidance, is considered in some detail. Firstly, a historical overview of the fear avoidance literature is provided, followed by the empirical support for particular aspects of the theories. Subsequently, other factors which might impact on the salience of pain related cues are considered and the relevant literature reviewed.

2.3 Pain and Avoidance

When an acute injury occurs, the presence of pain usually encourages the injured person to rest and to avoid activity. In the short term, avoidance behaviour associated with the experience of pain is considered to be adaptive, as it facilitates healing (Asmundson et al., 1999; Melzack & Wall, 1988; Wall, 1999). However, as Fordyce (1976) noted, pain behaviours, such as avoidance, may be subject to contingencies of reinforcement. For example, the presence of pain may lead the sufferer to engage in limping to minimise the pain, or, alternatively, particular activities such as lifting may be avoided. If these strategies prevent the aversive consequences of pain they are more likely to occur (Fordyce, 1976). Essentially, avoidance prevents confrontation of pain and alternative learning about the experience, which may have facilitated adaptation.

Unfortunately, if inactivity is prolonged, when reactivation is attempted pain may arise from disuse rather than the original injury. Regardless of the origin or cause, the presence of pain may discourage a person from resuming normal movement. Fordyce (1986) suggested that this reluctance may arise from a confusion between hurt and harm. The pain sufferer assumes because pain is increased that damage is occurring. These beliefs are then incorporated into the individuals perspective of pain. The later work of Fordyce clearly acknowledges that a formulation of pain as a stimulus producing a response is inadequate, and that learning and memory are implicated in the information processing involved in pain perception. Additionally, when pain persists concerns for the future will emerge; "Will I get better? Will it go away? Will I be able again to engage in my favoured activities?" (Fordyce, 1986, p. 50).

2.4 The Role of Fear and Avoidance

Lethem, Slade, Troup, and Bentley (1983) outlined a model of "exaggerated pain perception" defined as "Pain experience and/or pain behaviour (and/or physiological responses to pain stimulation) which are (is) out of all proportion to demonstrable organic pathology or current levels of nociceptive stimulation" (p. 402).

This early model incorporated fear of pain and avoidant or confronting responding. It attempted to explain how pain experience and behaviour become separated from pain sensation in "exaggerated pain perception". Based on the authors' experience with back pain sufferers they described four possible fear avoidance outcomes (Lethem et al., 1983). Two outcomes have synchrony of the sensory and emotional components of pain. Two have dysynchrony, or exaggerated pain perception. For instance, organic and sensory aspects of pain perception improve, while the emotional component worsens. Exaggerated pain perception is merged with the fear avoidance aspects of the model in the proposition that the coping responses of individuals lead to approach and confrontation, or to avoidance (although combinations are recognised). Approach over time reduces fear, whereas avoidance maintains fear which can progress to "a full-blown phobic state" (p. 404). Confrontation is seen as adaptive, while avoidance is maladaptive with negative physical and psychological sequelae. The psychosocial context of pain is also acknowledged as important. This includes life events, personal pain history, personal coping strategies, and personality (Lethem et al., 1983).

2. 5 Is Fear a Prerequisite for Avoidance?

In her model, Philips (1987) focused primarily on avoidance, which she indicated was the key component in pain behaviour. Rather than explaining avoidance as a reaction to pain, Philips proposed that avoidance actively reduces any sense of control over pain and contributes to the expectation that exposure will worsen pain. The research of Philips and Jahanshahi (1985b) indicated that avoidance increased with the chronicity of the pain problem. Philips (1987) explained these findings as indicative of dysynchrony between pain intensity and pain behaviour or avoidance. The model outlined by Philips recognised that pain and the environmental rewards accruing from avoidance will impact on avoidance, while there is likely to be reciprocal influence between withdrawal/avoidance and cognitions. Memories of previous exposure to pain, the expectation of increased pain following exposure, together with self-efficacy beliefs about the ability to control pain are all included under the broad heading of cognitions which are central to the explanation of avoidance. It is noteworthy that fear is not explicitly incorporated into the model. Indeed,

Philips and Jahanshahi (1985a) propose that avoidance is not mediated by fear of pain, contrary to the model of Lethem et al. (1983) in which fear is implied as a central component.

2.6 Current Status of the Fear Avoidance Model

Theories of fear avoidance have continued to evolve as researchers strive to elaborate key components of the constructs involved. Kori, Miller, and Todd (1990, cited in Vlaeyen, Kole-Snijders, Boeren, et al., 1995) introduced the notion of fear of movement or kinesiophobia. Vlaeyen, Kole-Snijders, Boeren, et al. (1995) have incorporated kinesiophobia into a model (see Figure 2) which includes the avoidant versus confrontational categorisation of Lethem et al. (1983). Vlaeyen, Kole-Snijders, Boeren, et al. (1995) suggested that injury and subsequent painful experiences will either lead to catastrophising or to adaptive cognitions. Catastrophising elicits fear of movement, which in turn leads to avoidance, disability, disuse, and depression. In contrast, adaptive cognitions prompt confrontation which facilitates recovery.

All of the theoretical models outlined so far share common ground, although there are also points of divergence. All the theories deal with avoidance, but only Lethem et al. (1983) and Vlaeyen, Kole-Snijders, Boeren, et al. (1995) emphasise fear of pain and cat-

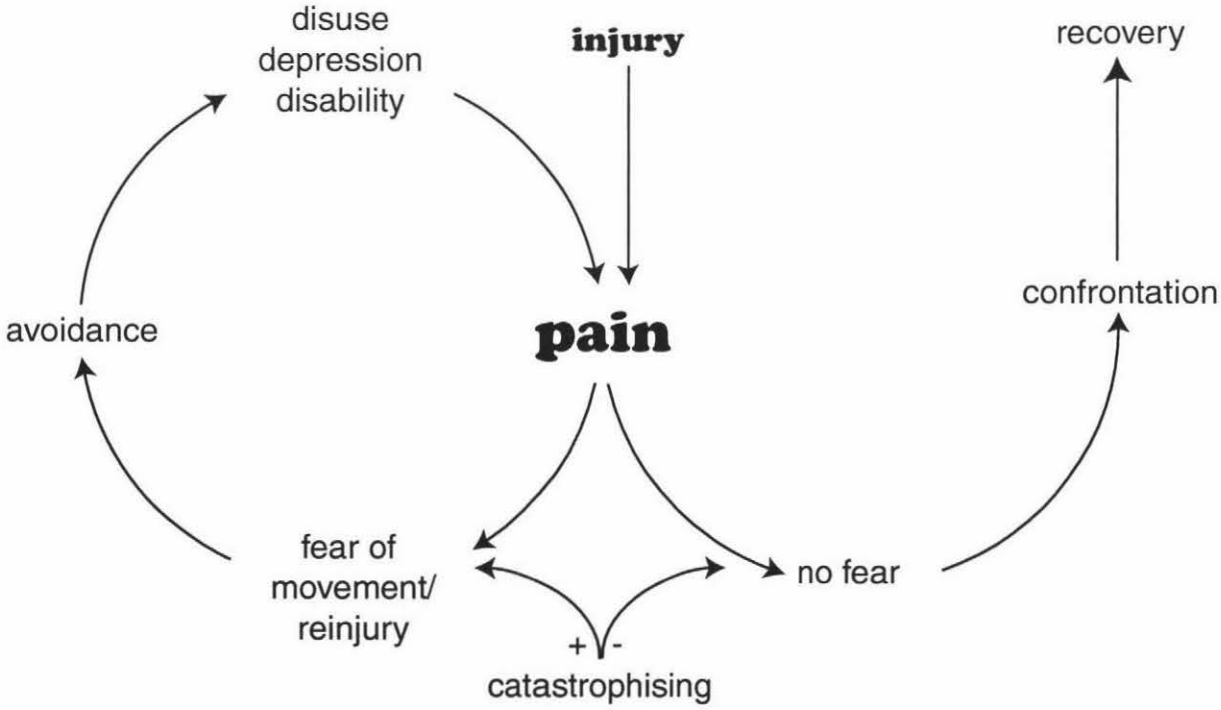

Figure 2 Fear of movement/reinjury model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995).

egorise chronic pain participants as "avoiders" or "confronters". Cognitive variables are not specifically included in the model of Lethem at al. (1983). Thus, Philips (1987) makes a useful contribution in terms of greater cognitive specificity, explicitly stating that expectations, self-efficacy beliefs, and previous pain exposures impact on avoidance. Vlaeyen, Kole-Snijders, Boeren, et al. (1995) develop the theory further by considering both the content of thought (catastrophising), the nature of fear, and the possible outcomes. The early model has recently been refined further as outlined in a review by Vlaeyen and Linton (2000), although this has not been empirically tested. In the revised model pain catastrophising leads to pain related fear, hypervigilance, avoidance, and disability. In the alternative pathway pain in the absence of fear leads to confrontation and recovery. Additionally, Vlaeyen and Linton (2000) hypothesised that "negative affectivity" and "threatening illness information" may play a role in catastrophising.

2. 7 Empirical Support for Fear/Avoidance Models of Pain

Slade, Troup, Lethem, and Bentley (1983) were amongst the first to evaluate fear avoidance ideas. They used a non-clinical sample to assess the relevance of coping strategies and pain history to the fear avoidance model, and found a significant association between pain severity and avoidance. Participants with a history of back pain, on average, provided higher ratings of external pain. When recent episodes of back pain were considered, participants who had experienced severe pain were more likely than those experiencing less severe pain to use passive/avoidant coping strategies. The results indicated that back pain severity and a recent experience of more severe pain were associated with avoidance and passive coping. However, it is important to consider the operationalisation of the construct of coping in this study. Participants were asked about preferred strategies for dealing with pains other than back pain. Endorsement of items such as "ignored it and carried on" or "took physical exercise" was categorised as active coping, while endorsement of "took analgesics" and "rested" was categorised as passive coping (p.411). Keefe, Salley, and Lefebvre (1992) questioned the labelling of taking medication as a passive strategy, pointing out that active compliance is required. In essence, the study of Slade et al. (1983) provided some preliminary evidence for a link between pain and avoidance but they did not elaborate upon the fear component.

The validity of the model of Lethem et al. (1983) was also tested by Rose, Klenerman, Atchison, and Slade (1992) in a study involving three chronic pain conditions, post-herpetic neuralgia, reflex sympathetic dystrophy, and low back pain, with a pain-free comparison group. Rose et al. (1992) stated that their findings supported the study hypothesis

of the fear avoidance model as a "unified psychological theory", accounting for the development of a range of chronic pain problems (p. 364). Significant differences between chronic and recovered participants were reported in relation to pain history, personality and life events, but not coping. The data obtained may not justify their wide ranging conclusion. There appears to be a lack of clarity in relation to interpretation of coping strategies. The pain coping strategies section included the same item content used by Slade et al. (1983), but exercising and ignoring pain were noted as being passive strategies, while resting or taking analgesics were reported as active strategies. This was contrary to the explanation of Slade et al. (1983) and raises questions about comparisons of results, as coping strategies appear to be used to quantify the presence of either avoidance or confrontation.

Concerns about the scope of the conclusions are also relevant to the study of Klenerman et al. (1995) which addressed the important issue of predicting chronicity in patients with acute back pain. These authors concluded that "fear avoidance variables" (p. 478), from the model of Lethem et al. (1983), were most useful in predicting outcome. However, stressful life events, bodily sensations, previously experienced pain, and a choice of five coping strategies in response to pain were the variables actually investigated. Taking pain killers and resting were construed as passive, going to the doctor was neutral, and exercising or ignoring pain, and carrying on were active. Active coping strategies were presumably construed as confronting, rather than avoidant, but it is not clear how fear variables were operationalised in this study.

2. 8 Fear Avoidance Beliefs

There have been a number of other projects using a range of different assessment instruments evaluating fear and avoidance in chronic pain participants. The findings of Waddell et al. (1993), using the Fear-Avoidance Beliefs Questionnaire (FABQ) with a back pain sample, provided support for a model linking fear and avoidance with disability. Fear avoidance beliefs contributed to disability (daily function and time off work), independently of pain severity. Furthermore, diagnostic uncertainty, rather than "pathological severity", was associated with an increase in fear avoidance beliefs (Waddell et al., 1993, p. 164).

Jensen et al. (1994) utilised a different measure, the Survey of Pain Attitudes (SOPA), in a mixed pain population. Amongst the results reported were those indicating that pain beliefs, including the ideas that one is disabled and that pain equates with harm, predict physical dysfunction. The notion of pain equalling harm was most prevalent in partici-

pants who had experienced pain for less than 2.4 years. Additionally, the association between pain intensity, disability, and adjustment was most pronounced in participants reporting pain of low/medium intensity. This replicated the work of Jensen and Karoly (1992), who found that the relationship between disability, beliefs, and activity level was most pronounced for those with low/medium levels of pain. In comparison, Jenson, Romano, Turner, Good, and Wald (1999) report an association between the belief of pain equalling damage, pain behaviour, and functioning in those with higher pain levels. Therefore, there are some contradictions in the literature about the relationships between beliefs, function, and pain intensity and duration, although there is support for the link between the belief that pain equals harm, and disability. There is also lack of clarity about the role of emotions. Jensen and Karoly (1992) did not report any links between the emotion scale and depression. However, Jensen et al. (1994) noted that the tendencies to believe that emotions influence pain and that one is disabled were both related to increased psychosocial dysfunction. More recently, Turner et al. (2000) investigated the effects of beliefs, coping, and catastrophising in a chronic pain sample. The independent contributions were as follows : Beliefs and coping significantly contributed to disability, while beliefs and catastrophising were significant predictors of depression (Turner et al., 2000).

The model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) linking fear of movement with depression, disuse, and disability has been thoroughly evaluated amongst low back pain populations and will constitute the major focus of the next section of this thesis as particular aspects of the theory of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) form the basis of a number of the present study hypotheses.

2. 9 Fear of Movement/Reinjury

Vlaeyen and colleagues have reported three lines of research enquiry in a number of studies. Firstly, they evaluated fear of movement/reinjury and related variables, and examined the usefulness of the fear of reinjury construct (Crombez, Vlaeyen, Heuts, & Lysens, 1999; Vlaeyen, Kole-Snijders, Boeren, et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). Secondly, they conducted a number of small studies investigating the impact of fear of movement/reinjury and avoidance on the performance of a behavioural task (Crombez, Vervaet, Lysens, Baeyens, & Eelen, 1998; Crombez, Vlaeyen, et al., 1999; Vlaeyen, Kole-Snijders, Boeren, et al., 1995). Finally, they reported an initial evaluation using a single case design of exposure in chronic back pain sufferers, which provides indirect support for the model and preliminary evidence of treatment efficacy (Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, in press).

2.9.1 Fear of movement/reinjury and prediction of disability

Vlaeyen, Kole-Snijders, Boeren, et al. (1995) investigated the relationship between fear of movement/(re)injury and the following three sets of variables in a sample with low back pain : Firstly, age, gender, duration of pain, use of support equipment, and compensation status; secondly, pain intensity, pain coping and pain cognitions, and finally, fear and depression. Fear of movement was shown to be associated with compensation status and gender. Pain related fear was also found to be more closely related to catastrophising and depression, than to pain coping and pain intensity. In a regression analysis, compensation status, but not gender or pain intensity, predicted fear of movement. However, after controlling for the effects of gender, pain intensity, and compensation status, catastrophising and then depression were most predictive of pain related fear.

Vlaeyen, Kole-Snijders, Rotteveel, et al. (1995) evaluated the role of fear of movement in the prediction of disability after controlling for current pain and medical status of the participants. In contrast to the research of Vlaeyen, Kole-Snijders, Boeren, et al. (1995), fear of movement was unrelated to compensation status. It was noteworthy that catastrophising which predicted fear of movement was not predictive of disability. Fear of movement explained most of the variance in disability. This finding was replicated by Crombez, Vlaeyen, et al. (1999) who noted that disability was significantly associated with all measures of pain related fear, but not with pain intensity or with negative affect. Additionally, it was reported that fear of movement was greater amongst participants with pain of sudden onset than those with a gradual onset of pain, but the relationship between fear of pain and pain duration is not clear. Vlaeyen, Kole-Snijders, Rotteveel, et al. (1995) noted a negative association between fear of pain and pain duration, indicating perhaps that fear reduces over time. However, only one of the two studies reported by Vlaeyen, Kole-Snijders, Boeren, et al. (1995) found a relationship between fear of movement and duration of pain and this was a positive correlation, greater fear of pain being linked to a greater chronicity.

2.9.2 Fear of movement/reinjury and behavioural tests

In their report about the relationship between fear/avoidance and behavioural performance, Vlaeyen, Kole-Snijders, Boeren, et al. (1995) presented data on chronic low back pain (CLBP) participants carrying out a single motor movement. Participants disclosing greater fear of movement demonstrated greater behavioural avoidance, but not significantly increased physiological reactivity, when asked to lift a weight. However, it was not clear whether avoidance was a function of increased pain, as Crombez, Vervaet, et al.

1998 pointed out, no pain rating was provided.

Subsequently, Crombez, Vervaet, et al. (1998) investigated differences in behavioural test performance between chronic pain patients, categorised on the basis of questionnaire data as either avoidant or confrontational, in relation to their pain. Avoidant participants were more afraid of pain and reinjury and reported more disability than the confronting group. They also manifested greater avoidance during the behavioural task. Additionally, the avoidant group paid more attention to pain and reported experiencing more frequent pain of longer duration, but not greater intensity, than the confronting group. The findings were replicated by Crombez, Vlaeyen, et al. (1999) who reported that scores obtained on the Tampa Scale for Kinesiophobia (TSK), and the physical subscale of the FABQ were better predictors of behavioural performance than pain intensity, pain duration, the experience of increased pain, negative affect, and catastrophising.

2.9.3 Exposure treatment - Single case design

Vlaeyen et al. (in press) looked at exposure in the context of treatment for a chronic low back pain population using a series of single case cross over designs with exposure and graded activity. The aim was to reduce pain related fear, catastrophising, and disability. Improvements in all these parameters were noted in the exposure condition, and amongst half the cases there was an associated increase in pain control.

In summary, fear of movement/reinjury and subsequent avoidance are linked to disability. Indeed, pain related fear may be more important than pain itself in the prediction of disability. When participants are categorised as avoidant or confronting the groups cannot be differentiated in relation to pain intensity, although avoidant participants may have more frequent pain which has lasted longer than their confronting counterparts. The construct catastrophising, which will be elaborated later in this chapter, seems also to play a role in the process impacting on fear of movement. The next sections review the literature in relation to expectations and attention to pain, factors that might be of relevance for the development of the model.

2.10 Factors Relevant to Fear of Pain : Expectation of Pain

There is evidence in the chronic pain literature that expectation of pain does not necessarily increase the experience of pain (Crombez, Vervaet, Baeyens, Lysens, & Eelen, 1996; Crombez, Vervaet, et al., 1998). In both of these studies the expectation of pain was higher

than experienced pain in the initial trial, but this was corrected subsequently. Furthermore Crombez et al. (1996) found that expected pain was higher than baseline in the initial trials, but the difference was not significant in the second series. There were no significant differences between baseline and experienced pain. Therefore, it does not seem that expectation causes pain.

Notably there were no significant differences between avoiders and confronters in relation to pain expectation (Crombez, Vervaet, et al., 1998). Crombez, Vlaeyen, et al. (1999) also reported no relationships between pain related fear, including fear of movement, and expectation of pain. It does not seem that being either fearful or avoidant increases the anticipation of pain. This contrasts with comments of Philips and Jahanshahi (1985a) who proposed that depression and the anticipation of an increase in pain are both factors in avoidant responding.

The negative finding in relation to the link between fear of pain and expectation of pain is also somewhat surprising. Rachman (1994) commented in relation to the literature on general fears that there is tendency to overestimate the amount of fear that will be experienced, and that some aspects of pain overprediction resemble the data on fear. Essentially there may be similar ways of responding to a range of aversive stimuli. However, Rachman (1994) also indicated that processes involved in prediction are amenable to change. For instance, disconfirmation of estimates of pain produces change in the direction opposite to the original prediction. If an individual overpredicts pain and their ideas are disconfirmed on initial exposure, prediction will be modified prior to the next exposure (Rachman, 1994). This is in accord with the results that expected pain was significantly greater than that experienced for an initial testing series, but not subsequently (Crombez et al., 1996; Crombez, Vervaet, et al., 1998).

Similarly, the findings of McCracken, Gross, Sorg and Edmands, (1993) showed that chronic pain patients corrected inaccurate estimations of pain, although overall the participants with low back pain underpredicted pain across trials. Classification of participants as having high or low levels of anxiety revealed that there were differences between-groups in terms of patterns of prediction. Those with high anxiety tended to make more correct predictions, while participants with low anxiety tended to underpredict pain. McCracken et al. (1993) explained that these results reflected the tendency of highly anxious individuals to initially overpredict, but to quickly correct predictions over the trials. In comparison, individuals with low anxiety improved predictive accuracy more slowly. It is noteworthy that individuals in this study who overpredicted pain had more limited movement on a behavioural task used to index avoidance (McCracken et al., 1993). The authors

suggested that both categories of prediction errors may lead to poor adaptation to pain. If injured individuals underpredict pain this may mean they are not adequately attending to the injury, whereas overprediction may precipitate unnecessary avoidance. This does not address the finding of greater predictive accuracy in the avoidant high anxiety group. If it is assumed that fear of pain combined with the anticipation of more pain is the major factor in precipitating avoidance, one would expect that the rapid learning of the high anxiety group would reduce avoidance. It may be that while this happened in the context of the research, in real life if overprediction is associated with avoidance, the individual avoiding will not be exposed to information disconfirming the original pain estimate. Although the literature is not in complete agreement about the relationship between avoidance and overprediction, it does appear that individuals make and correct estimates of pain. It is reasonable to assume that in order to make predictions it is necessary to attend to the stimulus that is being estimated.

2.11 Attention to Pain

Eccleston and Crombez (1999) provided a thorough review of how and why pain demands attention. Clearly in terms of species survival it is appropriate for attention to be diverted so that pain can be appraised and the organism primed for action (Eccleston & Crombez, 1999). However, other variables moderate interruption by pain. Eccleston and Crombez included the following : intensity, novelty, predictability, threat, and emotional arousal. A brief review of some of the relevant literature will follow, as vigilance for, or awareness of pain is another of the present study variables. This review will start with research in non-clinical populations.

Crombez, Eccleston, Baeyens, and Eelen (1998a) used a volunteer sample to investigate whether the threat of high intensity pain in the presence of a low intensity stimulus leads to task disruption. Results for the threat cue condition were compared with those from the control group. It was interesting that while controls and recipients of the threat did not differ in terms of their perception of stimulus intensity, the attention of those in the threat group was diverted from the task at the onset of the threat. However, as Crombez et al. (1998a) explained, the effects of the threat were quite confined, overall task performance did not suffer, and the disruption only occurred at time 1. They suggested that this was because the threat primed the individual, but when the stimulus was appraised as non-threatening attention could be redirected to the task.

Crombez et al. (1998a) also indicated that individual differences in relation to attention might be important. Heynemann, Fremouw, Gano, Kirkland, and Heiden (1990) found in

a non-clinical sample that there were differences between catastrophisers and non-catastrophisers in relation to the use of coping strategies for acute pain. Tolerance for a painful stimulus improved amongst catastrophisers when they used self-instructional training rather than attention diversion, while the opposite was true for noncatastrophisers. Therefore, differences in ways of thinking, particularly those that might impact on threat appraisal, may well be relevant.

Crombez, Eccleston, Baeyens, and Eelen (1998b) explored the idea that a tendency to catastrophise may make some individuals more attentive to threat cues and consequently more prone to exhibit signs of task interference. A primary task paradigm was used (a tone discrimination task) in a non-clinical population and two experiments conducted. Results of the first experiment, which used the threat of an intensely painful stimulus, indicated that a tendency to catastrophise was associated with greater task interference after the initial presentation of the threat cue. While attempting to account for this finding Crombez et al. (1998b) noted that self report data from their study does not support the idea of sensory amplification, as the catastrophising group did not differ from the non-catastrophising group on intensity ratings of either the threat or control stimulus. Additionally, they noted that while the explanation offered by Crombez et al. (1998a) for the temporal relationship between task disruption and threat cue has some appeal, it may not be adequate. Hence, in a second experiment they investigated whether the same pattern of interference resulted from the use of an internal threat cue. In this instance, participants were told that the electrocutaneous stimulus (ES) would directly affect pain fibres. Additionally, participants were asked to rate the unpleasantness, intensity, and perceptual features of the last ES and these data were compared with a pre-experimental rating. The aim in collecting this information was to evaluate the involvement of amplification of somatosensory information. The temporal pattern and between-group differences of experiment one were replicated. Additionally, catastrophisers rated the ES as more intense/unpleasant than non-catastrophisers during the task, but no between-group differences in ratings were apparent prior to the experiment. Crombez et al. (1998b) commented that the results demonstrate that catastrophisers are "hypervigilant for threatening somatic information" (p. 196), although a caution is added that the results may not generalise to a chronic pain population. It is interesting that in the literature this study is cited as providing evidence that the presence of a threat cue elicited greater fear amongst catastrophisers; however, in the original paper while students are asked about tension in relation to the stimulus, it is not apparent that fear of pain has been directly measured.

In similar investigations using chronic pain participants, Crombez, Vervaet, et al. (1998) demonstrated that paying attention to back pain sensations was more common amongst

avoidant participants than non-avoiding participants. The avoidant group was also characterised by greater fear of pain. Additionally, two separate projects revealed how pain intensity and bodily awareness (Eccleston, Crombez, Aldrich, & Stannard, 1997), and pain intensity and pain related fear (Crombez, Eccleston, Baeyens, Van Houdenhove, & Van Den Broeck, 1999) interact to produce attentional disruption in pain sufferers.

In the first of these studies, Eccleston et al. (1997) used a primary task paradigm to evaluate the relationships between pain intensity, somatic awareness, and task disruption. Amongst the participants with high intensity pain, only those who also had high somatic awareness experienced a disruption of attention. Eccleston et al. (1997) suggested that the results are best explained by pain distracting attention, rather than a process of amplification, but added that it was important to try to establish how pain gains entry into awareness.

In the second study, Crombez, Eccleston, et al. (1999) used the number numerosity test to assess whether fear of pain moderates the relationship between pain and attention. No main effect was found for pain severity alone, but the interaction of pain severity and pain related fear predicted task disruption. Similarly, Asmundson, Kuperos, and Norton (1997) reported that differences in predispositions to fear pain may account for variations in attention after pain related cues. Division of participants on the basis of scores on the Anxiety Sensitivity Index (ASI; Peterson and Reiss, 1992), showed high ASI participants had a similar pattern of responding for all stimuli, while low ASI participants diverted attention from pain related cues. This is a somewhat counterintuitive finding and as the authors themselves commented the clinical relevance remains to be established.

Other research has implicated factors such as task difficulty in determining attention disruption. Eccleston (1994) demonstrated that the performance of chronic pain patients was slowed only for complex attentionally demanding tasks when they were also experiencing high levels of pain. In an easier task no significant differences were found between the performance of controls or those with chronic pain. It is also possible, as Pincus, Fraser, and Pearce (1998) suggest, that attentional bias is attributable to mood rather than pain state. However, they produced equivocal results for attentional bias using a stroop paradigm. Further analysis using depression scores as a covariate eliminated the "apparent" difference between pain participants and controls (Pincus et al., 1998, p. 55). Pincus et al. proposed that when viewed in the context of other similar research their findings indicate that mood rather than pain accounts for attentional bias.

In summary, across all the studies it seems that while the threat of pain is sufficient to

divert attention it does not jeopardise task performance. The stimulus is appraised and attention redirected to the task. There are also indications that unless pain levels are very high, and the task is extremely complex, performance may not be adversely affected. Therefore, in particular circumstances it may be possible to divide attention between pain and the task in hand. Several findings are relevant to the present thesis. Pain intensity alone did not produce attentional interference. However, interactions between pain intensity and fear of pain, and pain intensity and somatic awareness, did. Additionally, the evidence that catastrophising impacts on stimulus appraisal and task disruption needs further investigation in clinical populations.

2.12 Negative Thoughts About Pain

Beliefs and expectations about chronic pain have been reviewed previously. The remainder of the chapter will provide a broad outline of the literature on cognitive distortions and catastrophising in relation to chronic pain. These constructs are considered separately, as this reflects different emphases in the literature. They could be subsumed under the more general heading of negative thinking. The ongoing debate about conceptual overlap between depression and catastrophising, and whether catastrophising is really a coping strategy, will also be covered.

2.12.1 Cognitive distortion

Much of the literature on cognitions and chronic pain has been influenced by cognitive theories of depression. Beck's (1979) theory of depression outlines several elements; negative views of the self, the world, and the future; activation of negative schema; and a predisposition to make a range of information processing errors. Personalisation, overgeneralisation, selective abstraction, and catastrophising are all examples of information processing errors (Beck, 1979).

Because the literature on cognitive distortion derives from theoretical approaches to depression much of the early work incorporates assessment of both depression and cognitive distortion in chronic pain. The Cognitive Errors Questionnaire (CEQ ; Lefebvre, 1981) was developed to evaluate whether depressed patients with or without low back pain demonstrated errors in thinking, and whether the distortions of depressed low back pain related only to the pain or were more general. Items pertaining to general life experiences and those relevant to chronic pain were included. The results showed that depressed people had similar levels of general cognitive distortion, whether low back pain was present or not. For both groups cognitive distortion was significantly greater than for controls.

Additionally, on pain-related questionnaire items, depressed participants with back pain made more cognitive errors than depressed participants without low back pain. Lefebvre (1981) concluded that the results support the idea that cognitive distortion is a central component of depression.

Subsequently, a number of studies have investigated cognitive distortion measured by the CEQ and its relationship to concurrent and future depression, and psychological distress in populations with arthritis, rheumatoid arthritis, and low back pain.

Smith, Aberger, Follick, and Ahern (1986) found that cognitive distortion measured by the CEQ in a low back pain population was significantly associated with psychological distress, but not somatisation (measured using the Minnesota Multiphasic Personality Inventory; MMPI; Hathaway & McKinley). The association reported between variables was independent of pain severity.

Smith Follick, Ahern, and Adams (1986) entered cognitive distortion variables into a regression analysis controlling for the effects of depression, pain intensity, and number of treatments. Cognitive distortions associated with low back pain were more closely associated than non-pain distortions with disability. Depression was measured using the depression scale from the MMPI and disability was assessed by the Sickness Impact Profile (SIP; Bergner, Bobbitt, Carter, & Gilson, 1981).

Smith, Peck, Milano, and Ward (1988) investigated cognitive distortion, depression, and disability in a population with rheumatoid arthritis. They found that the relationship between scores on the CEQ and depression were independent of disease severity. Additionally, there was a lesser relationship between physical disability and cognitive distortion. Measures of disability were significantly related to cognitive errors pertaining to arthritis, but not to general cognitive errors.

Much of the chronic pain literature relies on self report measures not diagnostic interviews of depression. Smith, O'Keeffe, and Christensen (1994) addressed this concern by using a standardised interview schedule, The Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) in their study of low back pain participants. The CEQ and Centre for Epidemiological Studies Depression Scale (CESD; Radloff, 1977) were utilised to capture the other variables of interest. Results supported a link between cognitive distortion and depression in both chronic pain and non-pain groups. Depressed participants made more cognitive errors than the non-depressed groups. Depressed chronic pain participants evinced more sign of cognitive distortion in pain situations than general

situations, whereas depressed patients without pain manifested equivalent distortion in both situations.

Extending these findings in relation to depression using a sample with rheumatoid arthritis, Smith, Christensen, Peck, and Ward (1994) carried out a prospective study. With initial levels of depression controlled, levels of depression four years later were significantly related to initial cognitive error scores.

Gil, Williams, Keefe, and Beckham (1990) suggested that existing measures of cognitive distortion such as the CEQ and the catastrophising subscale of the Coping strategies Questionnaires (CSQ; Rosenstiel & Keefe, 1983) may not be adequate to capture the variance in negative thinking. In particular neither measure addresses the thoughts that might occur during a flare up of disease. Gil et al. (1990) evaluated the relationship between negative thoughts during exacerbation of disease, pain, and psychological distress in three pain populations using their own measure the Inventory of Negative Thoughts in Response to Pain (INTRP). Negative thoughts were associated with the experience of pain in all pain populations and further these negative thoughts were linked to a range of symptoms of psychological distress. Additionally, there were between-group differences with the chronic pain group reporting more of all categories of negative thoughts than the other groups, which might be accounted for by the intensity and daily occurrence of pain in this chronic pain group (Gil et al., 1990).

Creation of new questionnaires may not adequately address concerns about reliability and mapping of constructs. Thorough validation of assessment measures and replication of findings are important parts of the process. Rather than generating a new questionnaire, Moss-Morris and Petrie (1997) revised the CEQ, developing the CEQ-R. An important point raised in the preliminary research with the CEQ-R is that the measure may not be confounded with depression. Participants with depression made comparable numbers of somatic cognitive errors, but significantly more general cognitive errors, than other participants. Moss-Morris and Petrie (1997) suggested that the CEQ-R might be useful to distinguish between conditions such as chronic pain and depression which tend to overlap. While this area of overlap was not specifically assessed in the present thesis there was interest in whether somatic, rather than general, cognitive distortions characterised some of the sample in the present study.

Although there is evidence of negative cognitions in chronic pain patients, there are some contradictory findings. Ingram, Atkinson, Slater, Saccuzzo, and Garfin (1990) found that depressed patients with back pain experienced more negative and fewer positive auto-

matic thoughts than either non-depressed chronic pain patients, or controls. However, non-depressed participants with chronic pain had more positive automatic thoughts than controls. Intriguingly, none of the groups differed significantly in relation to attributional style.

Additionally, Boston, Pearce, and Richardson (1990) found during development of a pain cognitions questionnaire that most responses to the question, "What do you find yourself thinking at times when you are in pain?" (p. 104), were positive. This is an interesting finding, and as Boston et al. (1990) discussed, amenable to a range of interpretations. It seems that rather than using negative coping strategies, chronic pain patients actually use positive strategies but these appear to be unsuccessful. Four factor, two negative and two positive, were derived from a principal components analysis of the questionnaire responses (Boston et al., 1990). The authors reported that negative thoughts seemed to be closely associated with mood and pain intensity. The association between positive thoughts, mood, and pain was not clear, although Boston et al. indicated that while active coping was negatively associated with pain and distress, passive coping was associated with worse adjustment. However, it was also noted that cognitions, pain, and anxiety were all related. Consequently, the relationships between the factors and pain may have been attributable to anxiety (Boston et al., 1990). Another explanation provided by Boston et al. was that cognitions and affect are closely linked and further that coping strategies have a closer relationship with mood variables than with pain.

Flor, Behle, and Birbaumer (1993) also reported "adaptive cognitions" in a chronic pain population, but noted that negative beliefs were stronger when comparisons were made with a control group. Furthermore, improvement during treatment was linked to reducing negative, rather than increasing positive, thoughts. This echoes suggestions of other commentators (Boston et al., 1990; Turk & Rudy, 1992) that the most efficacious approach may be to use fewer negative rather than more positive strategies.

Overall, there seem to be associations between symptoms of depression, chronic pain, and cognitive distortion. Those who are depressed and have chronic pain seem in general to have a preponderance of pain related, rather than general, cognitive distortions. Additionally, it appears that individuals with chronic pain experience both negative and positive thoughts about their pain, but that negative thoughts may be more influential in determining outcome.

2.12.2 Catastrophising

Catastrophising has received considerable attention in the chronic pain literature as a subcategory of cognitive distortion. A precise definition of catastrophising is lacking, although Sullivan, Bishop, and Pivik (1995) indicated that there was general agreement that "catastrophising involves an exaggerated negative orientation toward noxious stimuli." (p. 524). Much of the research focusing on catastrophising has utilised the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983). In this context catastrophising is described as "...a method of cognitively coping with pain..." (Keefe, Brown, Wallston, & Caldwell, 1989, p. 51). This notion of catastrophising as cognitive coping has been the subject of some debate, which will be discussed in more detail after a brief review of relevant research.

Keefe et al. (1987) evaluated pain coping strategies amongst participants with osteo-arthritis using the CSQ. Factor analysis of the CSQ produced two factors which were labelled : coping attempts, and pain control and rational thinking. These factors and disability status were shown to be key predictors of pain and psychological distress amongst individuals with osteo-arthritis. Catastrophising loads negatively on to the pain control, health, and rational thinking factor. Lower levels of catastrophising were associated with less pain, better health, and less distress (Keefe et al., 1987).

Keefe et al. (1989) conducted further investigations of catastrophising in a population with rheumatoid arthritis, using only the catastrophising subscale of the CSQ. Participants completed the measure on two occasions six months apart. Initial catastrophising scores were related to subsequent functioning, pain intensity, and depression. Keefe et al. (1989) concluded that catastrophising is a maladaptive strategy and that further research is needed to assess whether modification of the strategy improves outcome.

Catastrophising seems to be a key variable in relation to chronic pain and disability. However, the acceptance of its status as a coping strategy is not universal. The second debate in the literature about catastrophising relates to the extent of its conceptual overlap with depression.

2.12.3 Catastrophising or coping?

The appropriateness of a catastrophising subscale within a measure of coping has been questioned (e.g., Sullivan & D'Eon, 1990; Turner et al., 2000). Several authors have suggested that catastrophising is best conceptualised as a belief, not a coping strategy (Geisser,

Robinson, & Riley, 1999; Jensen, Turner, Romano, & Karoly, 1991). The presence of catastrophising in a measure of coping is proposed as a potential confound, particularly when the subscale score may be merged with others in factor analyses. There is some suggestion that conflicting results may derive from this process (Geisser et al., 1999). For instance, the use of combined scores obscures information about specific aspects of coping (Jensen et al., 1991). Additionally, reliance on factor scores can also make it impossible to isolate the contribution to outcome made by a particular variable (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998). In response to these issues Keefe, Lefebvre, and Smith (1999) reiterated their belief that catastrophising is a coping strategy regardless of any associated negative outcome, on the grounds that strategy and outcome should be evaluated independently. Whether people actively engage in catastrophising, as the phrase "cognitive effort" used by Keefe et al. (1999, p. 177) implied, or whether negative thoughts are automatic remains to be determined. However, Keefe et al. (1999) have made some relevant points about oversimplifying coping by categorising strategies as dichotomous variables such as active/passive. Ideally, approaches to coping need to incorporate the stable individual facets of coping within the changing parameters of the environment in which they occur (Lazarus, 1993).

2.12.4 Confounding catastrophising with depression

Sullivan and D'Eon (1990) posed the question of whether catastrophising is a pain related thought or a symptom of depression. They used the CSQ to explore the relationships between pain depression and catastrophising in a chronic pain population. Pain, catastrophising, and depression were shown to be associated, but overlap of item content for measures of depression and catastrophising were thought to account for this relationship. These findings prompted discussion about the usefulness of the construct and whether catastrophising is a distinct pain related thought, or a feature of disordered mood (Sullivan & D'Eon, 1990). In response to these concerns Haaga (1992) commented on the importance of evaluating psychological variables on a continuum and using multiple measures to help determine whether catastrophising and depression are positively related, or essentially alternate measures of the same construct.

There are additional concerns in relation to the measurement of depression in chronic pain populations. In particular, validity of the diagnosis of depression for chronic pain sufferers may be questioned, as there is some overlap between chronic pain symptoms and somatic symptoms of depression such as decreased energy and disturbed sleep (Romano & Turner, 1985). Williams and Richardson (1993) concluded on the basis of data from a

chronic pain sample that the use of a total BDI score may lead to overestimates of depression.

2.12.5 Empirical support for catastrophising

There is support in the literature for the independence of the catastrophising construct. Sullivan et al. (1995) found that, although there were significant correlations between catastrophising, depression, trait anxiety, negative mood, and fear of pain, only catastrophising contributed unique variance to the prediction of pain. Sullivan et al. (1995) concluded that catastrophising is not confounded with anxiety or mood variables. There is support for the validity of catastrophising as a separate construct from several other sources (Flor et al., 1993; Geisser, Robinson, & Henson, 1994; Sullivan et al., 1998). Furthermore, Sullivan et al. (1998) showed that catastrophising contributes to the prediction of disability independently of the effects of depression and anxiety, after controlling for the effects of pain. In addition to being linked to more intense pain, catastrophising was also associated with reporting of greater occupational disability and unemployment. Analysis of the various components of catastrophising (helplessness, magnification, and rumination) revealed that the latter was most strongly related to disability. According to Sullivan et al. (1998) greater pain related vigilance was linked to experiencing more pain and disability. The literature suggests that fear of pain interacting with pain intensity produces task disruption, and by implication greater attention to pain. Therefore, the correlational relationship outlined by Sullivan et al. (1998) may not be in the direction in which they report it. The model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) assumes that the routes to disability or recovery start with the initial injury and experience of pain. Following on from this catastrophising determines whether particular fears are activated. According to the model, it is then fear which leads to avoidance and disability. Vigilance or a heightened somatic awareness may be an intermediary between catastrophising and disability, as the fearful chronic pain sufferer is watchful for pain related signals. This still presupposes a key role for catastrophising. It is possible that neither pain related fear nor vigilance would be activated in the absence of catastrophic thinking.

These ideas about how the variables fit into a model which can explain their role in chronic pain will be returned to in Chapter 4 when aims and hypotheses are outlined. Prior to this, the next chapter will give an overview of the chronic pain conditions of interest and a definition of disability.

Chapter 3

Occupational Overuse Syndrome and Fibromyalgia

Occupational overuse syndrome (OOS) and fibromyalgia syndrome (FMS) are both musculoskeletal disorders. In the overview which follows they will be considered separately, but it will become apparent that there is substantial overlap between these conditions. (Carette, 1996; Macfarlane, Hunt, & Silman, 2000). The common ground pertains not just to similarities in symptoms but also to controversies of causation, disease status, psychological correlates, and disagreement over nomenclature. This is not intended as a review of the vast literature on OOS and FMS, but aimed at addressing some of the key points in relation to features, aetiology, and assessment of these disorders. It is also intended to provide a flavour of the controversial debate with information from several perspectives.

3.1 Occupational Overuse Syndrome

OOS is an umbrella term for a range of disorders recognised by the Accident Compensation Corporation (ACC) in New Zealand. This includes musculoskeletal disorders described elsewhere as repetitive strain or cumulative trauma injuries. Repetitive Strain Injury (RSI) is the preferred term in Australia, whilst in the USA Cumulative Trauma Disorder (CTD) is used. In the present study OOS is used quite specifically to refer to arm pain which is chronic in nature, with workplace features. In the following discussion terms from the article of origin will be used. Issues arising from naming will be returned to subsequently in this chapter.

OOS does not seem to be a new phenomenon. Melhorn (1998) provides a review of the history of the disorder. There are references to occupationally related symptoms dating back to the fifteenth century. However, the first thorough description of the problem came from an 18th century physician, Ramazzini, who noticed that scribes were suffering from fatigue in the hand and arm, attributable to the rigours of writing, (Macfie, 1995; Melhorn, 1998). The problems relating to overuse affect a substantial number of individuals. Melhorn

(1998) indicated that 15 - 20% of Americans have occupational diseases, while cumulative trauma disorders comprise just over 50% of occupational injuries.

3.2 The Nature of OOS

A task force for treatment providers was set up in New Zealand to help with the establishment of a programme of prevention. Part of this process was to decide on disorders that would be covered by the term OOS. A number of conditions were included under the OOS umbrella and these could be further grouped as localised inflammations, compression syndromes, or pain syndromes (Slappendel, 1996). The multifactorial nature of OOS causation was recognised by the task force, who argued that individual, organisational, and psychosocial factors all need to be considered in the management of the problem (Slappendel, 1996).

The definition recommended by the task force is as follows :

> Occupational Overuse Syndrome (OOS) is an umbrella term covering a range of disorders characterised by pain and/or other sensations in muscles, tendons, nerves, soft tissues and joints with evidence of clinical signs. Symptoms such as pain, discomfort, and muscle weakness may continue even after initial signs have diminished. (Slappendel, 1996, p. 21).

In addition to defining the problem and the disorders encompassed by the term 'OOS', a number of features of work related activity contributing to, or causing, the disorders were listed. These included the following : "prolonged muscle tension, repetitive actions, forceful movements, and sustained or constrained postures" (Slappendel, 1996, p. 21).

There is however no consensus in the literature that there is a clear pathway between particular activities associated with work and the development of musculoskeletal disorders as suggested by the term OOS. There are potential problems with using names such as OOS, CTD, and RSI, which imply different pathways of causation. Other disorders referred to in terms which are neutral in relation to causation, such as FMS, may also have occupational components. Macfarlane et al. (2000) advocated abandoning terms which mislead, such as CTD and RSI, and refers instead to 'forearm pain' (p. 678). However, this is potentially ambiguous. As Macfarlane et al. (2000) pointed out themselves arm pain seldom occurs distinct from other pain syndromes. The phrase 'localised fibromyalgia" suggested by Littlejohn (1995, p. 27) may be more useful, particularly as a more generalised variant may follow from the local form. Another issue raised by the lack of agreement

on nomenclature is that it makes comparison of the literature difficult. In his review, Littlejohn used the term RSI, but only as a "starting point" (p. 27). In this thesis OOS has been selected as the preferred New Zealand referent and this too is a starting point. Additionally, the literature predominantly deals with OOS and the term is recognised by sufferers. Thus, recruitment and review of the current state of knowledge were facilitated. The somewhat artificial separation of related disorders is also necessary in order to add to the growing body of knowledge that they are actually members of the same family.

The next section will outline features of OOS before moving to consider factors thought to be involved in causation of the disorder, methods of assessment, and the impact of psychological variables. This is a departure from the usual medical model of disease as described by Gerdle and Elert (1995), where pathology and how the condition is manifested follow from aetiology. The ordering in the next few sections fits better with the diversity of presentation and the controversy relating to causation.

3.3 Features

How an individual presents with OOS may vary greatly. For example, there are recognisable disorders of the muscle tendon unit including muscle strain, tenosynovitis, or nerve root entrapment (Littlejohn, 1995). However, in some cases (and Littlejohn (1995) referred to the "Australian epidemic of RSI", p. 27) pain and disability may not be congruent with damage to the muscle tendon unit. More persistent and widespread pain with features such as hyperalgesia and allodynia might be reported.

In the literature, OOS is divided into two variants. Helme, LeVasseur, and Gibson (1992) distinguished between '"well defined" and "poorly defined" musculoskeletal disorders (p.23). The latter category is characterised by "diffuse regional pain and an apparent lack of objective physical signs" (p. 23). These descriptions overlap with the variants referred to by Turner (1994) as Type 1 or Type 2 OOS. The former includes conditions such as rotator cuff syndrome, lateral and medial epicondylitits, nerve entrapment syndromes, tenosynovitis, and tendinitis. As outlined by Turner (1994), signs and symptoms of localised OOS depend on the particular syndrome implicated and the anatomical location. Additionally, the signs associated with injury may be more apparent in this subtype.

In contrast, Turner (1994) described the symptoms of type 2 OOS as "diffuse non-specific symptoms of a regional nature" (p. 6). Symptoms include aching, weakness, tenderness, stiffness, numbness, and tingling. Indicators of pathological processes may be lacking (Turner, 1994). Alternatively, swelling and discoloration may be visible and tender points

palpable (Turner, 1994). Pain varies from local to spreading pain, on a continuum from mild to severe, with associated sleep disturbance and psychological sequelae. Turner (1994) also suggested that as type 2 OOS becomes chronic it can be difficult to distinguish from fibromyalgia, indeed when treatment of OOS is unsuccessful it may progress to FMS.

The outline above highlights some difficulties in terms of the description of the phenomenon. What may start as an occupationally related musculoskeletal disorder progresses to a chronic pain syndrome, with levels of pain and disability dysynchronous with ongoing tissue damage (Littlejohn, 1995). Essentially, work related strain may develop into chronic pain involving the pathways and processes outlined in the first chapter of this thesis.

3.4 Aetiology

There is some controversy in the literature about the aetiology of OOS. Clear epidemiological models pertaining to work related musculoskeletal disorders are lacking (Davis, 1999; Melhorn, 1998; Wigley et al., 1992), although it is acknowledged that a number of factors are involved (Davis, 1999; Melhorn, 1998:). Davis (1999) commented that the varying pathological manifestations of musculoskeletal disorders were likely to develop as a result of individual and environmental factors, the latter category including those relating to work. The evidence for this statement was the incidence of the conditions across a broad range of occupational groups.

Thompson and Phelps (1990) commented on the association between symptoms of overuse and particular occupations, but they did not review the evidence for common musculoskeletal conditions. In contrast, Barton, Hooper, Noble, and Steel (1992) noted that clear associations between work and an upper limb disorder were reported only for peritendinitis crepitans, and for some cases of De Quervain's stenosing tenovaginitis. Writers cramp was also discussed as a recognised occupational disorder. There was equivocation in relation to a number of other disorders. For instance, awkward neck positioning at work could, it was suggested, lead to degenerative change. Additionally, carpal tunnel syndrome may be worsened by particular tasks. Tyrer (1994) appeared to be in agreement with Barton et al. (1992), classifying peritendinitis crepitans as work related and noting that hand cramps and pain were more prevalent in some occupations than others. However, Tyrer (1994) also stated that most other conditions are not clearly linked to particular occupational activities, apart from the use of vibrating hand tools. In contrast Ireland (1995) provided a long list of recognised overuse conditions. It would be fair to summarise the literature as lacking agreement on what conditions can be classified as OOS, with perhaps greater agreement on what does not constitute OOS. Tyrer (1994) was quite clear that the absence

of definitive signs, symptoms, and pathology associated with OOS indicated that the syndrome does not have disease status. Yassi (2000) agreed that overuse syndromes can not be considered as diagnoses, suggesting instead that they serve as labels for a collection of disorders resulting from particular postures or repetitive movements.

One theory of causation is that repetition leads to tissue changes in some individuals, with injury from repetition or overuse occurring when trauma exceeds the adaptability of the tissue (Sheon, 1997). However, while some authors have described limb pain resulting from repetitive actions (Thompson & Phelps, 1990), others have pointed out that a static posture may be more damaging than repetitive movement. For example, keyboard operators tend to complain of pain in the wrists rather then the fingers (Tyrer, 1994).

The mechanism by which repetitive movement or static posture might produce problems is not known, although a theory pertaining to muscle tension has received some attention in the literature. Researchers have proposed that sustained muscle contraction leads to a reduction in blood flow. Therefore, ischaemic muscle may be the origin of pain (Pritchard, Pugh, Wright, & Brownlee, 1999). Depletion of nutritional reserves means that the muscle switches to anaerobic metabolism and waste products accumulate (Turner, 1994; Wigley et al., 1992). Wigley et al. (1992) suggested that some of the products of anaerobic metabolism may stimulate pain receptors. Symptoms of numbness and tingling can also be explained by alterations in blood supply (Wigley et al., 1992). Additionally, Turner (1994) indicated that there is some evidence that OOS results from pressure on nerve roots. In a recent study Greening, Smart, Lynn, Leary, and Hall-Craggs (1999) found the mobility of the median nerve in the wrist was reduced amongst patients with chronic diffuse arm pain. Reduced mobility was thought to produce stress on the nerve during hand movements, further impacting on nerve function and pain.

3.5 Assessment and Diagnosis

There is no dependable test for the conditions encompassed by OOS. Evaluation of signs and symptoms, which are often non-specific, involves the assessors' interpretation (Davis, 1999). The lack of objective tests appears to make some medical practitioners sceptical about the existence of this disorder. Voiss (1995) comments on the need for "optimum and unequivocal objectivity" (p. 431) in relation to assessment and hypothesis testing. He goes on to assert that without rigour it is not possible to assess whether "...complaints of occupational or personal injury are fact, fantasy, or fraud." (p. 431). The implication is that without physical findings there is a tendency to regard presenting complaints as fantasy or fraud. Ireland (1995) writing about RSI is also troubled by the lack of objective

findings and labels the condition as a "psychosomatic symptom complex." (p. 54).

Research is still being conducted to establish markers for OOS. A study carried out by Helme et al. (1992) assessed flare response amongst participants with OOS (referred to in this study as RSI). Access to previous medical records revealed that when x-rays, bone scans, nerve conduction studies, and blood tests had been carried out no abnormalities had been detected. Helme et al. (1992) reported a reduction in capsaicin-induced flare response in the affected limb, while the flare response was increased in the unaffected limb. Pain intensity accounted for more than half of the variation in flare. While they indicated that the findings might be suggestive of an alteration in nociceptive function, it is not clear why opposing effects would be observed in the limbs. Mood disturbance was also noted and it was hypothesised that beliefs about the somatic origin of pain, together with denial of the impact of other factors, may have contributed to this (Helme et al., 1992).

Having outlined the variability in presentation, concerns about a clear pathway of causation, and issues arising from assessment and diagnosis, the final section of this chapter pertaining to OOS will provide an overview of the multiplicity of variables implicated in the onset and maintenance of the condition.

3. 6 The Multifactorial Nature of OOS

As already stated, there is no consensus in relation to causation of OOS. Hadler (1997) rejected the hypothesis that cumulative trauma produces regional arm pain. He reasoned that, for most people, particular occupations do not increase the incidence of arm pain. Following from this, Hadler proposed that pain, together with psychosocial factors in the workplace, results in an alteration in pain perception and increased incapacity.

Most commentators acknowledge that aspects of both the individual and the work place may be implicated in the aetiology of musculoskeletal disorders. Wigley et al. (1992) stated that OOS is caused by "bodily actions" (p.15) resulting from ergonomic, workload, and psychosocial factors in the workplace. Similarly, Bergqvist, Wolgast, Nilsson, and Voss (1995) reported on the link between individual, ergonomic and organisational factors, and activity related musculoskeletal disorders. In the study of Bergqvist et al. (1995), individual factors included age, negative affect, stress related symptoms, and female gender in association with having children at home. Amongst the organisational factors were flexibility in relation to tasks and rest periods, scarce or excessive peer contacts, and overtime. Peer contacts were particularly relevant for those with symptoms of stress. Finally, the ergonomic factors identified were particular static postures, hand positions and

arm support, together with repetitive movements, and keyboard location. In a study involving workers in the newspaper industry, Bernard, Sauter, Fine, Petersen, and Hales (1994) also reported that organisational, task, and psychosocial factors were associated with musculoskeletal pain conditions. In particular they showed that increased work demands, such as pressure to meet deadlines and more time spent at the computer, were linked with increased reporting of musculoskeletal disorders. After reviewing the literature, Buckle (1997) concluded that physical factors played a large role in musculoskeletal disorders, but that psychosocial factors such as time pressure and limited autonomy were also significant. Moreover, it is not easy to extract the relative contribution of each as interactions occur between them (Buckle, 1997).

In a New Zealand study, Stephens and Smith (1996) found that differences between groups reporting high versus low pain were related to aspects of the work environment. Amongst the factors associated with less pain reporting were increased support, more solidarity between colleagues, less pressure and stress, improved satisfaction, and more autonomy. However, it was also reported that more autonomy might buffer the effects of a higher work load. Although these data indicate that work place factors in New Zealand are related to differences in pain reporting, it is not possible to ascertain whether the above relationships are causal (Stephens & Smith, 1996).

Unfortunately, prospective and longitudinal studies are rare. Frederiksson et al. (1999) carried out a study to look at the links between social, psychological, and physical factors, and musculoskeletal disorders of upper limbs, neck, and shoulder. Data collected during a survey in 1969 was used for comparative purposes when follow-up was conducted 24 years later. The findings were that work and leisure activities, and an interaction between the two, were linked to musculoskeletal disorders. Notably, risk factors were not necessarily constant between genders, although age and cumulative effects were thought to be important factors generally.

Macfarlane et al. (2000) carried out a two year prospective study and concluded that a number of individual and workplace factors were independently related to development of forearm pain. In the first category, high scores on both the General Health Questionnaire (GHQ; Goldberg & Williams, 1988) and a measure of illness behaviour, in the presence of at least one somatic symptom, were linked to onset of forearm pain. In the workplace category, mechanical and psychosocial features were investigated. The results showed that both repetitive movements of the upper limb and satisfaction with support from peers and supervisors were reliably linked to future arm pain. Additionally, the authors commented that it was unusual for forearm pain to occur alone, more commonly pain was

present at several sites.

Research points to multifactorial aetiology for OOS, although clarity is lacking as to which factors are most salient in the development of either muscle tendon unit damage or the chronic pain syndrome. Many of the uncertainties alluded to in relation to OOS are relevant also for FMS. The sections which follow relate to FMS outlining features, aetiology, assessment issues, and research pertinent to this thesis.

3. 7 Fibromyalgia

Fibromyalgia syndrome (FMS) is a chronic musculoskeletal pain disorder which has only been known by this title since the late 1970s, although a number of the symptoms and signs have been recognised for considerably longer (Scudds & Li, 1997). Formerly, FMS was known by a variety of names including fibrositis, myofascial pain, muscular rheumatism, psychogenic rheumatism, and tension myalgia (Waylonis, Ronan, & Gordan, 1994). Prevalence estimates vary between 0.8% and 10.8%, with a lower incidence in underdeveloped countries (Scudds & Li, 1997). However, some of this variation may be attributable to the use of different diagnostic criteria (Okifuji & Turk, 1999). It is only comparatively recently that the American College of Rheumatology (ACR) formalised criteria for diagnosing FMS. The criteria are the presence of widespread pain, with localised tenderness at 11 out of 18 tender points (Wolfe et al., 1990). Widespread pain essentially means in all quadrants of the body and a minimum duration of 3 months is specified. Symptoms of fatigue, stiffness, and disturbances in sleep are common, but not universal in FMS, and are therefore not included in the diagnostic criteria.

3.8 Aetiology

A range of possible causative factors have been proposed for FMS. Okifuji and Turk (1999) categorised these as central or peripheral hypotheses, indicating also that the former probably offered the most useful explanation of the widespread nature of pain in FMS.

3.8.1 Central pain mechanisms

Yunus (1992) proposed that an interplay of peripheral and central factors leads to amplification of pain in FMS. Yunus' (1992) model outlined that neurohormonal dysfunction precipitated disordered central pain mechanisms, which reciprocally influence hormonal change. Yunus (1992) asserted that central mechanisms are most important and this view is endorsed by others (e.g., Okifuji & Turk, 1999; Wall, 1993; Yunus, 2000). Indeed Yunus

(2000) refers to FMS as a central sensitivity syndrome. Plasticity, which is involved in sensitisation in the CNS, has already been outlined in Chapter 1. Amongst the neurohormonal abnormalities enumerated by Yunus (1992) are alterations in the function of the hypothalamic pituitary adrenal (HPA) axis and a reduction of inhibitory neurotransmitters such as serotonin, or an increase in excitatory neurotransmitters. It is noteworthy that Yunus (1992) indicated stress or trauma may precipitate the neurohormonal changes. The second of these factors will be discussed later in this chapter.

3.8.2 Hypervigilance to pain or altered somatic awareness?

Rollman and Lautenbacher (1993) provided an alternative explanation of FMS arguing the centrality of hypervigilance to pain. This was based on a review of patterns of responding to clinical and experimental pain, and personality and response style research with FMS sufferers. Rollman and Lautenbacher proposed that FMS sufferers have a greater awareness of all aversive "perceptual experiences". Furthermore, they suggested that this is a "perceptual habit", rather than an "exaggerated appraisal of bodily experiences" (p. 156). It is not apparent how this distinction can be made.

Support for altered responding to pain in FMS comes from a number of sources, including the experimental research carried out by Kosek, Ekholm, and Hansson (1996) and McDermid, Rollman, and McCain (1996). One of the aims of Kosek et al. (1996) was to investigate whether sensory abnormalities amongst FMS patients were localised to areas of ongoing pain or were more generalised. The results indicated a generalised alteration in sensitivity, which the authors concluded probably implicated the CNS in the aberrant processes, although they do also raise the possibility that sensitisation of afferent pathways is involved. McDermid et al. (1996) compared participants with different rheumatological disorders and controls in relation to pain tolerance and threshold, and noise tolerance. Participants with FMS had lower stimuli thresholds and tolerance levels than both the rheumatoid arthritis and control participants. These findings indicated that hypervigilance may extend across sensory modalities.

Additionally, McDermid et al. (1996) showed that FMS participants reported experiencing more physical symptoms than either those with rheumatoid arthritis or controls, suggesting a raised awareness of somatic function. Robbins, Kirmayer, and Kapusta (1990) also looked at somatic awareness, together with concern about illness, collectively labelled "amplifying somatic style," (p. 50) to evaluate whether this impacted on distress and disability amongst sufferers with FMS and RA. The results showed that FMS was less disabling than RA, but more closely linked to somatic distress, although body awareness

was associated with somatic and depressive symptoms only amongst those with RA (Robbins et al., 1990). However, Peters, Vlaeyen, and van Drunen (2000) failed to demonstrate hypervigilance for innocuous stimuli amongst FMS sufferers. Peters et al. (2000) suggested that this may indicate that hypervigilance is only a feature of responding to noxious stimuli, countering the notion that individuals with FMS are hypervigilant in relation to all stimuli.

Different perspectives attributing FMS to either physiological or to perceptual disturbance are congruent explanations (Rollman & Lautenbacher, 1993). Okifuji and Turk (1999) pointed out that both involve disordered information processing. Additionally, both implicate central processes and a stronger relationship, one of reciprocal influence, might be argued in the light of the current state of knowledge regarding pain perception outlined in Chapter 1.

3.8.3 Sleep

Non-restorative sleep is a an important feature of FMS. Moldofsky, Scarisbrick, England, and Smythe (1975) provided evidence linking non restorative sleep and associated fatigue to what was then referred to as fibrositis syndrome. Disturbances in nonrapid eye movement (NREM) sleep were linked to increased tenderness at tender points and alterations in mood amongst FMS sufferers. Moldofksy et al. (1975) carried out a second study to establish whether disruption of NREM sleep would induce musculoskeletal pain and alterations in mood amongst healthy volunteers. The findings supported the hypothesis and this data forms the basis for theorising about the role of sleep abnormalities in FMS, although as Okifuji and Turk (1999) noted, later studies have not yielded consistent data.

3.8.4 Trauma

In New Zealand there has been recent interest in whether FMS satisfies criteria for accident or injury compensation by the ACC. The role traumatic injury may play in the development of FMS becomes a key factor in the ongoing debate. Rankin (1999) reported that a group of experts at a meeting in Wellington in 1998 were unable to reach a consensus on either causation or treatment of FMS. To satisfy the requirements of the Accident Rehabilitation and Compensation Insurance (ARCI) Act (1992) it is necessary to specify causation for entitlement to payment for medical treatment and other compensation (Rankin, 1999). Although, as Bennett (1998) noted, traumatic injury is often reported as preceding the onset of FMS, the literature is equivocal on this point. Rankin (1999) asserted that, in the absence of controlled studies evidence that FMS follows trauma only indicates "a

casual but not a causal relationship.." (p.19). A consensus report on fibromyalgia stated that the available evidence was not adequate to establish a causal link between trauma and FMS (Wolfe et al., 1996). However, an alternative perspective on the consensus report was provided by Yunus et al. (1997). This group indicated that while it is unusual in medicine to be able to clearly establish causality, there was a greater likelihood than the legally required 51% probability that trauma precedes FMS in some cases. Waylonis and Perkins (1994) conducted a follow up of post traumatic fibromyalgia patients. In this sample FMS was reported as following a range of injuries including motor vehicle accidents, work injuries, and surgery. It appears from the literature that an association is present between trauma and FMS, but this does not unequivocally demonstrate that the link is causal.

3.8.5 Muscle tissue abnormality

Many of the aetiological theories have focused their explorations on the increased sensitivity to pain that appears central to the condition. Because pain in FMS is in the muscle, early investigations concentrated on muscle tissue. Based on abnormalities in muscle tissue, early data suggested that FMS originated as a result of muscle tissue abnormality such as muscle degeneration or ischaemia (see Boissevain & McCain, 1991). More recent methodologically sound research has refuted this hypothesis (Simms, 1998; Weigent, Bradley, Blalock, Alarcon, 1998). Furthermore, Simms (1998) suggested that deconditioning amongst FMS patients may have produced some of the earlier findings.

3.8.6 Psychological factors

Theories attributing chronic pain to psychology have been disconfirmed. The literature on chronic pain populations generally indicates that emotional disturbance is a consequence, rather than an antecedent, of chronic pain (Gamsa, 1990; Turk & Rudy, 1992). No psychological characteristics have been reliably shown to discriminate between chronic pain sufferers and those without pain (Gamsa, 1994). Similarly, research findings amongst participants with FMS have not supported theories of psychological causation (Okifuji & Turk, 1999). However, research continues to evaluate the relative contribution of psychological factors to the development and maintenance of the disorder.

Yunus, Ahles, Aldag, and Masi (1991) investigated whether features of FMS are related to the musculoskeletal condition or to psychological abnormalities. The results showed that apart from pain no other FMS 'symptoms' were linked to MMPI categories. However, stress, depression, anxiety, and pain together did predict MMPI grouping. Yunus et al.

(1991) concluded that symptoms of FMS are syndrome related, but that pain severity may interact with psychological factors. There has also been research interest evaluating differences between chronic pain groups in relation to parameters other than pain. Birnie, Knipping, van Rijswijk, de Blecourt, and de Voogd (1991) compared FMS with a chronic pain group and a group without pain. They reported similar scores on measures of distress for both pain groups, which were significantly higher than scores obtained by participants without pain. Additionally, Ahles, Yunus, and Masi (1987) found that while individuals with either FMS or RA obtained a higher score than controls on a measure of depression, the symptomatic groups did not differ from each other. These findings all tend to support the notion that psychological aspects of FMS are related to chronic pain. The absence of between group differences in the study of Ahles et al. disproves the notion that depressive symptoms are more common in conditions where there is aetiological uncertainty than those with established causation. However, earlier comments about the overlap between somatic symptoms of depression and those of chronic pain are relevant to the research described in this section. As Birnie et al. (1991) acknowledged higher scores obtained by FMS participants on the somatisation scale of the Symptom Checklist (SCL90-R; Derogatis & Cleary, 1977) might reflect the extent of physical symptoms characterising this group.

In summary, the precise cause of fibromyalgia is not known, (Ledingham, Doherty, & Doherty, 1993). There are indications of the importance of CNS factors, although the mechanisms remains to be elucidated (Boissevain & McCain, 1991). Interactions between the CNS and other factors such as psychological distress may obscure some of the processes involved in the development and maintenance of FMS (Boissevain & McCain, 1991). The co-occurrence of psychological distress and FMS has been interpreted by some commentators such as Bohr (1995) as the former causing the latter, although categorising pain as organic or non-organic is becoming less common in the literature (Bennett, 1998). Neuroendocrine, immunological, muscle, and sleep abnormalities have been found in people with FMS, but these may be incidental findings. Evidence is lacking that tests of these variables are of diagnostic value (Wolfe et al., 1996). Additionally, the heterogeneity of FMS sufferers makes interpretation of findings difficult (Okifuji & Turk, 1999).

3. 9 Assessment Issues

In the absence of a clear aetiological basis for FMS the American College of Rheumatology (ACR) have published criteria for diagnosis of FMS based on tender points, a move which has been controversial (Ford, 1997). In particular, there has been debate about the number, site, and means of estimating tenderness (Cohen & Quintner, 1993). Wolfe et al. (1990) reported that tender points provided the best discrimination between individuals

with FMS and controls. Additionally the presence of 11 out of 18 tender points with widespread pain provides high levels of sensitivity and specificity in relation to diagnostic accuracy. However, research on tender points has produced equivocal results. Tunks, Crook, Norman, and Kalaher (1988), in a study to validate a pressure algometer, found that individuals with FMS were more tender than controls at tender and non-tender points. Notably, between group differences were most marked at non-tender points. As Cohen and Quintner (1993) commented, these and similar findings raise doubts about the validity of tender point assessment.

In more recent research, Wolfe (1997) investigated relationships between tender point count, dolorimetry, symptoms of FMS, and a global distress measure. The findings were that a linear relationship exists between tender points and FMS variables and distress. On the basis of this data, Wolfe (1997) suggested that symptoms of FMS occur on a continuum and that recognition of distress is important regardless of whether a diagnostic threshold is reached. Essentially, Wolfe (1997) is proposing that there is no clear cut off where FMS does or does not exist, which in a sense undermines the usefulness of the diagnostic criteria. Medical examination, including tender point count remains a key component of assessment but, as indicated earlier, evidence is lacking as to the utility of laboratory tests. Therefore, assessment of the severity of FMS relies almost exclusively on self report data (Wolfe et al., 1996).

Assessment of disability is similarly reliant on self report and subject to a host of influences (Waddell, 1998). White, Harth, and Teasell (1995) enumerate a number of reasons which make disability disputable in FMS. However, many of these are relevant for OOS and indeed for other chronic pain conditions. Therefore, reference to these issues in the present section on FMS is for convenience only. Uncertainties about diagnosis, difficulties with objective evaluation of disability, and co-occurring psychological difficulties are amongst the factors outlined by White et al. that make disability estimates problematic in FMS. Before considering the evaluation of disability, it is important to define what is meant by the term.

According to the World Health Organisation (WHO; 1980) : "In the context of a health experience, a disability is any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being." (p. 28). The definition includes impairment as a possible pathway to disability, but impairment is defined separately in the same context as "... any loss or abnormality of psychological, physiological or anatomical structure or function" (p. 27). Although pain

would be categorised as an impairment according to the WHO criteria (Gerdle & Elert, 1995), Waddell (1998) argues that the WHO classification system is more applicable to observable impairments. Pain is a symptom, which cannot be objectively measured, and it does not always equate with, or progress to, disability; other factors are involved (Waddell, 1998). Disability rather than impairment is used in the present thesis because of its broader scope, and because it has facilitated comparison with the extant chronic pain literature.

There are a number of ways of determining disability. Waddell (1998) suggested using activities of daily living as the basis of assessment for back pain can reliably elucidate the impact of pain. Further, he proposed that activity restriction rather than the presence of pain should form the focus of questioning. However, this separation may prove difficult for the pain sufferer and the assessment essentially relies on their subjective experience. Unfortunately, although objective measurement is an often stated ideal in psychological research it is probably not readily achievable. For example, functional capacity evaluation actually assesses performance, rather than capacity to perform, and thus depends to some extent on motivation (Waddell, 1998). Additionally as Gronblad et al. (1994) pointed out the results obtained from a functional assessment are only relevant for a particular day or time of day. Functional assessment may also require interpretation by the assessor and hence may be coloured by their appraisal of the client.

Waddell (1998) noted the links between pain, days lost from work, and disability, but also emphasised that other factors are involved in the development of disability and that pain and activity restriction should be assessed separately. In the final section of this chapter some of the literature pertaining to disability amongst FMS sufferers will be outlined.

3.10 Literature Linking FMS and Disability

The relationships between pain intensity, beliefs, and disability in chronic pain patients, predominantly those suffering with CLBP, were described in Chapter 2. Much of this literature demonstrated that factors other than pain were key determinants of disability. However, in a sample with FMS, Turk, Okifuji, Sinclair, and Starz (1996) found that pain intensity was highly correlated with disability. This relationship was replicated by Hawley and Wolfe (1991) amongst FMS participants in their study, but RA sufferers in the same research reported high levels of disability in association with lower levels of pain. It was interesting that Turk et al. (1996) also reported that in most cases physical functioning was not associated with disability. Commenting on this finding, Turk et al. (1996) drew attention to the restrictive operationalisation of physical function in the project, but nonetheless the result merits further investigation. As it stands the data suggests a discrepancy

between beliefs about functioning and actual function (Turk et al., 1996).

Robbins et al. (1990) found that FMS sufferers reported less disability than the RA group. However, disability and activity reduction were present in the FMS group and these variables were significantly correlated with illness concerns amongst FMS sufferers (Robbins et al., 1990). It may be, as suggested by Robbins et al. (1990), that uncertainty surrounding diagnosis of FMS is reflected in the importance of cognitive factors in relation to disability for these participants. The literature appears to be sparse in relation to cognitive factors and FMS, although two recent studies have looked at pain, disability, and coping measured by the CSQ in this population. Nicassio, Schoenfeld-Smith, Radojevic, and Schuman (1995) investigated the relationship between pain, coping, and disability amongst participants with FMS, conducting cross sectional and longitudinal analyses. The CSQ was used to quantify coping and two factors were derived. These were described by the authors as coping attempts, and pain control and rational thinking (PCRT). The latter comprised the catastrophising scale plus two items pertaining to both control and the ability to reduce pain. Low scores on the PCRT factor were linked with increased depression and pain behaviour, while high scores were associated with an enhanced sense of wellbeing. Active coping was negatively associated with concurrent depression as might be anticipated, but surprisingly was positively related to more pain, lower well-being, and increased depression over time.

Martin et al. (1996) also used the CSQ to evaluate the relationship between coping strategies and disability. In contrast to Nicassio et al. (1995), Martin et al. (1996) used a broader measure of disability and controlled for neuroticism rather than depression. They also found that the CSQ comprised two factors, coping attempts and catastrophising, the latter without any additional items. In relation to physical disability those endorsing greater use of coping attempts reported more physical disability, while catastrophising was not linked to physical or psychosocial disability. Interestingly, the scores of these participants on CSQ effectiveness ratings point to limited efficacy for coping strategies in this population, although these ratings are not linked to disability. Coping attempts were associated with lower levels of psychosocial disability, while catastrophising was related to psychosocial disability only in the presence of neuroticism. Additionally, catastrophising was related to total disability, in the absence of a relationship with the variables from which it was derived.

There are some surprising and counterintuitive results in these studies. For instance, the suggestion that active coping is associated with poorer outcomes and also that the effectiveness of coping does not impact on disability. Replication is needed to clarify the

relationship between catastrophising and disability amongst individuals with musculoskeletal disorders. The use of alternative assessment measures would also be advantageous in the light of the potential difficulties with the CSQ factors outlined earlier, which are particularly pertinent for the study of Nicassio et al. (1995) where catastrophising is incorporated into a factor with other items.

In the chapter which follows, aims and hypotheses are outlined and relevant points from the literature reiterated to provide some context for each.

Chapter 4

The Present Study

4.1 Aims

Data presented in this thesis derives from a larger study investigating vibration perception and hyaluronic acid levels amongst participants with FMS, OOS, and an asymptomatic comparison group. There were three broad aims in the present study. The first aim was to evaluate between group differences in pain-related and cognitive variables; and the second aim was to assess the relevance of the fear component of the model proposed by Vlaeyen and colleagues for this sample. The third aim, related to the second, was to explore the association between pain and disability considering the potential intervening effects of a number of cognitive variables. Commentary in the chronic pain literature (e.g., Keefe, Dunsmore, & Burnett, 1992) points to a general need to clarify which cognitive constructs are most informative in relation to pain and disability. Additionally, Turk and Rudy (1992) suggested that further investigation is needed to assess the extent of overlap between cognitive variables. These two issues appear to be linked. It is necessary to look at areas of overlap between cognitive factors in order to establish their unique contribution to disability. These points will be considered further in the results and discussion.

In the next section outlining study hypotheses, the first three hypotheses pertain to between group differences and the last three to the model of Vlaeyen, Kole-Snijders, Rotteveel, et al. (1995), and to the relationship between pain, cognitive variables, and disability.

4.2 Hypotheses

Hypothesis 1

That there will be significant differences between women with OOS and FMS in relation to pain, catastrophising, fear of movement, vigilance, and disability.

The literature suggests that the musculoskeletal disorders FMS and OOS have substantial

areas of overlap, rather than being discrete conditions (Carette, 1996; Macfarlane et al., 2000). The defining characteristics of FMS relate to the presence of tender points and the extent of pain; that is, how widespread the pain is. If the disorders do exist on a continuum then it might be anticipated that differences would not solely be a feature of diagnostic classification, but rather a function of symptomatic severity. Assuming that FMS is a more severe variant it was anticipated that, overall, there would be significant differences between participants with OOS and FMS in relation to pain, catastrophising, fear of movement, vigilance, and disability.

Hypothesis 2

That women with FMS will make more somatic cognitive errors than either women with OOS or a female comparison group.

Moss-Morris and Petrie (1997) found that somatic rather than general cognitive errors characterised populations with chronic illness. It was anticipated in the present study that participants with chronic pain would make more somatic cognitive errors than either those with OOS or a comparison group with no musculoskeletal symptoms.

Hypothesis 3

That tender points will serve as an index of general symptomatic severity and in particular be associated with more pain and disability, fear of movement, catastrophising, and cognitive distortion. Additionally, as a function of these relationships, tender points will contribute significantly to prediction of disability.

Wolfe (1997) found a strong relationship between tender points and distress variables and suggested that tender point assessment might prove useful to index symptomatic severity and distress amongst the population of FMS sufferers. It was expected in the present study that there would be a positive correlation between increasing numbers of tender points (paralleling pain extent), and fear of movement, catastrophising, cognitive distortion, and disability. Additionally, by virtue of these relationships, tender points would contribute significantly to the prediction of disability and thus serve a useful purpose as an index of general symptomatic severity.

Hypothesis 4

That higher levels of catastrophising and greater pain intensity will be associated with increased fear of movement, and a tendency to catastrophise will also be linked to greater vigilance for pain related cues and more pain reporting.

Vlaeyen, Kole-Snijders, Rotteveel, et al. (1995) proposed that identification of fear of movement together with catastrophising was the key to preventing chronic back pain disability. However, when the present study commenced there appeared to be no published literature investigating the relevance of fear of movement/reinjury for populations with FMS and OOS. In addition, data on catastrophising amongst these client groups are sparse. A preliminary assessment of the relevance of pain related fear for the current study population involved comparison of mean scores for fear of movement derived from the present sample with data from CLBP participants published by Crombez, Vlaeyen, et al. (1999), and the more recent research of Peters et al. (2000) involving women with FMS. It was anticipated that the data from these comparisons would be similar.

Vlaeyen, Kole-Snijders, Boeren, et al. (1995) reported that pain intensity was significantly associated with fear of movement/reinjury, but the correlations between fear of movement and catastrophising, and fear of movement and depression, were of greater magnitude than that between pain and pain related fear amongst CLBP sufferers. It was anticipated that higher levels of catastrophising and greater pain intensity in the present sample would both be positively associated with increased fear of movement. There is also data from non-clinical populations which points to catastrophisers attending more to painful stimuli than non-catastrophising counterparts, although they do not necessarily report more intense pain (Crombez, et al., 1998b). It was envisaged that symptomatic individuals in the present clinical sample who were more prone to catastrophise would also be more vigilant for pain cues and in this context be likely to report more pain.

Hypothesis 5

That symptom duration will be positively associated with disability, pain, and vigilance, but negatively linked to catastrophising and fear of movement.

The literature is not clear on either the relationship between pain duration and disability, or how beliefs about pain and duration of pain are linked. As outlined earlier, there are several studies associating pain-related beliefs and fears with pain of shorter duration (Jensen et al., 1994; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). Vlaeyen, Kole-Snijders, Rotteveel, et al. (1995) explained this result in terms of pain related fear extinguishing over time. However, Vlaeyen, Kole-Snijders, Boeren, et al. (1995) found in one

of their two studies that greater fear of pain was linked to greater chronicity. It was anticipated in the present sample that symptom duration would be positively associated with disability, pain, and vigilance, but negatively linked to catastrophising and fear of pain. This would mean that participants who had experienced symptoms for longer would describe themselves as more disabled and be more vigilant about pain, but would not be more likely to catastrophise or be fearful of pain.

Hypothesis 6

That higher levels of pain, catastrophising, fear of movement, and vigilance will all be positively associated with disability and that the positive correlations between pain and disability, and between cognitions and disability, will be comparable. Additionally, that after controlling for the effects of age and duration of pain, pain-related fear, vigilance, and catastrophising will be as disabling as pain itself.

Psychological factors are now known to be important predictors of disability in chronic pain sufferers. Some research amongst CLBP sufferers has demonstrated that pain- related fear is actually more disabling than pain itself (Crombez, Vlaeyen, et al., 1999; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). Additionally, Turner et al. (2000) reported that beliefs, coping, and catastrophising explained substantially more of the variance in disability than was explained by pain and demographic variables in a heterogeneous chronic pain sample. However, in a population with FMS; Turk et al. (1996) reported a strong relationship between pain intensity and perceived disability, but for many of the participants perceived disability and physical function were not closely associated. It was anticipated in the present study that pain and disability would be positively associated at the bivariate level, and that the correlations among fear of movement, attention to pain, catastrophising, and disability would also be significant. Furthermore, it was expected that the positive correlations between pain and disability, and between cognitions and disability, would be comparable in magnitude. In terms of the multivariate model, after controlling for the effects of age and duration of pain, it was anticipated that pain-related fear (fear of movement), vigilance, and catastrophising would prove to be as disabling as pain itself.

Chapter 5

Method

5.1 Participants

The inclusion criteria for the study were female gender aged between 16- 60 with symptoms of FMS or OOS, or for the comparison group, the absence of symptoms relating to these conditions. Ninety four participants were recruited. Group membership was established using The American College of Rheumatology (ACR) criteria for fibromyalgia, which were outlined in Chapter 3. On the basis of this diagnostic classification the group membership within the sample was as follows : 50 (53.2%) OOS; 18 (19%) FMS; and 24 (25.5%) comparison group. A tender point rating was not carried out for two participants and therefore diagnosis could not be made according to ACR criteria. These participants were excluded from the analysis. The age range of the sample was 17 - 60 with a mean of 42.5 years (SD = 10.75). There were no significant differences between groups in relation to age.

Symptomatic participants were recruited in one of four ways. 1) From three local rheumatologists, 2) From a Clinical Psychologist specialising in pain management. 3) Several symptomatic participants contacted the research team after noticing a flyer for control participants. 4) Symptomatic participants involved in FMS and OOS local support groups voluntarily alerted other members of these groups, who contacted the research team. The comparison group was recruited by either snowball sampling (participants were invited to bring a friend of similar age to take part in the study), or in response to a notice (see Appendix A) advertising the study, which was placed at Massey University and at the local rehabilitation medicine unit.

5.2 Design

A cross-sectional, self report method was used. Information was also obtained for the purposes of diagnosis by medical assessment and examination. The design was between subjects.

5.3 Procedure

Recruitment involved the symptomatic individual being approached by the clinician responsible for their care (i.e., any of the three rheumatologists, or the clinical psychologist). The study purpose and procedure were outlined. Those who indicated their willingness to participate were telephoned by the research assistant, who reiterated the study process. Potential participants who were willing to participate were reminded that involvement in the study was voluntary, and then were informed about the location of the study and given a time to attend. A request was made that if possible analgesic medication should be avoided for 24 hours before attending, but that all other regular medications should be taken as usual. Some participants were taking amitriptyline to regulate either mood, sleep, or pain. It was considered inappropriate to suggest participants should stop this medication as a number of the therapeutic effects are of long duration. Participants were also asked not to exercise vigorously 24 hours before attending, as it was hypothesised that exercise could produce fluctuations in hyaluronic acid (HLA) levels.

Participants were seen in two adjacent rooms on the third floor of the School of Psychology at Massey University. Participants moved sequentially from the psychology research assistant to a medical practitioner. On arrival, participants were provided with written information outlining study aims. They were given another opportunity to ask questions before signing the consent form (see Appendix A for copies of the information sheet and consent form). The questionnaires were presented as a booklet entitled "Questionnaires about your pain and associated thoughts" (see Appendix B). Titles were removed from the individual questionnaires. The ordering in the method section reflects that in the booklet. Questionnaires were completed prior to the vibration perception testing and blood test. Subsequently, participants progressed to the second room for the medical assessment which included tender point evaluation.

The first questionnaire in the booklet was explained to participants who were asked to complete the initial section in relation to their usual pain, putting a tick or cross by the adjectives most applicable. The visual analogue scale and present pain intensity score were also explained, and participants were asked to complete these in relation to pain they were experiencing at the time of completion. For the remainder of the questionnaires only a general overview was provided as each measure was accompanied by instructions for completion. It was explained that the items in the second questionnaire were about disability associated with pain, and that the remainder of the questions in the booklet were addressing different aspects of thoughts about pain. Participants were invited to ask the research assistant if they did not understand questionnaire instructions, or the content of

specific items.

5.4 Materials and Measures

The questionnaire booklet contained measures pertaining to pain, pain-related disability, vigilance and awareness for pain, fear of movement/reinjury, pain catastrophising, and cognitive errors. In addition, the medical practitioner also provided quantification of tender points. The section that follows will review each of the measures including content, reliability, and validity information.

The Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987)

Three pain scores can be derived from this measure which is a short form of the McGill Pain Questionnaire (MPQ; Melzack, 1975). The SF-MPQ comprises 15 adjectives relevant to the experience of pain, 11 of which are sensory and 4 affective, a visual analogue scale (VAS), and the present pain intensity (PPI). The respondent rates the adjectives on a 0 - 3 scale (0, 1, 2, and 3 corresponding to none, mild, moderate, and severe). When compiling the SF-MPQ the adjectives selected from the original MPQ were those chosen by 33% of respondents across a range of pain types (Melzack, 1987).

Reliability and validity data for the original longer variant of the MPQ are adequate. Correlations between the categories of the MPQ were reported by Melzack (1975) as evidence of internal consistency. A comparison of short and long forms demonstrated that there were significant correlations between them for all parameters, although the original variant does not include a VAS. Furthermore, this was true for a range of pain types, including musculoskeletal pain, post surgical pain, and labour pains, and for English and French versions (Melzack, 1987). There is also some evidence for the sensitivity of the SF-MPQ to changes resulting from interventions to treat pain again pointing to the validity of the measure (Melzack, 1987).

Multidimensional assessment of pain is considered to be important and the questionnaires were designed to capture information on three components of this experience (Melzack, 1987; Melzack & Katz, 1992). In the present study all three components were significantly correlated, although one of these pertained to usual pain, rather than pain on the day. A decision was made to use only the VAS in subsequent analyses.

The VAS has been extensively used to measure pain intensity (Carlsson, 1983; Melzack & Katz, 1992). Carlsson (1983) evaluated comparative and absolute forms of the VAS finding the latter to be least susceptible to bias. Additionally, Carlsson (1983) made some

interesting points of relevance to the measurement of pain in the present study. She suggested the presence of a carry over effect from completion of several pain rating scales on one occasion. This may explain the relationship between the components of the SF-MPQ, although they were used to tap current versus usual pain.

The Pain Disability Index (PDI; Pollard, 1984)

This instrument was used to assess the extent that pain interferes with a range of activities of daily living. The questionnaire elicits information in seven areas: family and home, recreation, social activity, occupation, sexual activity, self-care, and life support. Respondents are asked to endorse each item using a 0 - 10 rating scale, where 0 equates with no disability and 10 with total disability.

The PDI has been shown to discriminate between groups categorised as having high or low disability levels (Pollard, 1984; Tait, Pollard, Margolis, Duckro, & Krause, 1987). Higher PDI scores are related to increased levels of psychological distress, more intense pain, and greater activity restriction. Additionally, PDI scores have been linked with pain behaviours (Tait, Chibnall, & Krause, 1990). However, Tait et al. (1990) did add some qualifying comments in relation to both activity restriction and pain behaviour. Only three predictors explained more than 3% of the variance in disability. These were days in bed (26%), psychosomatic symptoms (7%), and stopping activities (5%). Tait et al. (1990) suggested that the findings might have been attributable to multicollinearity. Alternatively, there may be questions about the value of the PDI as an addition to estimates of up or down time, although the lack of correlation between the PDI and "down time" perhaps makes the latter option less likely (Tait et al., 1990). In the discussion, Tait et al. proposed that the absence of a relationship between disability and down time in this study may have resulted from treatment interventions to discourage this behaviour.

Strong, Ashton, and Large (1994) evaluated the reliability and validity of the PDI and the Oswestry Disability Questionnaire (OLBPDQ or ODQ; Fairbank, Couper, Davies, & O'Brien, 1980). Their findings provided evidence for the reliability and concurrent validity of the measures, but they indicated that more data was needed on construct and predictive validity. Additionally, they pointed out that both measures were moderately correlated with depression. Gronblad, Jarvinen, Hurri, Hupli, and Karaharju (1994) added to the knowledge base on construct validity of the PDI in a study investigating the relationships between performance impairment on three physical tasks, the PDI, and the ODQ, using a heterogeneous back pain sample. There were significant correlations between impairment on the physical tasks and self reported disability rated on both measures. However, adjustment for age and gender impacted on the significant association between the

tasks and both measures. In particular, the correlation between the PDI and sit ups became nonsignificant. It was also noteworthy in this study that participants on sick leave achieved higher scores on measures of disability with worsened performance on physical tasks. Gronblad et al. (1994) concluded that the data contributes to existing evidence for reliability and validity of the PDI, but noted that high correlations between pain and performance are not inevitable.

Reliability data for the PDI are adequate. Internal consistency is reported as ranging from .76 to .86 and .87 (Strong et al., 1994; Tait et al., 1987; Tait et al., 1990). Item-total correlations computed by Tait et al. (1987) ranged from .56 - .85. Removal of item 7 relating to life support altered the range, .70 - .85. A principal components analysis yielded a two factor solution with reliability data for the two factors of .85 and .70, (Tait et al., 1990).

Tait et al. (1990) expressed some concerns with initial test-retest reliability data for the PDI. There was a significant correlation between scores on both occasions of testing, but this was lower than expected $r = 0.44$. Gronblad, Hupli, Wennerstrand, Jarvinen, Lukinmaa, Kouri, and Karaharju (1993) compared the PDI and ODQ in terms of test-retest reliability, finding both to be acceptable. An intraclass correlation (ICC) of .91 was reported for the PDI. In the present study the Cronbach's alpha was .91. Deletion of items in this questionnaire would not have improved the internal consistency.

The Pain Vigilance and Awareness Questionnaire (PVAQ; McCracken, 1997)

This measure was designed to evaluate "... awareness, vigilance, preoccupation and observation of pain" (McCracken, 1997, p. 271). This measure includes 6 items from the bodily vigilance questionnaire (BVQ; Mueller, Telch, & Curry, 1992). The remaining items were constructed on a rational basis, the aim being to include ideas such as focusing or dwelling on pain. Respondents are asked to answer items in relation to behaviours during the preceding two weeks and to indicate frequency on a 0 - 5 scale, where 0 equals never and 5 always. In this single study, internal consistency and test-retest reliability were adequate, respectively .86 and .80. In the present study Cronbach's alpha was .89. Removal of item 16, which seemed to have caused some confusion for several of the early participants, would not have improved the internal consistency, so the item was included.

Evidence for the validity of the PVAQ comes from correlational data reported by McCracken et al. (1997). PVAQ scores were positively related to body consciousness, pain related interference, "cognitive disability", anxiety, depression, pain intensity, disability, and doctor visits (McCracken et al., 1997, p. 278.). Surprisingly, PVAQ scores were also positively associated with diverting attention from pain, and not related significantly to somatic

symptoms. A regression analysis revealed that vigilance for pain related cues predicted distress, disability, and use of health care, independently of pain intensity and of demographic variables (McCracken, 1997).

Tampa Scale for Kinesiophobia (TSK; Miller et al., 1991)

The Tampa Scale was developed to measure pain related fear, specifically fear of movement/reinjury. Subsequent published research has largely used a Dutch version of the measure. The TSK comprises 17 items accompanied by a 4-point likert scale ranging from "strongly disagree" to "strongly agree". The item total is calculated after inversion of scores for items, 4, 8, 12, and 16. Item 6 was altered for the present study, replacing the word accident with condition.

Published data on reliability pertains to the Dutch version of the Tampa Scale for Kinesiophobia (TSK-DV; Vlaeyen, Kole-Snijders, Boeren, et al., 1995). The distribution of scores was reported as being normal (Vlaeyen, Kole-Snijders, Boeren, et al., 1995). Adequate reliability for the measure has also been reported with internal consistency data in the range of .68 - .80 (Crombez, Vlaeyen, et al., 1999; Vlaeyen, Kole-Snijders, Boeren, et al., 1995). Factor analysis yielded a four factor structure : harm, fear of injury, importance of exercise, and avoidance of activity (Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). However, there were intercorrelations between factors and use of the total score may be more prudent in view of the reliability and validity of the total measure (Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). Additionally, Crombez, Vlaeyen, et al. (1999) cited correlations between the two subscales of the Fear-Avoidance Beliefs Questionnaire (FABQ) as evidence for construct validity. In the present study the internal consistency was even better than that reported elsewhere (Cronbach's alpha . 85).

The Pain Catastrophising Scale (PCS; Sullivan, Bishop, & Pivik, 1995)

This measure was developed to measure catastrophising in a range of populations. Participants are asked to recall a recent painful experience and in relation to this to rate 13 thoughts or feelings listed in the questionnaire on a five point scale (0 = not at all to 4 = all the time). The process of scale validation utilised student and clinical populations, and a comprehensive account is provided by Sullivan et al. (1995). Internal consistency of the scale was adequate. Sullivan et al. (1995) reported the Cronbach's alpha as .87. Comparable reliability was demonstrated for a Dutch translation of the scale (PCS-DV). Crombez et al. (1998b) reported the Cronbach's alpha as .85. In the present study the Cronbach's alpha was .93. Temporal stability of the PCS has also been shown, $r = .75$, over a ten week period.

Factor analysis has yielded varying results. Sullivan et al. (1995) demonstrated a three factor solution. The factors were labelled rumination, which accounted for the largest proportion of total variance, magnification, and helplessness. Moderate to high correlations were found between factors. Sullivan et al. (1995) reported Cronbach's alphas for rumination, magnification, and helplessness respectively as : .87, .60, and .79. In contrast, Osman, Barrios, Kopper, Hauptmann, Jones, and O'Neill (1997) produced a two factor solution, but in a smaller sample than that of Sullivan et al. (1995). However, they also provided further evidence for the reliability and validity of the measure as a whole. This includes a positive relationship between scores on the PCS and negative thoughts about pain, but no relationship between the PCS and demographic variables (Osman et al., 1997). Additionally these authors reported that the PCS subscales can be differentiated from anxiety and depression. This supports the conclusions of Sullivan et al. (1995) that catastrophising can be distinguished from mood and anxiety variables.

The Cognitive Errors Questionnaire - Revised (CEQ-R; Moss-Morris & Petrie, 1997)
This measure has been developed from the Cognitive Errors Questionnaire (CEQ; Lefebvre, 1981) The original instrument comprised 48 items. The item content consisted of vignettes followed by a cognitive error reflecting either catastrophising, over generalisation, personalisation, or selective abstraction. Participants were asked to rate how similar the thought expressed in the item was to their own in similar circumstances. Half of the vignettes were general, the remainder incorporated themes associated with a low back disorder. Reliability and validity data for the original were adequate (see Lefebvre, 1981).

Moss-Morris and Petrie (1997) modified the original CEQ producing the CEQ -R. The number of items were reduced but the scope of the measure broadened. Additionally, items relevant only to American respondents were removed. The general vignettes remain, but the pain specific vignettes were replaced by some of relevance to a range of somatic problems including, but not specific to, chronic pain (Moss-Morris & Petrie, 1997).

The original structure has been preserved. Each item comprises a vignette followed by a thought someone might have in that situation. The thoughts reflect cognitive errors such as catastrophising, over generalisation, personalization, and selective abstraction. The respondent is asked to rate these on a five-point scale from "almost exactly like I would think" to "not at all how I would think". Factor analysis yielded a two factor solution accounting for 56% of variance. The two factors match with the items included in the general and somatic CEQ-R (Moss-Morris & Petrie, 1997).

Cronbach's alphas reported by Moss-Morris & Petrie, (1997) for the total CEQ-R, So-

matic CEQ-R, and General CEQ-R were .95, .93, and .90 respectively. Test-retest reliability for the total CEQ-R was adequate amongst the sample of chronic fatigue participants, $r = .83$, but poor for depressed participants, $r = .35$. As the authors pointed out this might have reflected the small sample size. In the present study Cronbach's alphas for the total measure, general, and somatic subscales were : .92, .90, and .86 respectively.

Moss-Morris and Petrie (1997) reported that all symptomatic participants, which included individuals with chronic fatigue syndrome (CFS), depression, and chronic pain, obtained a significantly higher score than controls on the somatic cognitive errors subscale. However, only the scores of depressed participants were significantly different from controls on the general subscale of the CEQ-R. Hence the suggestion of Moss-Morris and Petrie that the instrument may help to distinguish between overlapping conditions. General cognitive errors were significantly positively associated with symptoms of depression, self and symptom focus, and had a significant negative association with self esteem. In comparison somatic cognitive errors are significantly associated only with symptom and self focus. Notably, the closest links exist between symptom focus, but not somatic symptoms and the somatic subscale, and between self focus and the general subscale (Moss-Morris & Petrie, 1997). Additionally, Moss-Morris and Petrie evaluated the links between the subscales, psychological variables, and disability. Negative affect and disability were predictors of somatic cognitive errors in participants with CFS, but the reverse did not hold. Moss-Morris and Petrie commented that it did not appear that somatic cognitive errors were causal factors, but the link between somatic cognitive errors, mood, and disability over time, indicated that particular thoughts may be involved in the persistence of conditions such as CFS. In contrast, the general subscale was associated with mood and anxiety variables but not disability (Moss-Morris & Petrie, 1997).

Tender Point Assessment

Tender point assessment was conducted by an experienced clinician. The assessment of tender points has been described as "partly objective" (Wigley, 1999, p. 3). The procedure allows the clinician to control force applied to the site while observing for behavioural responses such as flinching or verbalisation (Wigley, 1999). Additionally, as outlined in Chapter 3, tender points have been shown to discriminate well between those with FMS and controls (Wolfe et al., 1990).

The ACR criteria are as follows :

> The presence of 11 out of 18 tender points (defined as mild or greater tenderness) in the presence of widespread pain provided the most sensitive, specific, and accurate

criteria for the diagnosis of primary, secondary-concomitant, and the combined fibromyalgia syndrome. (Wolfe et al., 1990, p. 169.).

However, as alluded to in Chapter 3, the assessment of tender points is controversial. The means of measurement of tender points is one aspect of this controversy. The ACR criteria recommend "Digital palpation should be performed with an approximate force of 4kg" (Wolfe et al., 1990, p.171). Additionally, for the tender point to be counted the participant must state that it is painful, tenderness will not suffice (Wolfe et al., 1990). Either palpation or dolorimetry can be used to assess tender points. As Okifuji, Turk, Sinclair, Starz, and Marcus (1997) pointed out, both have some advantages. Dolorimeters indicate the degree of force applied to the area, but lack the benefits of direct manual contact. In the present study a combination of digital palpation (used at the occipital site), and syringe dolorimetry was used. The latter was shown to be a reliable and sensitive instrument for assessment of joint tenderness. Langley, Fowles, Sheppeard, and Wigley (1983) provide a detailed account of the assembly and calibration of the instrument. Tenderness at less than 2kg. of force is considered a lowered threshold or tender point. In normal individuals, a force of greater than 3kg. can be exerted at the usual FMS tender points (Wigley, 1999). Assessment was carried out at the sites specified by the ACR and at six additional sites.

5.5 Data Analysis

The Statistics Package for the Social Sciences (SPSS) for Windows, Release 10.0.5 (1999) was used to analyse data. The alpha level for hypothesis testing was $p < .05$ unless otherwise specified. Hypotheses were one-tailed. Adjustment of the alpha level for multiple comparisons is discussed in the text at the point where this was carried out.

Descriptive statistics were used to summarise the data. Correlation coefficients allowed preliminary exploration of the relationships between study variables. Independent *t* tests were used for between group comparisons in relation to single pairs of variables for symptomatic participants, while one way ANOVA allowed for comparison between all three study groups. Finally, hierarchical multiple regression was carried out to investigate the relative impact of study variables on disability, while controlling for the effects of possible confounds.

The comparison group only completed two measures, those pertaining to catastrophising and cognitive distortion. The criteria for group membership specified absence of symptoms, therefore these participants could not meaningfully have completed any of the other measures. Medical evaluation and assessment of tender points were carried out for all but

two participants. However, data derived from the comparison group is only included in the analysis pertaining to the second hypothesis.

In relation to symptomatic participants, listwise deletion was used for the descriptive statistics for individual groups and for the independent *t* tests. This meant that all available data was used to describe the sample. However, casewise deletion was applied for the combined descriptive data for FMS and OOS groups and this approach was consistent during bivariate and multivariate analyses. In the results section, the number and nature of participants will be specified.

Chapter 6

Results

6.1 Outline and Data Management

The study findings will be outlined in relation to each hypothesis. Information about the management of missing data, non normality, and the presence of outliers is presented in Appendix C. The assumptions of multiple regression are also dealt with in Appendix C.

6.2 Hypothesis 1

That there will be significant differences between women with OOS and FMS in relation to pain, catastrophising, fear of movement, vigilance, and disability.

Table 1 shows the means and standard deviations for all study variables for FMS and OOS groups separately and in combination. The available means and standard deviations for the comparison group are also included in Table 1. Untransformed data for symptom duration is presented (in months), to make interpretation easier. However, transformed data was used in subsequent analyses (see Appendix C for details on this process).

Independent samples *t* tests indicated that there were no significant differences between individuals with FMS and OOS for any of the variables : current pain, t (35.6) = -1.48; p > .05; disability, t (65) = -.66; p > .05; vigilance, t (65) = .18; p > .05; fear of movement, t (65) = .29; p > .05; and catastrophising, t (66) = .60; p > .05. Levene's Test indicated that variances were unequal for current pain, F 5.58; p = .02, therefore separate variance assessments were used. Equal variances were assumed for the other variables. The separate OOS and FMS columns in Table 1 indicate the number of participants by variable in the analysis. There were no significant differences between any of the groups in the study in terms of age F (2, 89) = 1.03; p > .05.

Table1. Means and standard deviations for demographic, pain related, and cognitive variables together with outcome variable disability

Variables	FMS			OOS			Comparison Group			OOS & FMS Combined		
	n	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
Age	18	42.33	12.58	50	43.58	10.13	24	39.75	10.56	66	43.42	10.47
PCS	18	15.72	9.46	50	17.50	11.10	24	10.13	7.48	66	16.92	10.79
CEQ-R Total	18	45.50	14.04	50	40.36	14.85	24	34.33	10.13	66	41.80	14.93
Gen Cog Err	18	24.44	9.55	50	22.06	9.13	24	19.58	6.06	66	22.67	9.37
Som Cog Err	18	21.06	6.61	50	18.30	6.81	24	14.75	4.74	66	19.14	6.89
Tender Points	18	13.61	2.50	50	4.50	3.58	24	0.67	1.01	66	6.83	5.12
Symptom Dur Months	18	58.67	72.53	50	68.38	75.25	-	-	-	66	67.26	74.77
VAS	17	4.85	2.26	49	3.84	2.90	-	-	-	66	4.10	2.77
PDI	18	36.42	11.48	49	33.61	16.72	-	-	-	66	34.28	15.56
PVAQ	18	41.42	8.91	49	42.07	14.13	-	-	-	66	41.99	12.95
TSK	18	36.72	8.93	49	37.48	9.55	-	-	-	66	37.25	9.39

Note : The dashes in the cells indicate data not obtained for asymptomatic participants.

6.3 Hypothesis 2

That women with FMS will make more somatic cognitive errors than either women with OOS, or a female comparison group.

A series of one way ANOVAS was used to assess the difference between FMS, OOS, and comparison participants in general, somatic, and total cognitive errors. Cognitive errors were entered as the dependent variables, and diagnostic category as the independent variables. There were unequal numbers of participants in each group, but the assumption of equality of variance between groups was not violated.

Significant differences were found between groups in relation to somatic cognitive errors $F(2, 89) = 5.36$; $p = .006$, but not general cognitive errors $F(2, 89) = 1.69$; $p = .19$. Means and standard deviations and numbers of participants for total, general and somatic cognitive errors are reported in Table 1 for each of the three groups.

Post hoc analysis using Bonferroni adjustments established that the significant differences were between the asymptomatic group and FMS sufferers. In comparison with controls, people with FMS tended to make more somatic cognitive errors ($p = .006$), which was reflected in the significant differences between these groups in total cognitive errors derived by summation of the general and somatic scores ($p = .03$).

6.4 Hypothesis 3

That tender points will serve as an index of general symptomatic severity and in particular be associated with more pain and disability, fear of movement, catastrophising, and cognitive distortion. Additionally, as a function of these relationships, tender points will contribute significantly to prediction of disability.

Although tender points were assessed in the comparison group, only data from those with FMS and OOS were included in the analysis ($n = 66$). There was a statistically significant positive correlation between tender points and pain, a greater number of tender points being associated with increased pain reporting $r = .265$; $p = .016$. Also, increased numbers of tender points were significantly associated with a tendency to make more somatic cognitive errors. This relationship was also positive $r = .223$; $p = .036$

There were no significant relationships between tender points and either disability, fear of movement, or catastrophising (see Table 2).

Table 2. Simple Correlations among Tender Points (TPACR), Age, Pain (VAS), Disability (PDI), Vigilance, (PVAQ), Fear of Movement (TSK), Catastrophising (PCS), Total Cognitive Errors (CET), General Cognitive Errors (GCE), Somatic Cognitive Errors (SCE), and Symptom Duration (SD), for Symptomatic Participants, (N = 66).

	AGE	VAS	PDI	PVAQ	TSK	PCS	CET	GCE	SCE	SD
TPACR	02	27*	12	03	-01	02	16	09	22*	-08
AGE		17	26*	23*	16	15	20	14	24*	20
VAS			64***	44***	42***	29**	31**	25*	32**	08
PDI				59***	56***	35**	28*	22*	31**	03
PVAQ					47***	45***	33**	30**	30**	-00
TSK						49***	44***	39**	41***	-09
PCS							56***	43***	63***	06
CET								94***	89***	07
GCE									68***	05
SCE										09

Note : Leading decimal point omitted for clarity.
**p<.05; **p<.01; *** p<.001*

6.5 Hypothesis 4

That higher levels of catastrophising and greater pain intensity will be associated with increased fear of movement, and a tendency to catastrophise will also be linked to greater vigilance for pain related cues and more pain reporting.

It was anticipated that fear of movement/reinjury would affect individuals with FMS and OOS in a similar manner to sufferers with chronic low back pain. The mean score obtained from a measure of fear of movement/reinjury in the present study was comparable to published data from a mixed gender sample of chronic low back pain sufferers (Crombez, Vlaeyen, et al., 1999), and a female sample of FMS patients (Peters et al., 2000). Means and standard deviations of TSK scores are reported in Table 3. The data appears to be comparable with chronic low back pain obtaining the highest scores. However, inferences cannot be made on the basis of this data.

The bivariate correlations for the variables in this hypothesis are presented in Table 2. The direction and magnitude of these correlations confirmed expectations. However, it is noteworthy that pain and catastrophising were less strongly associated $r = .294$; $p = .008$ than catastrophising and the other cognitive variables.

The relationship between vigilance and catastrophising described above suggests that higher levels of catastrophising are associated with greater vigilance. A median split of catastrophising was carried out in the present study and an independent samples t test performed to test whether high and low catastrophisers differed significantly in relation to vigilance for pain related cues. Analysis of the data from symptomatic participants revealed significant differences between catastrophisers in terms of the extent of their vigilance for pain $t\ (65) = -2.88$; $p < .005$.

6.6 Hypothesis 5

That symptom duration will be positively associated with disability, pain, and vigilance, but negatively linked to catastrophising and fear of movement.

The duration of symptoms was not significantly associated with disability, pain, or vigilance. Similarly, as shown in Table 2, catastrophising and fear of pain were not associated with duration of symptoms of pain and discomfort.

Table 3. Comparison of the Means and Standard Deviations for Fear of Movement for the Present Sample and Two Published Studies.

	Fear of Movement	
	M	*SD*
Current study: OOS & FMS	37.3	9.4
Current study : FMS	36.7	8.9
Current study : OOS	37.5	9.5
Peters et al. (2000) FMS	34.2	10.7
Crombez et al. (1999) CLBP	39.3	6.4

6.7 Hypothesis 6

That higher levels of pain, catastrophising, fear of movement, and vigilance will all positively associated with disability and that the positive correlations between pain and disability, and between cognitions and disability, will be comparable. Additionally, that after controlling for the effects of age and duration of pain, pain-related fear, vigilance, and catastrophising will be as disabling as pain itself.

Bivariate correlations between pain, all the cognitive variables, and disability are amongst the correlations presented in Table 2. There were statistically significant positive correlations between disability and each of the following variables: pain, fear of movement, vigilance, and catastrophising. Higher scores in each case indicated higher levels of the attributes. The association between pain and disability is most noteworthy $r = .643$; $p < .001$. The magnitude was greater than that for the correlations between any of the cognitive variables and disability.

In order to test the second part of this hypothesis, a hierarchical multiple regression was performed. Disability was the dependent variable, with age and symptom duration entered at step 1, and the cognitive variables and pain at step 2. This strategy was used to evaluate the effects of proneness to catastrophising, vigilance for pain related cues, and fear of movement/reinjury, after controlling for age and symptom duration. Age was significantly associated with disability at the bivariate level which adds support to the notion that age may function as a confound. See Appendix C for information on the assumptions of multiple regression.

The effect of the combination of age and duration of symptoms on disability reporting

Table 4. Hierarchical Regression Analysis of the Impact of Pain, Vigilance, Catastrophising, Fear of Movement, and Pain x Catastrophising on Disability.

	Step 1		Step 2		Step 3	
Predictors	β	*t*	β	*t*	β	*t*
Age	0.26	2.09*	0.08	0.92	0.08	1.00
Symptom Duration	-0.12	-0.15	0.01	0.14	-0.03	-0.35
Vigilance			0.28	2.71**	0.31	3.21**
Fear of Movement			0.26	2.47*	0.16	1.62
Catastrophising			-0.04	-0.40	0.10	0.96
Pain			0.41	4.14***	0.42	4.70***
Catast x pain					-0.31	-3.60**
Adjusted R^2	0.04		0.54		0.62	
F	2.23		13.67***		15.91***	
R^2 Change			0.52		0.08	
***F* Change**			18.17***		12.87**	

**p<.05; **p<.01; ***p<.001*

was non significant. Adjusted R^2 and *F* statistics are presented in Table 4. However, the relative impact of the two variables entered in step 1 differed. Age alone did impact on disability, indicating that as individuals get older they are more likely to report increasing disability. This finding is statistically significant at the .05 level. Standardised regression coefficients are presented in Table 4.

Data from step 2 indicated that pain, catastrophising, vigilance, and fear of movement/ reinjury collectively influenced reporting of disability. Adjusted R^2 indicates that together these variables account for 54% of the variance in disability, although only 4% is explained by age and symptom duration. (See Table 4 for R^2 change and *F* change data).

In terms of the relative impact of pain and the cognitive variables on disability, the standardised regression coefficients presented in Table 4 show that pain, vigilance for pain related cues, and fear of movement/reinjury, but not catastrophising, contribute significantly to women's reporting of disability. Of these, pain makes the greatest contribution to increases in disability reporting. The standardised regression coefficients also show that addition of the other variables into the model means that age no longer impacts on disability. This may mean that the effects of age are mediated by its relationship with another variable, or variables.

As noted by Tabachnick and Fidell (1996), relationships between independent variables can make assessment of the relative importance of each difficult. While standardised regression coefficients reflect the relative impact of independent variables on the dependent variable, they are still influenced by the presence of other variables in the equation. Therefore results are presented with some caution in the knowledge that they are context dependent.

Comparisons of zero order and part correlations confirm the likelihood of shared variance between the independent variables. This is most relevant in relation to age and catastrophising. The semipartial correlation for age is $r = .078$, whereas the zero-order correlation is $r = .256$. The semipartial correlation for catastrophising is $r = -.034$, whereas the zero-order correlation is $r = .350$. Squared semipartial correlations indicate the proportion of variance in the dependent variable uniquely associated with a particular predictor (Jaccard, Turrisi, & Wan, 1990). Squared semipartial correlations in the present model provide data on the unique contributions for the three significant predictors of disability. As follows : vigilance (5.3%), fear of movement (4.4%), and pain (12%). Therefore, vigilance, fear of movement and catastrophising were not found to be as disabling as pain itself. An additional consideration when reviewing the data is that the order variables entered into the equation is likely to influence the relative impact statistics. For example, entering catastrophising after age and symptom duration, and before the other variables would render its impact significant (β .319; t, 2.72; $p = .008$). This again points to the possibility of some shared variance between the independent variables. However, in the present study there was no theoretical justifications for prior entry of either pain or any of the cognitive variables. Therefore, they were entered together after controlling for the effects of age and symptom duration.

In summary, results suggest that, after controlling for the effects of age and symptom duration, the following three variables impact on disability reported by women in the present sample: pain, vigilance, and fear of movement. The absence of an effect for catastrophising seems to be attributable to shared variance between catastrophising and one of the other independent variables. It seems most likely in relation to the model outlined earlier that catastrophising would moderate the relationship between pain and disability. Therefore, a post hoc hypothesis was formulated that catastrophising might function as a moderating variable.

To test this additional hypothesis, a cross product term was derived from pain and catastrophising deviation scores (Baron & Kenny, 1986). This was entered in at step 3, repeating the first two steps of the original multiple regression analysis. This allows for

assessment of the variance produced by the interaction term, after controlling for the main effects of the variables involved (Flett, Biggs, & Alpass, 1995).

Adjusted R^2, F values, and significance levels after step 3 of the analysis are reported in Table 4. Adjusted R^2 indicates that the new model including the cross product term accounts for 62% of the variance in disability, 8% being contributed by the interaction term. The post hoc hypothesis formed after the first regression, that catastrophising is moderating the relationship between pain and disability, is supported. Thus, the impact of catastrophising on disability varies at different levels of pain. It is also noteworthy that the inclusion of the interaction term means that fear of movement no longer exerts an independent impact on disability. It is possible that the relationship between fear of movement and disability may have been mediated by the pain catastrophising interaction.

Figure 3 provides a schematic representation of the pain and catastrophising interaction in prediction of disability. Following the literature (Flett et al., 1995) a median split of catastrophising and pain scores was carried out to illustrate the effects. As indicated earlier, the variables were treated as continuous in all the analyses. As shown in Figure 3, under conditions of high pain participants report similar levels of disability, regardless of whether they have high or low levels of catastrophising. However, under conditions of low pain participants with higher levels of catastrophising report significantly greater disability than those with lower levels of catastrophising.

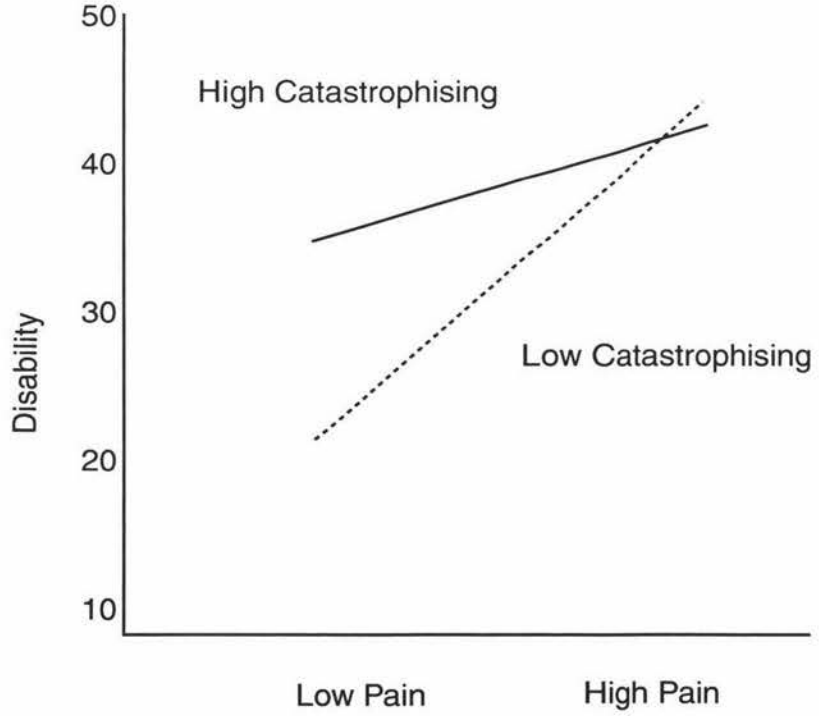

Figure 3. Schematic representation of the pain and catastrophising interaction in the prediction of disability.

Chapter 7

Discussion

7.1 Outline of Study Findings

In the first part of the discussion, findings will be considered in relation to individual hypotheses, or pairs of hypotheses. Then the overall findings will be considered in an attempt to integrate these with the relevant published literature.

7.1.1 Hypothesis 1

That there will be significant differences between women with OOS and FMS in relation to pain, catastrophising, fear of movement, vigilance, and disability.

There were no differences between women with FMS and OOS in levels of pain, catastrophising, fear of movement, vigilance, or disability. OOS and FMS share common features, but differ in relation to the distribution of pain and the presence of tender points. Additionally, the literature implies that while FMS is a more severe musculoskeletal disorder than OOS, they both exist on a continuum. For example, Turner (1994) proposed that, if untreated, localised pain can progress to FMS. Hypothesis 1 incorporated both ideas of location on a continuum and categorical separation in terms of severity. This may at first glance appear to be an oxymoron, but it is possible that OOS and FMS progress along a continuum which has a clear demarcation between the variants. Essentially this implies a clear progression of severity within and between disorders. The present study demonstrated that there were no differences between groups in relation to the parameters measured. This indicates that there is no clear cut off point between OOS and FMS. Therefore, it seems likely that the conditions vary together on a severity continuum. The data supports the suggestion of Wolfe (1997) that no precise demarcation exists between OOS and FMS. "... in a broader sense, there is no discrete point where fibromyalgia does or does not exist ..." (p. 270).

7.1.2 Hypothesis 2

That women with FMS will make more somatic cognitive errors than either women with OOS, or a female comparison group.

Women with FMS tended to make more somatic cognitive errors, but not more general cognitive errors, than those with no symptoms. There was a trend showing increasing somatic cognitive errors, from women presenting with no symptoms to those who had the most widespread, if not necessarily more intense pain. The presence of a trend is suggestive of continuum for somatic cognitive distortions, with women who have FMS located at one extreme. This might appear a contradiction of the commentary accompanying hypothesis 1. However, no significant differences were apparent between pain groups, which paralleled the findings on other variables. The tendency of FMS sufferers, rather than those with OOS, to make more somatic cognitive errors than did controls may be explained by the presence of more widespread pain, and a greater number of tender points, which might lead them to focus more on somatic concerns.

There is one caveat in relation to interpretation of findings for these first two hypotheses, which in a sense also supports the findings. Adherence to the ACR criteria meant that several participants who almost had the requisite number of tender points, and who satisfied the other criteria for FMS, were still classified as OOS not FMS. Therefore, there would have been some blurring at the category boundaries between OOS and FMS. This may have had a specific impact in relation to the first hypothesis, in that symptomatically similar women may have been placed in different groups. Unfortunately, this situation inevitably arises where there are distinct cut off points for a particular diagnostic label.

7.1.3 Hypothesis 3

That tender points will serve as an index of general symptomatic severity and in particular be associated with more pain and disability, fear of movement, catastrophising, and cognitive distortion. Additionally, as a function of these relationships, tender points will contribute significantly to prediction of disability.

Women with a greater number of tender points reported more pain and an increased tendency to make somatic cognitive errors. However, tender points were not related to fear of pain or disability reporting, or to a tendency to catastrophise. The absence of a relationship between tender points and disability indicates that the former are unlikely to be useful in the prediction of the latter. Therefore, further analysis was not carried out. This

hypothesis was formulated to test the suggestion of Wolfe (1997) that the tender point count might serve as a "sedimentation rate for distress" (p. 268). The erythrocyte sedimentation rate (ESR) is a blood test which is a non-specific measure of inflammation. Wolfe advocated looking more broadly at symptomatic presentation and also recognising distress, regardless of whether diagnostic criteria were satisfied. Data in the present study indicate that a greater number of tender points is linked to more reporting of pain and more somatic cognitive errors. This may reflect a greater somatic focus amongst those with more tender points. Thus, there is partial support for Wolfe's (1997) proposition that tender points have value as a marker for rheumatological distress. The absence of a relationship with most variables investigated argues against Wolfe's proposition. However, tender points may turn out to be selective markers for pain and somatic focus. It has been established in the present study that OOS and FMS do not differ in relation to either pain or somatic cognitive errors. This suggests that tender point count may index specific aspects of distress in both populations, again supporting the notion of Wolfe (1997) that there is no precise demarcation between FMS and other musculoskeletal pain disorders.

7.1.4 Hypothesis 4

That higher levels of catastrophising and greater pain intensity will be associated with increased fear of movement, and a tendency to catastrophise will also be linked to greater vigilance for pain related cues and more pain reporting.

Fear of pain, and more specifically the notion of fear of movement or reinjury, has been investigated quite thoroughly in research with chronic low back pain patients. Review of the published literature did not yield any information on the relevance of this aspect of the fear avoidance model for individuals with FMS or OOS. It is only in recent months that data has been published which incorporates information on the construct of fear of movement measured by the TSK amongst FMS sufferers (Peters et al., 2000).

Preliminary assessment of fear of movement amongst individuals with OOS and FMS involved comparison of the present findings with published data from a sample with back pain, and one with FMS. Mean scores for fear of movement were higher amongst a mixed gender back pain group, than two female FMS groups. However, reported pain levels were higher in the back pain group than either of the FMS groups. OOS participants had greater fear of pain than the FMS groups, and lower reported pain than any of the groups. However, no statistical comparison was made between the results from the present study and those in the published literature, therefore the above is merely a discussion of trends. Additionally, statistical analysis carried out to test hypothesis 1 in the present study did

not reveal any significant differences between those with FMS and OOS in relation to either fear of pain, or pain intensity.

Fear of movement was associated in the present study with both a greater tendency to catastrophise and with more intense pain. The link between fear of movement and catastrophising was more noteworthy than between either pain and fear of movement or pain and catastrophising in line with the data reported by Vlaeyen, Kole-Snijders, Boeren, et al. (1995).

Vlaeyen and Linton (2000) modified the earlier model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995), by highlighting the role of vigilance in association with avoidance on the pathway between fear and disability. The data presented in this thesis implicates vigilance as a factor in disability reporting. Vigilance may follow from a catastrophic style of thinking and impact on the development of disability. The data demonstrated that catastrophising was linked to pain, pain-related fear, and greater vigilance for pain related cues. More specifically, high levels of catastrophising were more closely associated with higher levels of vigilance than were low levels of catastrophising. This parallels the findings of Crombez et al. (1998a), although a different population and methodology were used. It is important also to note that the correlational nature of the data means that symmetry exists in the relationships between all of these variables. Therefore, while perceptions of pain of increasing intensity may lead to a tendency to catastrophise more, the reverse may also be true.

Findings pertinent to fourth hypothesis will be considered further in the discussion of hypothesis 6.

7.1.5 Hypothesis 5

That symptom duration will be positively associated with disability, pain, and vigilance, but negatively linked to catastrophising and fear of movement.

Symptom duration was not related to pain, disability, vigilance, catastrophising, or fear of movement. It had been anticipated that in line with the literature, greater chronicity would be linked to more disability reporting, but that fearful beliefs about pain would be most prevalent early in the course of the pain condition. Several authors (e.g., Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995) have noted a negative association between fear of pain and pain duration, indicating that fear reduces over time, perhaps as a result of extinction. The present study failed to replicate either of the relationships reported in the literature.

The absence of a link between either perceptions of pain or beliefs about pain and symptom duration perhaps indicates that in this population other factors apart from chronicity influence perceptions and beliefs.

7.1.6 Hypothesis 6

That higher levels of pain, catastrophising, fear of movement, and vigilance will all be positively associated with disability, and that the positive correlations between pain and disability, and between cognitions and disability, will be comparable. Additionally, that after controlling for the effects of age and duration of pain, pain-related fear, vigilance, and catastrophising will be as disabling as pain itself.

The significant relationships found in the present study indicate that pain, proneness to catastrophise, and a tendency to be fearful about movement and to be vigilant for pain are all associated with increased disability reporting. Amongst women with FMS and OOS, pain was most closely associated with disability reporting. None of the other significant correlations were of the same magnitude. Thus, there was only partial support for the first part of this hypothesis.

Bivariate data relevant to this hypothesis and to hypothesis 4 can be related to the model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995). However, there is the proviso that in the subsequent discussion it is recognised that neither study incorporates methodology to evaluate causation. In this population of female chronic pain patients, pain and the cognitions investigated are to varying extents linked with disability. The present findings in relation to the first part of hypothesis 6 have some common ground with those of Vlaeyen and colleagues. There are, for example, associations between fear of movement and catastrophising, and fear of movement and pain, with fear and catastrophising more closely associated with each other than either of the constructs are with pain. However, data from the present study diverges from that of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) and Crombez, Vlaeyen, et al. (1999) in relation to the links between cognitions, pain, and disability. Preliminary analysis of bivariate data does not point to pain-related fear being more disabling than pain itself as suggested by Crombez, Vlaeyen, et al. (1999), or even as disabling as pain itself, as hypothesised in the present study. Instead, it appears that pain has the major role. Data from the present study might be incorporated into the model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) in the following way. Increasing levels of pain activate what may be an existing tendency to catastrophise. In the presence of higher levels of catastrophising, a woman with FMS or OOS may be more fearful about pain and what it means in terms of harm to the body. According to the work of Vlaeyen and col-

leagues fear of movement is closely linked to avoidance. Indeed, avoidance may follow from fear, although causality has not been demonstrated.

In relation to the second part of this hypothesis, the decision to consider age and duration of symptoms as potential confounds was partially supported by the analysis between pairs of variables. Specifically, age was significantly associated with disability and with somatic cognitive errors, although the latter was not included in the regression analysis. This indicates that with increasing age women are more likely to report disability, to be more vigilant for pain cues, and perhaps to have negative ideas about their physical capabilities.

The results demonstrated that the combination of age and duration of symptoms did not impact on disability, but age alone did. This may mean that the effects of age on disability may be mediated by other variables, as its impact vanished with the introduction of pain, vigilance, and fear. After controlling for the effects of age and duration of symptoms, only vigilance for pain cues, fear of movement, and pain uniquely contributed to disability reporting. An additional finding was that catastrophising moderated the relationship between pain and disability.

The interaction between pain and catastrophising possibly explains the significant bivariate association between catastrophising and disability, while at the multivariate level catastrophising failed to exert any individual influence on the solution. It appears that when women with musculoskeletal disorders experience high levels of pain, similar levels of disability are reported regardless of whether they tend to catastrophise or not. However, amongst women reporting low levels of pain those with high levels of catastrophising report greater disability than those who catastrophise less. Additionally, fear of movement no longer exerts an independent impact on disability, perhaps indicating that the relationship between fear of movement and disability may be mediated by the association between pain and catastrophising. However, given the correlational nature of the data the interaction effect could also be interpreted as pain moderating the effects of catastrophising. Essentially, the second part of the hypothesis was not supported because pain contributed most of the variance in disability reporting.

7.2 Towards an Integrated Account

In this section, the aims of the study will be revisited, and findings integrated and discussed in relation to three broad categories : Between group differences, the relevance of fear of movement for FMS and OOS, and finally an exploration of the pain disability relationship considering the intervening effects of cognitive variables.

7.2.1 Between group differences

One of the main aims of the present study was to evaluate whether FMS and OOS can be considered as discreet entities in terms of pain experience and the prevalence of particular pain related thoughts. Additionally, one comparison was undertaken which utilised the asymptomatic group. There does not appear to be published data comparing OOS and FMS although, as alluded to elsewhere in this thesis, there has been commentary suggesting that the two musculoskeletal pain conditions are related. The absence of differences between OOS and FMS in the present study indicates that they are similar, at least in relation to the parameters investigated.

The data from this study raised further questions about the already controversial issue of diagnosis, and the role that tender points have in the process. (Some debate from the literature was included in Chapter 3). In particular, questions arise from the present data about the specificity of tenderness and how it relates to widespread pain. Notably, tenderness in the cervical area was elicited in several participants in the comparison group, whose defining characteristic was lack of symptoms of FMS and OOS. Additionally, several participants had a subthreshold number of tender points with widespread pain, while one participant in the OOS group had the requisite number of tender points but highly localised pain. Therefore, while it would be anticipated that individuals with FMS have more tender points than other rheumatological conditions, as tender points characterise the disorder, numerous tender points may be present in other musculoskeletal disorders. Additionally, tender points were shown to be linked to pain, but in the present study, OOS and FMS participants did not differ in terms of pain. Consequently it seems most likely that pain and tender points are features of the experience of both groups, while extent of pain distinguishes them. The widespread nature of pain may be the key to greater endorsement of somatic cognitive errors in the FMS group. Two points can reasonably be taken from this data. Firstly, that some tender point sites (e.g., the cervical) may not discriminate well between symptomatic and asymptomatic individuals for the purposes of rheumatological assessment. Secondly, for most variables investigated in the present study, it seems that individual variability in symptom severity may be more marked than differences between groups.

As a post script to this subsection a quotation is included from an elegant essay written by Patrick Wall (1993) about the mechanisms involved in FMS. "I suggest only that central sensory mechanisms beginning in the spinal cord possess and create an Achilles heel in their search for an optimal state of sensitivity. That Achilles heel could be the tender point" (p. 59). If the tender point is implicated in the processes of central sensitisation then

it is not surprising that they are present in other musculoskeletal chronic pain syndromes. It may be that a re-evaluation of tender points is required, in particular looking at the usefulness of different sites. Additionally, consideration of the tender point as a non-specific marker of musculoskeletal disorder, rather than a key component of FMS, may be more appropriate.

7.2.2 The relevance of fear of movement for OOS and FMS

The positive association between fear of movement and disability provided some preliminary validation of the relevance of the construct in this population. Additionally, fear of movement was a significant predictor of disability prior to the introduction of the pain catastrophising interaction term. In the final stage of the analysis, the unique impact of fear of movement was rendered non-significant when catastrophising moderated the effects of pain on disability. Thus, it is possible that the relationship between fear of movement and disability is mediated by the association between pain and catastrophising. Findings from the present study diverge from those in the extant research involving chronic low back pain populations. Present findings do not support the notion that pain-related fear is more disabling than pain itself. The main effect of pain contributed most to disability, followed by the contributions of vigilance for pain, and being fearful of movement or reinjury, although the impact of the latter was later lost.

It was noteworthy that beliefs about pain such as fear of movement were not related to duration of symptoms in this population. However, factors contributing to fear of movement associated with symptom onset and the process of diagnosis were not explored in the present study. It is possible they may have improved understanding of the relationship between pain-related fear, symptom duration, and disability. For instance Crombez, Vlaeyen, et al. (1999) evaluated the contribution of sudden versus gradual onset of pain to fear of movement, finding greater fear amongst those whose pain started suddenly. The process of diagnosis may also influence beliefs about pain and adaptation to chronic pain, and be entangled in some way with duration of symptoms. Waddell et al. (1993) noted diagnostic uncertainty, rather than the severity of the pain problem, contributed most to fear and subsequent avoidance. Similarly, Geisser and Roth (1998) reported that participants who disagreed with the diagnosis attributed to their pain were more likely to consider that pain was indicative of harm. These same participants also tended to catastrophise more, and to report greater disability.

Essentially, rather than beliefs such as fear of reinjury altering with time, it may be that factors relating to the onset of pain, and the client's knowledge and understanding of their

condition are more relevant to different phases of adjustment. Additionally, the correlational relationships between beliefs, focus on pain, and thoughts may involve more reciprocal influence than estimated in the current model. In the next section some discussion of the overlap between variables will be included, a topic which will be returned to later in the chapter.

7.2.3 Exploration of the pain disability relationship and the role of cognitions

In this subsection an attempt will be made to integrate the present findings into a model drawing on the ideas of McDermid et al. (1996), Rollman and Lautenbacher (1993), and the evolving models of Vlaeyen, Kole-Snijders, Boeren, et al. (1995), and Vlaeyen and Linton (2000). Vigilance or a tendency to focus on pain will be considered first.

Vigilance for pain was shown to be a significant predictor of disability in the present study. This may be related to data on hypervigilance in FMS outlined in Chapter 3. McDermid et al. (1996) noted an increase in symptom reporting amongst participants with FMS. Extrapolating from the findings of both projects, it is possible that generally amongst women with musculoskeletal pain syndromes as pain increases there is a tendency to become more focused on pain or vigilant for pain cues, which might be linked to greater symptom reporting. Alternatively, particular characteristics of the condition may predispose to greater vigilance. For example, as hypothesised earlier, more widespread pain may be the key factor in development of somatic cognitive distortion and more pain reporting. Developing on this theme, Rollman and Lautenbacher (1993) proposed that the vigilant 'response style" manifested in FMS may be attributable to a failure of what they describe as "cognitive filtering mechanisms" (p. 156). Combining these two perspectives it is possible that greater symptom reporting reflects heightened somatic awareness, when normal cognitive coping responses are not activated. However, as discussed in Chapter 3, physiological and perceptual processes are inextricably linked. Thus, as pain intensity increases there will be interplay between physiology, perception, and cognitions contributing to the overall experience of pain. These ideas would cohere with the model of Vlaeyen, Kole-Snijders, Boeren, et al. (1995) which positions catastrophising at the divergence of pathways to recovery or disability. The failure of cognitive filtering described above may be manifested as activation of catastrophising and foregrounding of particular pain related fears with a greater awareness or focus on pain. These tentative explanations may also corroborate the suggestions in the literature (e.g., Boston et al., 1990; Flor et al., 1993; Turk & Rudy, 1992) that fewer negative, rather than more positive, thoughts are most likely to influence outcome for individuals with chronic pain. Unfortunately, some of the

data from the present study cannot be readily accommodated into the outline above. In particular, findings in this study provide a different emphasis in relation to pain and catastrophising. Intense pain appears to follow a pathway to disability, irrespective of the presence or absence of a tendency to catastrophise, although in conditions of low pain a greater predisposition to catastrophise also leads to more disability reporting. It is possible that when pain is intense catastrophising becomes irrelevant because of the salience of pain related cues. Alternatively, drawing on the work of Eccleston et al. (1997) and Crombez, Eccleston, et al. (1999) referred to earlier another hypothetical pathway may involve the interaction of pain with either somatic awareness or fear. More severe pain in the presence of fear, may disrupt the ability of the individual to attend to stimuli other than those relating to pain. Vigilance or an intense focus on pain may also predispose the pain sufferer to avoid situations or activities which might be perceived to exacerbate the experience of pain. These ideas warrant further exploration. Additionally, research is also needed to establish whether the pain catastrophising association mediates the impact of fear of movement on disability, and if so under what circumstances.

In essence the data can still be construed as supporting the suggestions in the literature (e.g., Moss-Morris & Petrie, 1997) that when there is a discrepancy between pain and disability, cognitive variables can help account for the mismatch. Data from this study involving participants with FMS and OOS does, however, provide a different emphasis to the recently reported results involving CLBP sufferers.

7.3 Limitations of the Present Study

The design of the present study limits the conclusions that can be drawn. Two points will be briefly made which relate to sample size and selection, and methodology.

The size of the sample is important in relation to statistical power, or the certainty that relationships can be detected at a given significance level (Hair, Anderson, Tatham, & Black, 1995). In the present study the modest sample size limits the power levels for small effect sizes. It was anticipated that the effect sizes for the main effects would be large. However, the sample size made further analysis of interaction effects of doubtful value. If these effects were present they would probably be small.

The convenience sampling was a function of the relatively small potential pool of participants and the nature of the experimental assessments not reported in this thesis. Many of the participants were from the practice of the medical practitioner involved with the study. The medical practitioner specialises in occupationally related musculoskeletal disorders,

which may have biased the sample. The female sample is another feature which limits the generalisability of results. The decision only to use women was based on the epidemiology of the musculoskeletal conditions. Therefore, the nature of the sample and its procurement may limit generalisability of findings to other individuals with musculoskeletal disorders. However, a strength of this sample was that it incorporated individuals with chronic pain at various stages in the process of adaptation, not just those referred from a pain clinic. This feature may mean that it is more representative of individuals living with pain on a daily basis, who are not currently involved in rehabilitation programmes.

Finally, in relation to the procuring information, a cross-sectional non-experimental design means that firm conclusions cannot be made about causation. However, as Jensen et al. (1994) indicated, correlational designs cannot prove causation, but they can rule it out. If there is no relationship between variables it is not likely that one will cause the other. Most of the data was derived from questionnaires and was largely 'on the day' information. However, one questionnaire asked participants to consider items in the last few weeks, while another asked for information "when you are in pain". The latter was completed by all participants. It would require those without symptoms to remember a recent painful experience, without specifying the nature of pain. Therefore, there would be no guarantee that similar pains were being recalled, or that the experience would be accurately recollected. Loftus (1980) outlined some of the evidence for alteration of memory over time. There was recognition in the planning and execution of this project that alternative methods for obtaining information using a prospective design would have been better, but the time constraint of one year, together with the need to fulfil the requirements of a larger study which was already quite demanding for participants, precluded these options.

7.4 Issues of Inclusion

Arguably the present study should have looked at mood as there is evidence in the literature that mood variables are factors involved in chronic pain, some of which has been referred to where relevant. However, as already alluded to, assessing mood can be problematic because of overlap between somatic symptoms of depression and those of chronic pain. Gamsa (1994) advocated caution in the use of tests in this population because endorsement of somatic and contextual items may reflect physical symptoms of chronic pain and the limitations it imposes on function. The decision not to include mood variables was a pragmatic one. It was decided that the evaluation of cognitive variables alone, not as a symptom of another problem, was a worthwhile enterprise. If fear of pain is a feature of chronic pain it is important to deal with the fear rather than to look for another label for it.

It might also have been useful to draw in factors such as compensation status, in view of the recent debates surrounding ACC involvement in musculoskeletal disorders that may be occupationally derived. This may be a topic for future research.

7.5 Measurement and Conceptual Overlap

Gamsa (1994) pointed out the need to strive to improve methodology, whilst also being cognizant of the issues faced by those undertaking psychological research. In particular, Gamsa alluded to the difficulties posed by the complexity of the variables being evaluated " ...we cannot isolate and control the variables: they can't be separated and they won't stand still" (Gamsa, 1994, p. 25). The issue of interacting variables is pertinent for the present study in relation to the overlap between constructs of fear of pain, catastrophising, vigilance, and disability. A key question is how to separate theoretical associations, or the interplay between variables, from construct validity issues, or from other measurement issues such as relationships attributable to shared method variance. The first two of these will be discussed in more detail in the section which follows.

Associations between variables in the present study can be incorporated in terms of the theoretical models outlined, but other aspects of the relationships need to be considered too. Vigilance, fear of movement, and pain made individual contributions to the prediction of disability in the initial analysis, but this was relatively small compared with the overall explained variance in the model, suggesting some overlap between them. Vigilance, catastrophising, and fear of movement may be similar constructs. Alternatively, there may be other interrelationships not explored in the present study. For example, there is a possibility that the relationship between fear of movement and disability is mediated by the pain catastrophising interaction. A criticism that might be levelled at the present study is that it would be expected for someone with high levels of catastrophising to obtain a high score across all measures, particularly as the psychological variables rely on self report. Thus a participant prone to catastrophising would also indicate high levels of pain. However, this does not fit with the discovery of the pain catastrophising interaction.

The validity of measurement is another consideration. Murphy and Davidshofer (1994) proposed that it is important to look at content and construct validity to assess whether a test measures what it sets out to measure. Content validity looks at the domains covered by the items in the test, while construct validity is more concerned with comparisons between the test and other measures (Murphy & Davidshofer, 1994). This issue will be briefly discussed in relation to the instruments measuring cognitions used in the present study. Firstly, in relation to item overlap, there are some similar items on the PVAQ and

the PCS. Item 13 on the PVAQ, "I pay close attention to my pain", and item 15, "I become preoccupied with pain" are similar to each other, and to two items in the PCS, item nine, "I can't seem to keep it out of my mind" and item ten, "I keep thinking about how much it hurts". Additionally, items in the TSK may cover several domains, not just those relating to the danger of pain. For instance, item 12, "Pain lets me know when to stop exercising so that I don't injure myself" and item 15, "I can't do all the things normal people do because it's too easy for me to get injured" may reflect the reality of pain for that person. That is, they do not necessarily reflect a fear based pattern of responding. It does not appear that the psychometric properties of some of the measures used in the present study have been widely investigated.

7.5.1 Objectivity of measurement processes

Concerns have been expressed in the literature about objectivity in measurement particularly in relation to pain and disability. As outlined in earlier sections of this thesis, objectivity becomes problematic in pain research as pain is, by definition, whatever the individual says it is. There has also been some discussion (e.g., Flor & Turk, 1988, p. 263) about the need for more "objective" measures of disability. As discussed earlier in the present chapter, this would not be an easy task.

7.6 Implications for Treatment Programmes

Jensen et al. (1991) noted that chronic pain does not inevitably equate with depression and disability, and that it is important to isolate factors which improve function for those in pain. Greater understanding of pain can help with the design and targeting of interventions. To date there have been some mixed research findings. Nielson, Walker, and McCain (1992) provided some evidence that Cognitive Behavioural Therapy (CBT) may be useful in the treatment of FMS. In contrast, Vlaeyen et al. (1996) reported little overall improvement in a trial incorporating components of group education with cognitive treatment, group education with discussion, and a wait list condition. Additionally, those who experienced the discussion component did better than the participants who were allocated to the cognitive educational condition. Participants from the discussion with group education condition indicated improved pain control and coping, but the expected increments in knowledge did not occur. It was also surprising that those in the group education and cognitive treatment condition did not show improvements in pain coping and relaxation. Vlaeyen et al. (1996) provided a number of possible explanations for their results including the benefit of support in the condition incorporating group discussion. However, they do not appear to account for the overall lack of improvement. In a multimodal programme

for FMS incorporating CBT for tackling cognitive distortion, Mason, Goolkasian, and McCain (1996) found that although initial gains were made, these were not maintained at follow up and they concluded that relapse prevention needs to be addressed. The study of Mason et al. (1996) was interesting as improvement in function was not matched by a decline in symptoms. They speculated that failure to practice what had been learned during treatment may have accounted for the failure to maintain gains, but adherence to the programme after treatment was not measured in the study. The work of Vlaeyen et al. (in press) alluded to earlier, although only single case design, appears to offer some hope as a therapeutic intervention for chronic pain using exposure. The results were that exposure led to reduced catastrophising, disability, and pain related fear while pain control increased. It remains to be seen whether gains are maintained. Further research is needed to determine the key components of interventions.

Nicassio et al. (1995) suggested that the presence of interactions between pain, other features of the chronic pain condition, and coping might lead to different pain outcomes and that this requires further research amongst populations with FMS. Thus it seems that expanding knowledge of key cognitive variables and possible interactions between variables that influence outcome in chronic pain populations is important for the development of effective treatment programmes. Therefore, the current findings may prove useful clinically, by evaluating the role of cognitions amongst chronic pain sufferers, other than those with CLBP, and by investigating relationships between different cognitive constructs, which may impact on individual function. The potential offered by increasing knowledge is shown by the research of Turner et al. (2000), who demonstrated that catastrophising and beliefs exerted independent effects on outcome and, as they suggest treatment targeting only one of these, might have limited efficacy.

7.7 Further Research

More research is needed to investigate the content and source of pain-related fear (Crombez, Vlaeyen, et al., 1999; Vlaeyen & Linton, 2000). A key question raised by Crombez, Vlaeyen, et al. (1999) was "..what exactly do patients with pain-related fear, fear?" (p. 336). As Crombez, Vlaeyen, et al. (1999) have suggested, it may be that chronic pain patients fear longer term rather than immediate consequences of engagement of activities. It is arguable that this is anticipation, rather than fear, and based on prior experiences. Alternative methodologies may be a more appropriate means of answering some of these questions, increasing the scope of existing research by providing a greater breadth of data in relation to pain related fears and beliefs. Large and Strong (1997) suggested that a focus on

idiographic rather than nomothetic research may expand understanding of some of the adjustment processes in chronic pain. Strong and Large (1999) also provided a different and more positive framing of somatic focus, which also merits more investigation. They commented that a focus on the somatic can allow chronic pain participants to plan their lives, which helps rather than hinders adaptation.

Keefe and France (1999) discussed the need to investigate how components of the biopsychosocial model interact, rather than looking at each component alone. Also they recommended investigating how responses to pain change over time and how plasticity in the CNS interfaces with psychological change. As Keefe et al. (1992) suggested, causal modelling would be useful in this area of research to establish direction of effects. The family of techniques described as structural equation modelling also allow for combination of exploratory and confirmatory analyses, but large samples are required (Kline, 1998).

7.8 Conclusions

The findings from the present study demonstrate that there is no clear demarcation between OOS and FMS in terms of severity of pain, beliefs about pain, or cognitions. Widespread pain, or pain extent, but not severity, may be a key feature accounting for the differences between FMS sufferers and the comparison group in terms of somatic cognitive distortions. Data presented in this thesis indicate that the tender point may be more useful as a non-specific marker of musculoskeletal disorder, rather than a key component of FMS diagnosis.

In the present study, pain made the largest independent contribution to perceived disability. This result contrasts with recent published data indicating that fear of pain is more important than pain itself in the development of disability. The finding of an interaction effect involving pain and catastrophising, and the possibility that the impact of fear of movement on disability was mediated by this effect, are indicators of the complexity and interdependence of the chronic pain experience highlighted in the recent literature.

More research is needed to evaluate the content domain of some of the constructs commonly referred to in the literature. For example it would be useful to elaborate the threat accompanying pain alluded to by Price (1999). Associated with this idea, the key question awaiting a more clear-cut answer is: What is it that chronic pain patients fear most? If as Philips and Jahanshahi (1985a) suggested avoidance is not mediated by fear of pain, alternative pathways need to be explored as avoidance may be most directly relevant to outcome. However, while recognising the consequences of disuse which may arise from

systematic avoidance in chronic pain, it is also possible that avoidance of specific situations known to seriously exacerbate pain may be a necessary part of successful adaptation. It does not follow that avoidance of all activities flows from the avoidance of a few. Additionally, it would be useful to find an innovative way of capturing all dimensions of vigilance, or attention to pain which, as noted by Strong and Large (1999), may also feature positively in adaptation to pain. In summary it appears that qualitative methodologies may be more appropriate to explore the meanings associated with chronic pain. As Melzack and Wall (1988) indicated the experience of pain is determined by the whole person, including their thoughts, hopes, and fears. Further investigation at the level of the individual may improve our understanding of this phenomenon.

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Appendices

Appendix A

Fibromyalgia and Occupational Overuse Syndrome Study Information Sheet

We would like to invite you to participate in an important study to confirm the usefulness of a blood test to diagnose fibromyalgia (FMS) and occupational overuse syndrome (OOS). We will also be carrying out a test of vibration sensation. In this study we will be comparing those who have problems associated with FMS and OOS with those who do not. It would be helpful if you could bring a friend of the same gender and similar age unaffected by FMS or OOS to allow us to compare data. We hope at the end of this study to be able to offer laboratory tests for FMS and OOS to help with diagnosis.

A routine blood sample will be taken and tested for hyaluronic acid, which is a substance present in connective tissues in the human body. This involves minor discomfort and no risk. A recent study carried out in Israel showed that the level of hyaluronic acid was raised in people with FMS.

We will also be testing your sensation of vibration in the hands. Usually there is no discomfort associated with this test. If there is any it will be momentary. There have been reports from University College London that this procedure is a sensitive test for overuse syndrome associated with keyboard use. If we confirm these reports vibration sense may be another laboratory test useful for diagnosing OOS and FMS.

Confirming the value of these simple laboratory tests would make diagnosis of conditions such as FMS and OOS quicker and easier. This will help with treatment and research looking into causes, eventually perhaps leading to better understanding of how to prevent fibromyalgia and OOS. Our aim is to increase the knowledge base about fibromyalgia and OOS. Though these tests may not provide definite proof of presence or absence of these

problems, we hope they will be of value in diagnosis and control of OOS and FMS.

In addition to the blood test and vibration sense assessment you will be asked to undergo a brief examination of arms, neck and shoulders and complete several short questionnaires. The questionnaires will focus on individuals thoughts about pain. We would like you not to take pain killing tablets or engage in vigorous exercise for 24 hours prior to the testing.

If you decide to take part in this study your help will be appreciated by the research team and our results may generally benefit those suffering with fibromyalgia and OOS. Once enrolled you may decide that you do not want to continue with the study. You may leave the study at any time without this influencing your medical care. Similarly if you decide you do not want to take part this will not alter the treatment you receive. Your decision will be respected.

Any information you give us will be treated as confidential. A code number, rather than a name and address will be used to identify those participating. This code will be known only by the clinician responsible for your care and the research assistant involved with the project.

The blood sample you provide will be stored and Ethical committee approval sought for future use or testing. This project has been approved by the Manawatu-Wanganui Ethics committee. This means that the Ethics committee may check that the study is running smoothly and that appropriate ethical procedures are followed.

For further information about the study you can contact Dr Wigley on 357 8429, Dr Gear via the Palmerston North hospital switchboard 356 9169, or Dr Jane Grant at the rehabilitation unit at the hospital. Also, Malcolm Johnson, (Clinical Psychologist Massey University), or Liz Chambers (Research Assistant Massey University) available on 350 5196.

Participant code ❑❑❑

Consent Form

I consent to the researchers storing a specimen of my blood for one year for use as part of this study. Yes/No

I wish to receive a copy of the results of this study. Yes/No

I wish to have study results sent to my doctor. Yes/No

If yes, my doctor's name is .....

I understand that this study has received ethical approval from the Manawatu-Wanganui Ethics Committee. If I have any queries regarding ethical issues and this study, I may contact the committee on tel/fax 356 7773.

I fully understand the explanation I have been given about this study and have had the opportunity to have my questions answered.

I agree to participate in this study.

Signature

Printed Name

Date

Witnessed by :

Signature

Printed Name

Date

Can you Help?

We are looking for volunteers to form a control group in a study carried out in the School of Psychology involving participants with Fibromyalgia and Occupational Overuse.

We hope that this study will validate some simple assessment procedures to distinguish between those who have occupational overuse, and fibromyalgia, and individuals who do not have either condition.

If you are female between 20 and 50 years of age we would like to hear from you.

Ideally we are looking for women who use a keyboard frequently in the course of their work and some who use a keyboard occasionally.

Participation would take approximately an hour of your time. You would be asked to complete some questionnaires and there would also be a brief test, a medical examination, and a blood test.

If you would like more information, do get in touch with me. There is no obligation to take part.

You can contact me for further information either via email : liz.chambers@clear.net.nz, or by leaving a message at the School of Psychology, Massey University, tel no : 350 5799 ext 2040 to speak to somebody, or ext 7098 to leave a message on the answer phone.

Appendix B

Participant code ❑❑❑

Questionnaires About Your Pain And Associated Thoughts

Participant code ❑❑❑

QUESTIONNAIRE ONE

DESCRIBE YOUR PAIN BY CHECKING THE APPROPRIATE SPACES

	NONE	MILD	MODERATE	SEVERE
THROBBING	0)_____	1_____)	2)_____	3)_____
SHOOTING	0)_____	1)_____	2)_____	3)_____
STABBING	0)_____	1)_____	2)_____	3)_____
SHARP	0)_____	1)_____	2)_____	3)_____
CRAMPING	0)_____	1)_____	2)_____	3)_____
GNAWING	0)_____	1)_____	2)_____	3)_____
HOT-BURNING	0)_____	1)_____	2)_____	3)_____
ACHING	0)_____	1)_____	2)_____	3)_____
HEAVY	0)_____	1)_____	2)_____	3)_____
TENDER	0)_____	1)_____	2)_____	3)_____
SPLITTING	0)_____	1)_____	2)_____	3)_____
TIRING-EXHAUSTING	0)_____	1)_____	2)_____	3)_____
SICKENING	0)_____	1)_____	2)_____	3)_____
FEARFUL	0)_____	1)_____	2)_____	3)_____
PUNISHING-CRUEL	0)_____	1)_____	2)_____	3)_____

PLACE AN "X" ON THE LINE TO INDICATE YOUR CURRENT LEVEL OF PAIN

NO PAIN _____ WORST POSSIBLE PAIN

WHICH WORD BEST DESCRIBES THE PAIN YOU ARE PRESENTLY FEELING?

PPI

0 NO PAIN _____
1 MILD _____
2 DISCOMFORTING _____
3 DISTRESSING _____
4 HORRIBLE _____
5 EXCRUCIATING _____

Participant code ❑❑❑

QUESTIONNAIRE TWO

The rating scales below are designed to measure the degree to which several aspects of your life are presently disrupted by chronic pain. In other words, we would like to know how much your pain is preventing you from doing what you would normally do, or from doing it as well as you normally would. Respond to each category by indicating the overall impact of pain in your life, not just when the pain is at its worst.

For each of the 7 categories of life activity listed, please circle the number on the scale which 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 which you would normally be involved have been totally disrupted or prevented by your pain.

1. Family/ home responsibilities

This category refers to activities related to the home or family. It includes chores or duties performed around the house (e.g gardening) and errands or favours for other family members (e.g driving the children to school).

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

2. Recreation

This category includes hobbies, sports, and other similar leisure time activities.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

3. Social activity

This category refers to activities which involve participation with friends and acquaintances other than family members. It includes parties, theatre, concerts, dining out, and other social functions.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

4. Occupation

This category refers to activities that are a part of or directly related to one's job. This includes non-paying jobs as well, such as household duties or volunteer work.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

Participant code ❑❑❑

5. Sexual behaviour

This category refers to the frequency and quality of one's sex life.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

6. Self-care

This category includes activities which involve personal maintenance and independent daily living (e.g taking a shower, driving, getting dressed etc).

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

7. Life-support activity

This category refers to basic life-supporting behaviours such as eating, sleeping, and breathing.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

Participant code ❑❑❑

QUESTIONNAIRE THREE

When answering this questionnaire, please think back over the last two weeks and indicate on a scale from 0 (never) to 5 (always), how each item was a true description of your behaviours in relation to pain.

1. I am very sensitive to pain

0 1 2 3 4 5
never always

2. I am aware of sudden or temporary changes in pain

0 1 2 3 4 5
never always

3. I am quick to notice changes in pain intensity

0 1 2 3 4 5
never always

4. I am quick to notice effects of medication on pain

0 1 2 3 4 5
never always

5. I am quick to notice changes in location or extent of pain

0 1 2 3 4 5
never always

6. I focus on sensations of pain

0 1 2 3 4 5
never always

7. I notice pain even if I am busy with another activity

0 1 2 3 4 5
never always

8. I find it easy to ignore pain

0 1 2 3 4 5
never always

Participant code ❑❑❑

9. I know immediately when pain starts or increases

0 1 2 3 4 5

never always

10. When I do something that increases pain, the first thing I do is check to see how much the pain was increased

0 1 2 3 4 5

never always

11. I know immediately when pain decreases

0 1 2 3 4 5

never always

12. I seem to be more conscious of pain than others

0 1 2 3 4 5

never always

13. I pay close attention to pain

0 1 2 3 4 5

never always

14. I keep track of my pain level

0 1 2 3 4 5

never always

15. I become preoccupied with pain

0 1 2 3 4 5

never always

16. I do not dwell on pain

0 1 2 3 4 5

never always

Participant code ❑❑❑

QUESTIONNAIRE FOUR

Please circle the number that best describes what is true for you at this time.

	Strongly Disagree			Strongly Agree
1. I'm afraid that I might injure myself if I exercise.	1	2	3	4
2. If I were to try to overcome it, my pain would increase.	1	2	3	4
3. My body is telling me I have something dangerously wrong.	1	2	3	4
4. My pain would probably be relieved if I were to exercise.	1	2	3	4
5. People aren't taking my medical condition seriously enough.	1	2	3	4
6. My condition has put my body at risk for the rest of my life.	1	2	3	4
7. Pain always means I have injured my body.	1	2	3	4
8. Just because something aggravates my pain does not mean it is dangerous.	1	2	3	4
9. I am afraid that I might injure myself accidentally.	1	2	3	4
10. 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
11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body.	1	2	3	4
12. Although my condition is painful, I would be better off if I were physically active.	1	2	3	4
13. Pain lets me know when to stop exercising so that I don't injure myself.	1	2	3	4
14. It's really not safe for a person with a condition like mine to be physically active.	1	2	3	4
15. I can't do all the things normal people do because it's too easy for me to get injured.	1	2	3	4
16. Even though something is causing me a lot of pain, I don't think it's actually dangerous.	1	2	3	4
17. No one should have to exercise when he/she is in pain.	1	2	3	4

Participant code ❑❑❑

QUESTIONNAIRE FIVE

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.

0 - not at all **1** - to a slight degree **2** - to a moderate degree **3** - to a great degree **4** - all the time

When I'm in pain...

1. ❑ I worry all the time about whether the pain will end.
2. ❑ I feel I can't go on.
3. ❑ It's terrible and I think it's never going to get any better.
4. ❑ It's awful and I feel that it overwhelms me.
5. ❑ I feel I can't stand it anymore.
6. ❑ I become afraid that the pain will get worse.
7. ❑ I keep thinking of other painful events.
8. ❑ I anxiously want the pain to go away.
9. ❑ I can't seem to keep it out of my mind.
10. ❑ I keep thinking about how much it hurts.
11. ❑ I keep thinking about how badly I want the pain to stop.
12. ❑ There's nothing I can do to reduce the intensity of the pain.
13. ❑ I wonder whether something serious may happen.

_____ **Total**

Participant code ❑❑❑

QUESTIONNAIRE SIX

Written below are a number of situations that might occur in daily life, each followed by a thought "in quotations", that a person in the situation may have. Underneath each situation is a group of responses that describe how similar the statement is to one you might think. Please read each situation and vividly imagine it happening to you. Then:

read the thought *(which is in quotations)* following that situation.
CIRCLE one response under each thought that best describes how similar that thought is to the one you might have
be sure to rate the thought, rather than the relevance of the situation to you. If the situation is not one you have experienced, please try and imagine that it is happening to you.

1 Last week you painted the living room and your spouse said that it really looked great. When you were cleaning up, you found that you had got paint on the carpet and thought *"Oh no, this wasn't a very good painting job."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

2 You are a manager in a small business firm. You have to give an employee, who has been doing a terrible job, a severe warning that she is running the risk of losing her job. You have been putting off meeting with her for a few days and think to your self, *"I just know that she will resent me giving her the warning and will turn all the other employees against me. "*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

3 You work at a job which requires some lifting and carrying heavy boxes. The other day, you felt really weak and your muscles felt stiff and sore at the end of the day. Driving home from work, you find yourself thinking, *"If this keeps up I won't be able to work or even walk, and might land up permanently bedridden. "*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

4 You and your partner went to a party the other day and you had a bad time because you felt very fuzzy headed and had to ask the host if you could lie down in the bedroom for half an hour. When your partner asks you to go to a party the following weekend, you think to yourself *"I don't want to go because I am going to have to lie down again."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

5 Recently a number of your friends are learning to play tennis. You would like to learn, but remember difficulty you had the time you tried to ski. You think to yourself, *" I was useless at skiing so I doubt if I can learn to play tennis."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

6 You hand in a report to your boss that has taken you four hours to write. Your boss, however, doesn't say anything about it. You think to yourself, "(S)he must think I did a lousy job."

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

Participant code ❑❑❑

7 You have just started a new job, and were obliged to attend the annual office Christmas party. You didn't really know anybody there and had a terrible time. When new neighbours invite you to their house warming party, you think, *"I will have a terrible time, just like at the office party."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

8 A friend has just asked you to go out for a walk. You remembered how very fatigued and sore you felt after playing a game of tennis the other day and you think to yourself, *"I guess there is no way I could hold up if I went out for a walk with him/her."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

9 You played golf for the first time today with some of your friends who play regularly. Everybody seemed bit disappointed with their play, and the group seemed a bit subdued on the way home. You thought to yourself, *"I guess I held them back and spoilt the game for them."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

10 Recently your job has been so demanding that you have worked straight through your lunch hour. As a result you have been feeling increasingly fatigued and have had difficulties concentrating. Driving home from work, you think, *"If I don't get some time to relax during the day, I am going to have a total collapse and be unable to work. "*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

11 You have been feeling very weak and tired of late, but have continued to work. Although you got quite a bit done today, you finished work early because you were feeling particularly exhausted. You think to yourself, *"What a terrible day; it seems like I can't get anything done."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

12 You and your family went to an afternoon rugby game. You enjoyed the first half of the match, but then you started to feel tired and your back was aching. You find yourself thinking, *"What an awful way to spend an afternoon."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

13 Your nine-year old son normally does well at school. Last week, he brought back his maths homework which he had done incorrectly and was supposed to do over. You think to yourself, *"Oh no, now he is having trouble with his schoolwork."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

14 Earlier today, your partner asked to have a serious talk with you after work about some things that were troublesome at home. You have no idea what is going on and you think, *"We don't communicate enough; our marriage is going to fall apart."*

almost exactly like I would think | a lot like I would think | somewhat like I would think | a little like I would think | not at all like I would think

Participant code ❑❑❑

15 On your last job, you had not received a raise even though a co-worker with similar experience had. You are now up for a raise in your present job and think, *"I didn't get a raise last time and I probably won't get one now."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

16 You run a day care centre. Today, the mother of a child you have been having difficulties with calls and notifies that she has quit work and will be withdrawing her child from your programme. You think, *"She probably thinks I wasn't handling him as well as I should."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

17 You have just returned to your job after a sick leave during which you were recovering from yet another bout of flu. You worked hard all afternoon, but didn't finish everything you wanted to do. You think to yourself, *"Because of these recurring episodes of flu, I can't do my job."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

18 You met with your boss today to discuss how you have been doing in your job. (S)he says that you were doing a really good job, but asked you to improve in one small area. You think to yourself, *"(S)he really thinks I am doing a lousy job."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

19 Your favourite exercise is swimming. Even though you have been feeling very tired and your muscles feel sore after swimming, your doctor has urged you to keep up your exercise regime. Today when you were having your normal swim, your muscles started to ache before you were finished. You think to yourself, *"Pretty soon, I won't be able to swim at all."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

20 You teach at a primary school. The last time it was your turn to stand and watch the children in the playground during lunch break, your muscles felt weak and ached for the rest of the afternoon. You notice that it is your turn again and think, *"If I have to watch those kids during break, I just know my body will hurt for the rest of the day."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

21 You have an argument with a friend. When she doesn't call you as usual during the week, you think, *"Our friendship is ruined, and she doesn't want to speak to me again."*

almost exactly like I would think	a lot like I would think	somewhat like I would think	a little like I would think	not at all like I would think

Appendix C

Missing Data and Evaluation of Assumptions

Missing data was handled by mean substitution. The mean score for participants' responses to the questionnaire as a whole was substituted for the missing item. If the item was reverse keyed the mean reverse keyed items on that measure was substituted. The impact of this approach was assessed by comparing the sample mean with the case deleted and the sample mean after mean substitution. Mean differences were in the order of .1 for only two items. The other items did not alter. Therefore, it was considered that the strategy used was appropriately conservative.

Two cases were not included in the analyses as the tender point rating was missing and they could not be diagnosed according to ACR criteria. Listwise deletion was used for descriptive statistics, and independent *t* tests used in the assessment of between group differences for individuals with FMS, OOS, and for the comparison group. Casewise deletion was used for both overall means scores of symptomatic participants and for bivariate and multivariate analyses. This combination was used to extract maximum information, but also to ensure consistency of data used in the regression analysis.

As outlined in the method, only one of the three scores derived from the pain questionnaire was used in the analysis. The relationship between the three scores was evaluated. The scores pertaining to pain on the day were significantly correlated. These measures were also significantly related to "usual pain". Therefore, one measure of current pain, the visual analogue score, was used in the analysis.

Outliers

An assessment of univariate outliers was carried out by converting data for each variable into standard scores also by assessment of histograms and box plots. Two outliers were identified for catastrophising and two for pain and discomfort, while somatic cognitive errors and total cognitive errors each had one outlier. These were only identified in the box plot as outliers, rather than extreme outliers. Individual cases were analysed before a decision to maintain the individuals within the sample. Analyses repeated without the outliers confirmed that they did not unduly influence the results. The pattern and magnitude of significant relationships did not alter. Outliers for the variable symptoms of pain and discomfort were more extreme. Additionally, there were issues of non-normality for

this variable and logarithmic transformation was performed to address both skewness and non-normality.

Normality

Univariate normality was assessed visually by means of a histogram and normal probability plot, and statistically from derivation of standard scores and the Kolmogorov-Smornov statistic. The following variables were normally distributed; pain, disability, fear of movement/reinjury, vigilance, age, and tender points, the latter only after removal of asymptomatic individuals, whose counts would not be included in analyses. The variables catastrophising and cognitive errors (total, somatic, and general) deviated slightly from normality in relation to several of the parameters assessed. However, bearing in mind the robust nature of this assumption data was not transformed. The variable symptom duration deviated substantially from normality on all parameters. This finding, together with the presence of two extreme outliers, made transformation necessary as described above.

Multiple Regression Assumptions

There were 66 participants and six independent variables. The ratio of cases to independent variables was 11. This meets the minimum requirements for a multiple regression advocated by Tabachnick and Fidell (1996). Residual analysis allowed for assessment of the assumptions of multivariate analysis, normality, linearity and homoscedasticity, between predicted scores on the dependent variable and errors of prediction (Tabachnick & Fidell, 1996). Additional assessment of assumptions was carried out using partial regression plots to assess linearity of individual variables plotted against the dependent variable. The normal probability plot was also examined to evaluate assumptions of normality. Finally, the value reported for the Durbin Watson statistic (1.97) indicated independence of residual error. Variance inflation factors and tolerance data indicated that multicollinearity was not a problem. The Mahalanobis distance, was checked for the presence of multivariate outliers and none were present. Univariate and multivariate assumptions were re-evaluated for the cross product term (pain/catastrophising), and did not appear to be violated.