Cognitive Behavioural Models of Chronic pain

&

The Role of Selective Attention

Mohsen Delgittani

Submitted in fulfilment of the requirements for the degree of
Doctor of Philosophy

School of Psychology
Faculty of Science
University of Sydney

September 2003
Table of Contents

1 Chapter One .......................................................................................................................... 8
   1.1 Introduction ..................................................................................................................... 8
   1.2 Types of pain .................................................................................................................. 9
       1.2.1 Phasic pain .............................................................................................................. 9
       1.2.2 Acute pain ............................................................................................................. 9
       1.2.3 Chronic pain .......................................................................................................... 10
   1.3 Prevalence, incidence, and costs of chronic pain .......................................................... 11
   1.4 Theories of Pain ............................................................................................................ 13
       1.4.1 Neurophysiological models of acute pain ............................................................. 13
           1.4.1.1 Specificity Model .......................................................................................... 13
           1.4.1.2 Gate Control theory .................................................................................... 15
       1.4.2 Neurophysiological mechanisms of pain ............................................................... 17
           1.4.2.1 Peripheral mechanisms ................................................................................ 17
           1.4.2.2 Dorsal horn mechanisms ............................................................................. 18
           1.4.2.3 Supraspinal mechanism .............................................................................. 19
       1.4.3 Psychological theories of chronic pain ................................................................. 20
           1.4.3.1 Cognitive-behavioural theories ..................................................................... 23
           1.4.3.2 A cognitive model: Fear of pain .................................................................... 24
           1.4.3.3 Vulnerability Model .................................................................................... 30
           1.4.3.4 Anxiety sensitivity (AS) and chronic pain .................................................... 36
           1.4.3.5 Anxiety Sensitivity and Hypervigilance ....................................................... 38
           1.4.3.6 Is Anxiety Sensitivity different from Trait Anxiety? .................................... 40
           1.4.3.7 Stress-Diathesis Model .............................................................................. 44
       1.4.4 Conclusion and overview of the thesis ................................................................. 47

2 Chapter Two ......................................................................................................................... 49
   2.1 Introduction ................................................................................................................... 49
   2.2 Method .......................................................................................................................... 54
       2.2.1 Measures .............................................................................................................. 56
           2.2.1.1 Fear of Pain Questionnaire – III (FPQ-III) (McNeil and Rainwater, 1998) .... 56
           2.2.1.2 Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992) ......................... 57
           2.2.1.3 Depression, Anxiety, and Stress Scale (DASS) (Lovibond  & Lovibond, 1995)58
           2.2.1.4 Tampa Scale of Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990) ........... 59
           2.2.1.5 Pain severity ................................................................................................. 59
           2.2.1.6 Roland and Morris Disability Questionnaire (RDQ) (Roland & Morris, 1983) 59
           2.2.1.7 The Pain Responses Self Statements (PRSS) (Flor et al, 1993) .................. 60
           2.2.1.8 Pain self-efficacy Questionnaire (PSEQ) (Nicholas, 1989) ....................... 60
           2.2.2 Subjects ............................................................................................................. 61
           2.2.3 Structural Models ............................................................................................... 61
       2.3 Results ......................................................................................................................... 64
           2.3.1 Participants ........................................................................................................ 64
           2.3.2 Self-report data ................................................................................................. 65
       2.4 Discussions ................................................................................................................. 73

3 Chapter Three ...................................................................................................................... 81
   3.1 Selective memory in chronic pain ................................................................................ 82
   3.2 Selective attention ........................................................................................................ 87
       3.2.1 Stroop Task .......................................................................................................... 88
       3.2.2 Dot Probe Paradigm ........................................................................................... 99
   3.3 Summary of limitations of the research to date ........................................................... 103

4 Chapter Four ........................................................................................................................ 106
   4.1 Introduction ................................................................................................................... 106
   4.2 Method .......................................................................................................................... 109
       4.2.1 Design .................................................................................................................. 109
       4.2.2 Participants .......................................................................................................... 109
       4.2.3 Measurements ..................................................................................................... 110
5 Chapter Five .......................................................... 132
5.1 Introduction .......................................................... 132
5.2 Method ................................................................. 134
  5.2.1 Design ............................................................. 134
  5.2.2 Subjects ........................................................... 134
  5.2.3 Measures ......................................................... 134
    5.2.3.1 Anxiety Sensitivity Index (ASI) ....................... 135
    5.2.3.2 Fear of Pain Questionnaire-III (FPQ-III) ........ 135
    5.2.3.3 Depression, Anxiety, Stress Scales (DASS) ....... 135
    5.2.3.4 Selective attention measure ......................... 135
  5.2.4 Procedure ....................................................... 136
  5.2.5 Analyses .......................................................... 137
  5.3 Results ............................................................... 137
    5.3.1 Demographic features ................................... 137
    5.3.2 Self-report measures ....................................... 139
    5.3.3 Dot probe task ............................................... 140
  5.4 Discussion ........................................................ 142

6 Chapter Six .............................................................. 148
6.1 Introduction .......................................................... 148
6.2 Method ................................................................. 152
  6.2.1 Participants ...................................................... 152
  6.2.2 Procedure ....................................................... 153
  6.2.3 Measurements .................................................. 154
    6.2.3.1 Fear of Pain Questionnaire – III (FPQ-III) (McNeil and Rainwater, 1998) ....... 154
    6.2.3.2 Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992) ............................. 154
    6.2.3.3 Depression, Anxiety, Stress Scale (DASS) .......................... 154
    6.2.3.4 Pain severity .................................................... 155
    6.2.3.5 Roland and Morris Disability Questionnaire (RDQ) (Roland & Morris, 1983) ... 155
  6.2.4 Dot probe task .................................................. 156
  6.2.5 Analyses .......................................................... 157
  6.3 Results ............................................................... 158
    6.3.1 Participants characteristics ............................... 158
    6.3.2 Attentional bias analysis ................................. 159
  6.4 Discussion ........................................................ 164

7 Chapter Seven .......................................................... 171
7.1 General Scope ....................................................... 171
7.2 Limitations .......................................................... 172
  7.2.1 Cross-sectional nature ....................................... 172
  7.2.2 Long term follow up ......................................... 173
  7.2.3 Sample size ...................................................... 173
  7.2.4 CBT intervention ............................................ 174
  7.2.5 Validity of Dot Probe Task ............................... 175
  7.3 Strengths ............................................................ 176
  7.4 Psychological models of chronic pain ....................... 177
  7.5 Attentional biases in chronic pain patients ................ 179
    7.5.1 Study Two (chapter 4) ..................................... 179
Table of Figures

Figure 1.1 Asmundson's model of anxiety sensitivity and fear of pain........................................34
Figure 1.2 Contemporary models of chronic pain........................................................................35
Figure 2.1 Fear avoidance model (Vlaeyen et al., 1995)..................................................................50
Figure 2.2 Structural model as predicted based on Vlaeyen et al. (1995) Theory. Paths marked with “1” are fixed to identify the model. Neg/aff: Negative affectivity (LV); Avoidance: Fear avoidance (LV)........................................................................................................62
Figure 2.3 depicts predicted relationships based on model 2. Paths marked with “1” are fixed to identify the model. R1-R4: residuals; Z1: equivalent with constant in regression equation; FPQ: Fear of pain questionnaire; TSK :Tampa Kinesiophobia Scale; PRSS-CAT: catastrophising subscale of PRSS; Avoidance: Fear/avoidance of pain (LV); PSEQ: Pain self-efficacy questionnaire; RDQ: Roland & Morris disability checklist. Pain: MPI pain severity subscale; ASI: Anxiety sensitivity scale; DASS-A & DASS-D: Anxiety and depression subscales of DASS respectively. ....................64
Figure 2.4 Shows the standardised path coefficients for the variables in the model 1. (all coefficients are significant, p<.001). R1-R8: residuals; Z1: equivalent with constant in regression equation; FPQ: Fear of pain questionnaire; TSK :Tampa Kinesiophobia Scale; PRSS-CAT: catastrophising subscale of PRSS; Avoidance: Fear/avoidance of pain (LV); PSEQ: Pain self-efficacy questionnaire; RDQ: Roland & Morris disability checklist. Pain: MPI pain severity subscale; ASI: Anxiety sensitivity scale; DASS-A & DASS-D: Anxiety and depression subscales of DASS respectively. ....................68
Figure 2.5 shows the structural model of pain fear/avoidance and disability and the impact of pain self-efficacy (All coefficients are significant, p<.001). .................................................................70
Figure 2.6 reflects a model based on the combination of models one and two.

Figure 4.1 Mean reaction times for sensory words by fear of pain group.....................................121
Figure 4.2 Mean reaction times for disability words by fear of pain group...................................122
Figure 4.3 Mean Reaction times for affective words by fear of pain group.................................122
Figure 4.4 Mean reaction times for threat words by fear of pain group........................................123
Figure 4.5 Pattern of attentional biases for different type of words. .................................................125
Figure 5.1 presents the pattern of attentional biases for different word types for patients with chronic pain and healthy control subjects. .................................................................142
Figure 6.1 Demonstrates the change in attentional bias towards sensory-related pain words relative to other types of word at three occasions of assessment.................................161

List of Tables

Table 2.1 shows the means and (SDs) for self-report questionnaires..........................65
Table 2.2 Inter-correlation between measurements...............................................................66
Table 3.1 summarises the studies used Stroop task in chronic pain population..................98
Table 4.1 Word pairs used in the Dot-probe Task.................................................................114
Table 4.2 Characteristics of participants .............................................................................117
Table 4.3 Inter-correlation between questionnaires .........................................................119
Table 4.4 Mean reaction times (ms) of each word group (affective, disability, sensory, and threat), word location (upper/lower), and probe location (upper/lower) for total sample and fear of pain groups.................................................................120
Table 4.5 Word pairs used in the Dot-probe Task. .................................................................136
Table 5.1 Word pairs used in the Dot-probe Task. .................................................................136
Table 5.2 Presents the demographic characteristics of experimental groups................138
Table 5.3 Means (SDs) of the chronic pain patients and healthy controls on self-reported measures.............139
Table 6.1 Psychological profile of patients at pre, post, and follow up sessions. Means (SD) ....158
Table 6.2 shows raw scores on dot probe at three occasions.

Table 6.3 Inter-correlation of change scores for clinical measures, demographic features, and sensory index.

Table 6.4 presents the regression model used to predict change score in attentional biases to sensory words from time 2-time 3. Predictor variables are change scores during the treatment period.
Abstract

Cognitive-behavioural based models of chronic pain contend that appraisals of harm affect the individual’s response to pain. It has been suggested that fear of pain and/or anxiety sensitivity predispose individuals to chronicity. However, other factors such as pain self-efficacy are believed to mediate between experience of pain and disability. According to this view, pain is maintained through hypervigilance towards painful sensations and subsequent avoidance. Four studies were conducted in order to evaluate the structure of fear-avoidance models of chronic pain, and also, to examine the role of hypervigilance as an underlying mechanism in maintenance of pain.

In study one, using a sample of 207 consecutive patients, two models were tested. First, fear of movement model as proposed by Vlaeyen et al. (1995a) was examined. It was found that negative affectivity has direct effects on the fear and avoidance of pain, which in turn, contributes to disability. In total, fear/avoidance accounted for a significant amount of the variance of disability. In addition, severity of pain was found to increase pain disability, while itself is influenced still by negative affectivity. These findings supported the model of fear of pain as described by Vlaeyen et al. (1995a). Further, we found that self-efficacy may mediate the impact of fear of pain on disability and reduces the perceived physical disability. At the same time, self-efficacy was shown to have direct reductive impact on disability. However, both studies indicated that people who are fearful in response to pain are more likely to develop disability, although self-efficacy may play a moderating role.
In the studies one, two, and three, the role of hypervigilance in over attending to pain was investigated. In study one a large sample of 168 chronic pain patients were studied. Questionnaires measuring different aspects of pain and a computerised version of the Dot-Probe Task were administered. Four types of words related to different dimensions of pain and matched neutral words were used as stimuli. Reaction times in response to the stimuli were recorded. A factorial design 3x4x2x2 and ANOVAs were employed to analyse the data.

Chronic pain patients showed a cognitive bias to sensory pain words relative to affective, disability, and threat-related words. However, contrary to expectations, those high in fear of pain responded more slowly to stimuli than those less fearful of pain.

These results suggest that patients with chronic pain problems selectively attend to sensory aspects of pain. However, selective attention appears to depend upon the nature of pain stimuli. For those who are highly fearful of pain they may not only selectively attend to pain-related information but also have difficulty disengaging from those stimuli.

In study two, 35 chronic pain patients were compared with the same number matched healthy subjects. Both groups completed measures of fear of pain, anxiety sensitivity, depression and anxiety, in addition to dot probe task. Results indicated that both groups show similar attentional bias to sensory words in comparison with other word types. However, the level of this biasness was higher for chronic pain patients. Lack of significant differences between patients and controls is discussed in the context of possible evolutionary value of sensitivity to
pain as an adaptive reaction in healthy controls, and contrary, as a maladaptive response to pain in chronic pain patients.

The results of the previous research suggest that chronic pain patients demonstrate cognitive biases towards pain-related information and that such biases predict patient functioning. The forth study examined the degree to which a successful cognitive-behavioural program was able to modify the observed attentional bias towards sensory pain words. Forty-two patients with chronic pain conditions for more than three months were recruited prior to commencing a cognitive-behavioural pain management program. Participants were assessed before the program, after the program and at one-month follow-up. Results confirmed that chronic pain patients exhibited biased attention towards sensory pain-related words at pre-treatment. These biases were still evident at post-treatment, but were no longer statistically significant at follow up. Multiple regression analyses indicated that the changes in attentional bias towards sensory words between post-treatment and follow-up were predicted by pre- to post-treatment changes in fear of movement (Tampa Scale for Kinesiophobia) but not other relevant variables, such as fear of pain or anxiety sensitivity. These results demonstrate that successful cognitive-behavioural treatments can reduce selective attention, thought to be indicative of hypervigilance towards pain. Moreover, these biases appear to be changed by reducing the fear associated with movement. Theoretically, these results provide support for the fear of (re)injury model of pain. Clinically, this study supports the contention that fear of (re)injury and movement is an appropriate target of pain management and that reducing these fears causes patients to attend less to pain-related stimuli.
Chapter One

Literature Review

1.1 Introduction

With notable exceptions, pain is a universal phenomenon, which is experienced by all individuals at some stage in their life. Although adaptive as a warning to injury, persisting pain can threaten individual well-being. Indeed, a World Health Organisation study has confirmed pain as the single most important predictor of poor quality of life (Skevington, 1998).

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” (IASP Sub-committee on Taxonomy, 1979; p.250). The application of words like “unpleasant”, “emotional”, and “potential” in this definition have served to challenge the traditional view of a direct link between pain and tissue damage. Today, pain is conceptualised as a complex subjective, perceptual, and multidimensional phenomenon that varies in intensity, quality, time course, and personal meaning (Merskey & Bogduk, 1994). There is now a general agreement amongst experts in the field that physiological factors cannot completely account for the experience of pain (Turk, 1996).
1.2 Types of pain

Different types of pain have different aetiologies, correlates, and functions for the individual. Within the literature, three distinct types of pain have been identified and referred to as phasic, acute, and chronic pain (Loeser and Melzack 1999; Melzack and Wall, 1988).

1.2.1 Phasic pain

Phasic or transient pain reflects the activation of nociceptive transducers in skin or other tissues of the body in the absence of any tissue damage (Loeser and Melzack, 1999). Phasic pain has a brief time course usually subsiding within minutes (Craig, 1999). For example, when a person stubs his/her toe, it is painful but once the immediate cause of the pain resolves, the pain will subside quickly. With phasic pain, the removal of the stimulus that provoked the pain makes it possible for the person to continue the task that he/she was doing before (Melzack and Wall, 1988). It is rare for this kind of pain to be associated with significant emotional responses. However, since phasic pain is not a matter of clinical significance and not the source of interference in normal life, research focuses on acute and chronic types of pain.

1.2.2 Acute pain

Acute pain is likely to be associated with specific tissue damage. Acute pain occurs following an injury and during the period of recovery (Melzack and Wall, 1988). Acute pain reflects an injury and signals a source of threat to allow the individual to take action. At this point, pain is acting as a useful and protective mechanism. Acute pain resolves once healing is completed and gradually improves allowing the individual to gradually resume their normal activities. Although most people who experience an acute injury successfully return to
normal functioning within 6 – 12 weeks, it is estimated that for about 10% of adults who sustain musculoskeletal injury, the experience of pain persists long after any identifiable pathology would be expected to have healed (Waddell, 1987). However, this period varies depending on the site and the presence of injury. Low back pain patients who make a recovery are reported to return to work most commonly within 4 weeks (Pengel et al., 2003). Pain that persists in this way is usually considered to be chronic in nature.

1.2.3 Chronic pain

The term chronic pain is usually used to describe pain that has persisted beyond the expected healing period (Bonica, 1977). Merskey and Bogduk (1994) defined pain as a chronic type when it persists at least for 3-6 months. Although pain is usually perceived as a signal of potential physical damage that interrupts normal activity, when pain persists beyond healing and becomes chronic, the pain no longer represents new damage. In contrast to phasic and acute pain, chronic pain does not have any obvious protective value. Once chronic pain develops, it is typically resistant to medical therapies and is often associated with increasing levels of disability. As a result, chronic pain is frequently associated with psychological consequences for individuals with chronic pain.

Chronic pain is frequently associated with depression, anxiety and anger (Craig, 1999). Most researchers agree that as pain and disability continue and increase, the emotional consequences become more marked creating a vicious cycle. In response to chronic pain, an individual may become unable to continue their gainful employment, normal family life, and/or their social interactions. Chronic pain is not a symptom that exists in isolation. The experience of chronic pain is
frequently associated with a cluster of related problems such as chronic fatigue, sleep disturbance, excessive rest and withdrawal from activity, compromised immune function, and mood disorder (Chapman and Gavrin, 1999).

1.3 Prevalence, incidence, and costs of chronic pain

Chronic pain is recognised as an important problem internationally. The exact prevalence of chronic pain is difficult to estimate, due to conflicting findings reported in the literature. However, much of the confliction is due to differing definitions and applied methodologies (Blyth et al., 2001). In a systematic literature review of prevalence studies of chronic pain conducted between 1966-1998, Walker (2000) analysed 56 studies considering their definition of pain, sample representativeness, and the quality of their data (e.g. questionnaires not tested adequately or inadequate interview). Out of the 56 studies reported, Walker (2000) considered only thirty studies to be methodologically acceptable. Even so, amongst the remainder, point prevalence of pain ranged from 12% to 33%, 1-year prevalence ranged from 22% to 65%, and lifetime prevalence ranged from 11% to 84%.

The large differences in prevalence estimates of chronic pain arise from the different methodologies employed. For example, studies have used different definitions of chronic pain, have recruited samples differently leading to questionable representativeness, and varied other methodological issues (e.g. inclusion of qualitative description of pain to patients or utilising screening questionnaires or interviews related to frequency of experiencing pain) (Walker, 2000; Blyth et al., 2001). For example, Elliott et al. (1999) found one of the highest rates of chronic pain amongst prevalence trials. In their study, chronic pain was defined as pain or discomfort, which was rated on a seven item self
report scale. The sample ($N=3605$) was recruited from one region, and included respondents aged over 25. Depending upon where they placed their cut-off, the reported rates of chronic pain were between 30 and 80%. Using more conservative criteria, that included only “severe” and “significant pain” that had lasted for at least three months and required treatment, Smith et al. (2001) found prevalence estimates of 14.1%.

A recent well-conducted study investigated the prevalence rates of chronic pain in New South Wales, Australia (Blyth et al., 2001). The sample was large ($N=17,543$) and was selected randomly in a careful design from the adult population of NSW. Participants were considered to have chronic pain if they were “experiencing pain every day for three months in the six months prior to the interview” (Blyth et al., 2001, p128). This study indicated that the overall chronic pain prevalence was 17.1% for males and 20% for females. From this population 11% - 13.5% of the sample reported that their daily activities had been affected directly as a result of pain. These figures are consistent with Lawson’s earlier prevalence study (1991) in Australia, which found musculoskeletal disorders and chronic disabilities affected 20% of the Australian population each year.

Despite the wide ranging estimates of prevalence, even the most conservative estimates indicate that chronic pain affects at least 10% of the population worldwide (Crook & Tunks, 1985). Further, approximately 1% of the population are severely disabled as a result of chronic pain (Von Korff, Dowrkin & Le Resche, 1990). Although there is no evidence of a consistent increase in the prevalence of chronic pain, international studies in western countries reveal that the rate of permanent disability has increased dramatically. Statistics in New South Wales show that the assignment of permanent disability associated with chronic pain in

The increasing rates of permanent disability, have contributed to the growing cost of chronic pain. Guo (1999) found that back pain is the most commonly cited reason for filing workers’ compensation claims in the US and leads to loss of 101.8 million workdays annually. The annual costs of medical care for back pain alone in the United State have been estimated at approximately $ AUS 50 billion (Frymoyer and Cats-Brail, 1991). Similarly high costs have reported in Australia, with Blyth et al., (2001) reporting that the associated costs of chronic pain to the community are $AUS10 billion annually. These figures clearly indicate that chronic pain is a common problem, that increasingly leads to permanent disability and is associated not only with a personal burden to people with chronic pain but also with a large economic burden to most Western communities.

1.4 Theories of Pain

1.4.1 Neurophysiological models of acute pain

1.4.1.1 Specificity Model

Descartes described a pain system in 1664. He developed the hypothesis that there was a direct channel from the skin to the brain that accounted for the experience of pain. The theory underwent little change until the nineteenth century, when physiology as an experimental science emerged (Melzack and Wall, 1988). The physically oriented theories of pain remained dominant in both the explanation of pathology and treatment of pain until the middle of the twentieth century. At this time, it was acknowledged that the purely sensory approach failed to provide a complete explanation of pain (Melzack and Wall, 1988). For example, the
specificity model cannot explain phenomena such as phantom pain, where pain is not dependent on activity in nociceptors or nociceptive fibres (Nicholas, 1988).

Specificity theory hypothesised that a specific pain receptor transfers the pain message from the skin to a pain centre in brain (Melzack and Wall, 1988). According to this completely physiological mechanism, neurons were specialised to conduct patterns of nerve impulses from a receptor to the brain. Based on this theory, the assumption was that an individual experienced pain when there was a specific biological stimulus that produced physical damage (Turk, 1996). In the 1960’s, pain became recognised not just as a sensory quality, but also as a factor that motivated individuals to take an action to stop the pain and the related threat of injury. Pain continued to be seen primarily as a physiological sensation, hence the motivational contingencies were usually considered as reactions to pain, and viewed as secondary factors (Melzack and Wall, 1988). Based on this dualistic view, it was assumed that as the condition was treated, these secondary reactions would disappear.

According to dualistic point of view, pain was equated with nociception, and no role was given to non-physiological (i.e. psychological and social) factors. Accordingly, pain was considered a pure function of sensory input and a consequence of quality and intensity of the painful stimulus (Turk et al., 1983). However, the model could not account for the observation that pain sometimes occurred in the absence of physical damage or physical damage occurred without pain, as seen among injured soldiers in battlefield (Melzack and Wall, 1988). The dualist theory remained the dominant model of pain, until new developments in medical technology and science emerged that allowed the theory to be tested. These developments, together with failure of the dualistic theory to satisfactorily
account for the experience of chronic pain, led to the formulation of the still influential Gate Control Theory of pain (Melzack and Wall, 1965).

1.4.1.2 Gate Control theory

The Gate Control Theory (Melzack and Wall, 1965) was the first attempt to give an integrated and comprehensive model of pain (Turk, 1996). Melzack and Wall (1965) proposed that the substantia gelatinosa functions as a “gate” to transmission of nociceptive information. This gate consists of interneurons that balance the level of activity among the small fibres (A-Delta and C) and large (A-beta) sensory fibres. The degree to which this gate is opened or closed depends on the modulated balance of input from these two types of sensory nerves and the influence of descending signals from brain. According to this theory, peripheral sensory input is regulated at this gate before transmission to the brain through the spinothalamic tract.

Gate Control Theory, by incorporating the central control of the brain in pain modulation, implies the influence of the psychological or cognitive processes such as attention, anxiety, and memory. Since 1965, this theory has been refined (Wall, 1979), mostly due to new findings, such as both small and large fibres input being shown to result in presynaptic inhibition (Nicholas, 1988). However, one of its principal contributions has been the recognition of pain as a much more complex phenomenon than the previous theories had conceptualised. It has been argued, that any theory of pain should explain a) the highly variable relationship between
pain and injury, b) the experience of pain in absence of injury or after healing, and c) the multi-dimensional nature of pain (sensory-emotional nature) (Melzack and wall, 1988). It is well recognised that the Gate Control Theory significantly changed the way pain was conceptualised and influenced the definition of pain that has since placed a much greater emphasis on psychological variables. The influence of the Gate Control Theory can be attributed partially to its recognition that pain has both emotional and sensory components (Craig, 1999).

The Gate Control Theory emphasises the modulation of perceptual inputs in the dorsal horn of the spinal cord, and the role of the brain in processing pain perceptions that could regulate the flow of nociceptive information from the peripheral nervous system to brain (Rhudy and Meagher, 2001). Melzack and Wall (1965) postulated that mechanisms existed through which psychological processes emanating from higher brain centres could functionally “close the gate” through descending pathways, thus modulating the experience of pain.

Melzack and Wall (1965,1988) formulated a relatively comprehensive description of the reciprocal activities between brain and spinal cord to explain the mechanisms that previous theories of pain had failed to explain, however, many of the physiological details of the gate control theory have been questioned in the light of subsequent research (Turk, 1996). Nonetheless, this model has proved flexible in the face of new scientific notions and has proven to be a powerful theory in provoking future research. Since the gate control theory was proposed, no other theory that has attempted to explain pain in terms of purely physiological factors has emerged (Turk, 1996).
1.4.2 Neurophysiological mechanisms of pain

Since the publication of Gate Control Theory in 1965, many advances have occurred in our knowledge of the neurobiology of acute pain and new laboratory models have been developed (Besson, 1999). However, it has been argued that chronic pain differs substantially from acute pain not only in terms of the persistence of pain, but also due to adaptive neurological and biological changes (Besson, 1999). In recent years there has been a move to develop an understanding of pain on the basis of proposed neurophysiological mechanisms. This has been applied especially to pain based on inflammatory as well as neuropathic mechanisms (e.g. Besson, 1999). It has been hoped that this approach would lead to the development of drugs for different neurophysiological mechanisms.

1.4.2.1 Peripheral mechanisms

The peripheral mechanisms of pain are complex and an exhaustive review of physiological accounts of how acute pain can become chronic is beyond the scope of this review. However, a general understanding of the major mechanisms is important to provide a context in which to situate psychologically based theories. Many forms of acute pain are direct product of activation or sensitisation of primary afferent neurons, especially C fibres polymodal nociceptors (Siddall and Cousins, 1995, 1998). However, the process of nociceptor activation together with other processes contributes to and modifies responses to further stimuli (Siddall and Cousins, 1998). In adaptive responses to acute pain, pain stimuli activate high threshold nociceptors, which signal this information to the first relay in the spinal cord.
However, these messages can be distorted when damage occurs to the system. For example, if a noxious stimulus is prolonged, traumatic, and associated with tissue damage, then sensitisation can occur. That is, a low-intensity stimulus, which would not cause pain under normal situation, may now be perceived as painful (Siddall and Cousins, 1998). Alternatively, if peripheral nerves become damaged, a number of biomedical, physiological and morphological changes occur that act as a focus of pain in themselves, which maintain sensory input that may occur. These changes can maintain sensory input even after the acute tissue injury has resolved, resulting in the continued experience of pain (i.e. chronic pain) (Cousins and Power, 1999).

1.4.2.2 Dorsal horn mechanisms

The dorsal horn is the site where primary afferents terminate, and the site of a complex interaction among afferent fibres, local intrinsic spinal neurons, and the endings of descending fibres from the brain (Siddall & Cousins, 1998). Different elements have been shown to be involved in the dorsal horn mechanism of pain.

Termination sites of primary afferents. Primary afferent nociceptors terminate primarily in the dorsal horn, where they connect with several classes of second order neurons. There are two main types of neurons termed as “nociceptive specific” or “high threshold” neurons, which respond to noxious stimuli. The second type is “wide dynamic range” neurons, which respond to both noxious and non-noxious input (Siddall and Cousins, 1998). It is thought that if these neurons become sensitised and hyperresponsive, they may contributes to maintenance of pain.
Neurotransmitters. Pharmacological studies have identified many neurotransmitters involved in pain processes in the dorsal horn. Among them, the excitatory amino acids glutamate and aspartate have a major role in nociceptive transmission in the dorsal horn. With prolonged release of glutamate or activation of neurokinin receptors, a secondary process occurs which seems to be crucial in the development of abnormal responses to further sensory stimuli, such as is thought to be the case in chronic pain.

Intracellular changes. Further activation of neurokinin contributes to the activation of N-methyl D-aspartate (NMDA) receptor. Activation of NMDA receptors appears to set the stage for intracellular events, which lead to changes within the cell that increase the responsiveness of the nociceptive system to stimuli (Besson, 1999).

Central sensitisation. Similar to peripheral sensitisation, following injury, an increased responsiveness to normally innocuous mechanical stimuli occurs in an uninjured tissue surrounding the site of injury. These changes are believed to be a result of processes that occur in the dorsal horn of the spinal cord following injury and may contribute to the prolonged pain and the development of chronic pain (Siddall and Cousins, 1998).

1.4.2.3 Supraspinal mechanism
Second order neurons and their relays terminate in different supraspinal structures such as brain stem, thalamus, and cortex. These structures are subject to inhibitory and also excitatory influences (Cousins and Power, 1999). In the thalamus, these relays can be divided into two groups: Those involved in the sensory discriminative component of pain, and those regions involved in the affective motivational aspect of pain. However, this division might be an
oversimplification, and the role of the cortex in pain perception remains unclear (Siddall and Cousins, 1998). Putting together, neurophysiological models of pain although have uncovered many mysterious aspects of pain, there is a general consensus that there is no hardware association between these pathologies and the actual experience of pain; especially when pain becomes chronic (Turk and Okifuji, 2002). Although the neurophysiological mechanisms have been better understood in recent years, it remains unclear why some people recover from an acute injury, yet for others the pain develops into a chronic condition. This has led to the development of biopsychosocial explanations for chronic pain. Psychosocial factors are believed to intermediate between neurophysiological mechanisms of pain and the experience or perception of pain.

1.4.3 Psychological theories of chronic pain

The Gate Control Theory was an important milestone in consideration of psychological factors in pain. However, the mechanism(s) by which the myriads of behaviours that are associated with chronic pain develop remained unexplained. Pain behaviours like grimacing, limping, lying down, bracing and using aids, are behaviours that refer to the different motor, verbal and non-verbal responses that often occur in response to the experience of pain (Fordyce, 1976). These behaviours are observable by others and are thought to be influenced by actual or expected environmental consequences, which were not considered in earlier formulations of the gate control theory (Loeser and Melzack, 1999).

Fordyce (1968, 1976, 1982) was the first to recognise and emphasise pain behaviours in our understanding of the experience of chronic pain. At the time, behaviourism was a dominant approach in psychology, and Fordyce (1968, 1976)
used behavioural principles to account for the development of pain behaviours. Fordyce (1968, 1976) argued that pain behaviours were learned through both operant and classical conditioning paradigms. The basic assumption of this model is that if reinforcing consequences follows pain behaviours, then they will be learned. As an example of classical conditioning, if a person becomes anxious when they experience pain, pain may becomes a conditioned stimulus for anxiety and the adaptive response to situations of threat result in either avoidance or escape. As a result the person may learn to avoid situations in which they experience pain or expect to experience pain. From an operant conditioning perspective, avoidance of activities may be reinforced by reduction of pain or threat of pain and injury (Fordyce, 2001). For example, an individual lifting a box may experience a sharp pain in his back, and then he may limp to a chair and avoid performing such an unpleasant task later (Fordyce, 2001). Although avoidance or escape is an adaptive response to real situations of threat, when pain persists after injury has healed, avoidance may become a maladaptive behaviour.

Fordyce (1976) and Fordyce et al. (1982) argued that pain behaviours are an integral aspect of the experience of chronic pain problems. In the extreme, the behavioural point of view claims that pain behaviours can continue as a consequence of learning paradigms without substantial ongoing nociceptive input. In addition to the contingencies between pain, anxiety and avoidance, described above, Fordyce (2001) proposed other operant factors that were likely to influence pain behaviours. That is, pain behaviours may persist at least in part as a result of social contingencies through financial or emotional support from friends and families (Kerns and Rosenberg, 1995). Potential sources of reinforcement for pain behaviour may include behaviours endorsed by others, through sympathy, encouraging the patient to rest, or leave their responsibilities to others (Friedman,
et al. 1998). The role of the significant others in response to behaviours associated with pain has been emphasised (Romano et al., 1992; Turk, 1996; Sharp and Nicholas, 2001). On the other hand, “well behaviours” (activity and return to work and activity) may not be sufficiently reinforced by others. In addition, such well behaviours may be accompanied by exacerbations in pain that further inhibit this way of responding.

In summary, based on operant and classical conditioning paradigms, behavioural theory proposed that independent of the nociceptive stimuli, pain behaviours persist if the appropriate reinforcement contingencies are present. Much of the evidence in support of the behavioural perspective comes from the success of behavioural treatments in reducing pain behaviour (e.g. Fordyce et al, 1982). However, in a recent experimental study, Flor, Knost, and Birbaumer (2002) examined the role of operant conditioning in maintenance of pain in a sample of chronic pain patients and matched healthy controls. Experimental pain was inducted by electric stimuli followed by randomly up-training (subjects were given positive feedback when their actual pain rating was higher than the average rating of the last ten trials, and lower ratings were followed by negative feedback) or down-training (positive/negative feedback assignment was defined conversely) verbal feedback. Both groups in up-training condition significant increase in pain rating in comparison with down-trained condition. However, while healthy controls showed fast extinction, chronic patients maintained their elevated pain ratings throughout the extinction phase. Flor et al. (2002) concluded that this might suggest a susceptibility to operant conditioning for pain behaviour in this group. However, since patients had already developed chronic pain, it is not clear whether this delayed extinction is a consequence of being in chronic pain, or as the authors have suggested might be a predisposing factor. Nevertheless, together
with similar studies by Linton and Gotestam (1985), these results do provide experimental support for the idea that pain behaviours can be learnt independent of noxious input.

Turk (1996) criticised the behavioural approach for its exclusive focus on motor behaviour, neglecting emotional and cognitive aspects of pain and ignoring the importance of patients’ interpretations of the environmental changes. Similar criticisms have led other authors to consider what the cognitive and emotional correlates of chronic pain might be and how they might contribute to the experience of chronic pain (e.g. Turk and Rudy, 1991). It has been also argued that behavioural perspective has neglected the fact that learning might not be the same in all people and the need to consider individual differences (Linton, 2002). Linton (2002) also suggested that the behavioural accounts of chronic pain could appear to imply that human beings are passive respondents to physical sensations and environmental contingencies. Therefore, while pain behaviours appear to contribute to the chronic pain experience, they are not sufficient to explain the complex cognitive and emotional correlates of chronic pain (Sharp, 2001).

### 1.4.3.1 Cognitive-behavioural theories

In contrast to the behavioural accounts of chronic pain, recent cognitive theories argue that individuals appraise their experience of situations and it is their appraisal of that situation that is the basis for their chosen action (Turk, 1996; Sharp, 2001). Turk, Meichenbaum and Genest (1983) emphasised the importance of individual’s beliefs, cognitive processes, problem solving and coping skills in the development and maintenance of chronic pain. Their model was largely derived from cognitive theories of emotional disorders as applied to the
experience of chronic pain (e.g. Beck, 1976, Lazarus, 1982, Bandura, 1977). Turk, Meichenbaum and Genest (1983) proposed a transactional model from acute to chronic pain and emphasised the role of the perception and appraisal of physical symptoms as a determining factor in illness stages. These appraisals were supposed to define the way that the individual perceives and responses to pain. Philips (1987) combined the emphasis on cognitive factors with the behavioural concept of avoidance described by Fordyce (1976; and Fordyce et al, 1982). She argued that avoidance behaviour occurs as a result of the expectancy that further exposure to certain stimuli will exacerbate pain. Phillips’ (1987) account is the first that draws on theories of ‘fear’ to explain aspects of ‘chronic pain’.

Phillips’ (1987) model is important for three main reasons: a) the model argued that avoidance behaviour in chronic pain can continue through anticipation of pain and increased anxiety resulting from these beliefs, b) avoidance will be determined by the motivation to avoid pain, coupled with the belief that a certain behaviour will induce pain (Philips, 1987) and c) there is a reciprocal relationship between cognitions and behaviour that each one reinforces the other one. These theories have led to the emergence of cognitive factors as important elements in development of chronic pain.

1.4.3.2 A cognitive model: Fear of pain

Lethem, Slade, Troup, and Bentley (1983) highlighted the role of fear avoidance, and developed the Fear Avoidance Model of Exaggerated Pain Perception. This model attempted to explain how ‘pain experience’ and the consequent ’pain behaviour’ could become desynchronised with sensations of pain. In other words, Lethem et al. (1983) attempted to explain why fear and avoidance remain after physical injury has healed. Evidence for such an assumption came from research
failing to find simple correlation between pain intensity and tissue damage, and the finding that avoidance was associated with affective qualities rather than sensory qualities of pain (Slade, Troup, Lethem, and Bentley, 1983).

Another essential assumption of Lethem et al.’s (1983) model is that central to ‘fear avoidance’ is the fear of pain. It was argued that the normal response to the threat of pain is fear of that pain. This model proposed two extreme types of response to pain: “confrontation” and “avoidance”. According to this model, confrontation (i.e. approaching situations that may provoke pain) is conceptualised as an adaptive response to chronic pain that promotes recovery. On the other hand, avoidance is described as a maladaptive response, which increases limitations in activity, and has physical and psychological consequences that contribute to disability. Lethem et al. (1983) listed four factors, in addition to fear of pain, that they believed influences the individual’s response to pain: stressful life events; personal pain history; personal coping strategies; and personality characteristics.

As the first attempt to provide evidence for this model, Slade et al (1983) emphasised the role of avoidant versus confrontational coping in chronicity of pain. They studied personal pain history and personal coping strategies in a normal population, and reported significant relationships between reports of back pain and the subjects’ personal history of pain. Students were requested to report if they had any back pain. Students who reported having at least two back pain episodes were divided into two subgroups: those who reported the most recent attack equal or more severe than first attack (increased pain), and those who reported the most recent attack was less severe than first one (decreased pain). Results revealed that first group reported a significantly lower percent of active coping strategies in comparison with the latter group.
According to Lethem et al.’s (1983) model, the establishment of fear of pain is a precursor to avoidance, which was seen as central to the development of chronic pain. Research has generally confirmed the importance of the fear of pain. McCracken et al. (1992) found correlations between pain anxiety as measured by the Pain Anxiety Symptoms Scale (PASS), and standard measures of anxiety, misinterpretation of bodily sensations, depression, and disability in a group of chronic pain patients. Further, McCracken et al. (1993) showed that in chronic low back patients, higher levels of pain-related anxiety were associated with higher expectations of pain and more limitations in a physical test.

Later, Vlaeyen, Kole-Snijder, Boeren, and Van Eek (1995) provided a revision to this model noting the central fear as a specific ‘fear of movement/(re)injury’, rather than a fear of pain, per se. This theory builds strongly on Lethem et al’s (1983) model, and extends the concept of ‘kinesiophobia’ (Kori, Miller, and Todd, 1990). Vlaeyen’s model is based on the concept that it is a specific fear that physical activity may cause (re)injury that promotes fear avoidant behaviour. As in Lethem’s (1983) model, Vlaeyen et al. (1995a) propose two different types of pain appraisal that influence the choice of behaviour. That is, patients who do not catastrophise their pain tend to confront activities associated with pain, however, patients who catastrophise will avoid situations that might promote pain. This fear that further movement is dangerous and will produce increased harm, results in subsequent avoidance, and increased disability.

Vlaeyen et al. (1995b) examined the construct of fear of movement/(re)injury, and studied its relation to behavioural performance. Thirty-three chronic low back pain patients participated in the experiment. Their ability to lift and hold a 5.5 kg weight for up to 300 second was recorded. A significant negative correlation was found between the Tampa Scale for Kinesiophobia and performance on a
behavioural test. Patients who reported fear of movements/(re)injury avoided motor activities more than patients who did less so. However, due to the correlational nature of this study, and its reliance only on physical and motor performance, the results should be treated cautiously.

Vlaeyen, et al. (1995b) investigated the fear of pain/(re)injury in prediction of disability in comparison with pain intensity, catastrophising, and level of impairment. Thirty-three chronic low back pain patients completed questionnaires related to pain cognitions (catastrophising subscale of Pain Cognition List), fear, disability, pain intensity, and self-reported pain symptoms based on a medical coding system. Utilising a hierarchical regression procedure, they found that the only predictors of disability were scores of Tampa Scale for Kinesiophobia and catastrophising. Unfortunately, the authors did not measure psychological indices such as anxiety and depression. This is particularly problematic since depression and anxiety are important in terms of their independent contribution to disability (Vlaeyen and Linton, 2000), and is highly associated with catastrophising (Sullivan et al., 2001). Further research is needed to confirm whether in the presence of depression and anxiety, catastrophising and fear of re-injury remain as important in the prediction of disability. Given the number of variables entered into the regression in Vlaeyen et al.’s (1995b) analyses, it seems that a study with a larger sample and considering other important variables (e.g. anxiety and depression) is needed to investigate above findings with enough statistical power.

The models of Lethem et al. (1983), Phillips (1987), and Vlaeyen et al (1995a) give fear a central role in relation to pain or re-injury. In general, these models have received empirical support from a number of studies. Recently, for example, Ciccone and Just (2001) found that pain and injury expectations collectively explained 35% to 40% of the variance in the work disability after controlling for
pain duration, depression, somatization, and current pain severity in a sample of patients with acute and chronic pain. Ciccone and Just (2001) concluded that fear-avoidance beliefs, in the form of cognitive expectancies, may influence both the duration of pain and disability. Similarly, Fritz, George, and Delitto (2001) reported that fear of pain and subsequent avoidance predicted prolonged work absence and future disability in a sample of acute low back pain patients. Fritz et al. (2001) suggested that fearful beliefs about pain might be a predispositional factor contributing to transition from acute phase to chronic phase, although further research is needed to confirm this.

While the role of fear of pain and anticipation of further pain in predicting future disability has been receiving some empirical support (Al-Obaidi, et al., 2001, 2003), the role of catastrophising, as proposed by Vlaeyen et al. (1995a, 1995b, 1999), has been questioned. For example, Crombez, Vlaeyen, Heuts, and Lysens (1999) found that disability was significantly correlated to pain-related fear and not correlated to pain intensity or negative affectivity. These findings conflict with those of other authors who have found a high association between these constructs (e.g. Rhudy, Meagher, 2001; Fritz and George, 2002). However, these apparently different findings highlight the importance of differentiating between constructs of fear, negative affectivity and catastrophising. These findings support the indirect association between disability and negative affectivity that is in consistence with previous findings that negative information appraisal and catastrophising contribute to disability. However, negative affectivity may contribute to disability through avoidance of activity, as suggested by Vlaeyen et al. (1995a). However, a more recent study by Turner, Jensen, and Romano (2000) found that catastrophising was independently associated only with depression, whereas pain beliefs, as measured by The Survey of Pain Attitudes (Jensen et al.,
(1994) and The pain Beliefs and Perceptions Inventory (Williams and Thorn, 1989), were found significantly correlated to physical disability. This model seems inconsistent with Vlaeyen et al.’s model (1995a, 1995b), which proposes a direct causal relationship between catastrophising and fear avoidance.

Further support for fear/avoidance models of chronic pain has been reported by Al-Obaidi, Nelson, Al-Awadhi, and Al-Shuwaie (2000). In a sample of 63 chronic low back pain patients, stepwise regression analyses revealed that anticipation of pain and the fear-avoidance belief, measured by Fear of Pain Questionnaire, about physical activities rather than perceived pain in itself were the strongest predictors of the variation in physical performance as measured by maximal isometric torque of the back muscles. This would be consistent with the hypothesis that sensory perception of pain does not alone explain spinal physical capacity. If Vlaeyen’s theory is correct, pain intensity should explain a significant proportion of physical performance variance in those who used to report more catastrophic beliefs. Findings that suggest catastrophising is not the only predictor of disability also support that there are other mediating or moderating variables that might be involved between injury and chronic pain related disability.

For example, Burns, Mullen, Higdon, Wei, and Lansky (2000) tested whether Pain Anxiety Symptoms Scale scores were related to behavioural performance (lifting and carrying weights). PASS scores from 98 patients were correlated negatively with amount of weight lifted and carried. Hierarchical regressions showed that PASS scores accounted for additional variance in physical performance variables (i.e. lifting and caring) when measures for trait anxiety, depression, and pain severity were controlled for. This finding allows the tentative
conclusion that an underlying anxiety-related construct contributes avoidance of painful perceived activities, independent of catastrophising.

Vlaeyen and Linton (2000) in a modified version of the fear of movement and (re)injury model, argued that pain-related fear increases hypervigilance related to threat signals and postulated catastrophising as a consequence of a primary appraisal of threat. However, they did not discuss the mechanism which links hypervigilance to catastrophising, fear of pain, and/or avoidance. Even so, they acknowledged that the constructs of negative affectivity and anxiety sensitivity overlap considerably with catastrophising.

In summary, the conceptualisation of fear of pain as central in the development of chronic pain explains more efficiently the transactional mechanism(s) from acute pain to chronic pain relative to biomedical explanations. Although the theory of fear of movements/(re)injury focuses on the importance of fear of movement and further possible injury, some issues remain unexplained. The authors do not explain why one person has a particular appraisal of pain when another does not have the same appraisal. In other words, the question is that what makes people prone to develop fear in response to pain. Vlaeyen et al. (1999) have described hypervigilance as a possible pathway. That is, hypervigilance might be the mechanism that leads to detection of pain or injury related signals, and as a result, those who fear pain may become sensitive to pain sensations and avoid them. This approach is derived from theories of fear and anxiety.

1.4.3.3 Vulnerability Model

Although fear and avoidance behaviour have long been identified as crucial factors in development and maintenance of chronic pain (Fordyce, 1976; Slade et
al, 1983; and McCracken et al, 1996), few investigations have addressed the source of this fear. Building on the previous models (Phillips, 1987; Vlaeyen et al., 1995a; Slade et al, 1983; Vlaeyen et al., 1999), Asmundson and Taylor (1996) combined the concept of specific fear of movement with concepts from the anxiety literature. Specifically, Asmundson and Taylor (1996) proposed that anxiety sensitivity, as an underlying trait, independent of pain severity, may predict the development of fear of pain in response to injury. Anxiety sensitivity refers to the fear of anxiety-related symptoms that are based on the beliefs that these sensations will have negative somatic, social, or psychological consequences (Reiss and McNally, 1985).

There is evidence that elevated anxiety sensitivity is associated with panic attacks (Taylor, 1995). Asmundson et al. (1999) generalised from such findings from research on panic and phobia to chronic pain, and conceptualised the existence of “fear of pain” as a pain phobia. Asmundson (1999) and Asmundson et al. (1999) claimed that there is an underlying pain-specific anxiety that might explain the mechanisms of fear avoidance behaviour. Based on the expectancy model of fear (Reiss and McNally, 1985), Asmundson et al. (1999) posited that anxiety sensitivity is a fundamental trait, which increases the possibility of fear reactions in response to interoceptive cues, such as breathlessness, changes in heart rate or pain. Accordingly, people who are higher in this dispositional factor are more likely to fear stimuli or situations associated to proprioception. Asmundson et al. (1999) applied two fundamental concepts (i.e. the role of attention, and distraction) from the anxiety literature and applied them to chronic pain. Asmundson suggested that fear and avoidance responses resulted from an underlying anxiety about physical sensations that led people to appraise physiological sensations as potentially threatening. This perceived threat, in turn,
primes individuals to selectively attend to pain-related cues, which in turn confirms their original fears. Therefore, anxiety sensitivity was hypothesised as a risk factor not only for the development of anxiety disorders, but also for developing high levels of fear of pain in response to injury. Asmundson et al. (1999) suggested that individuals with higher levels of anxiety sensitivity may be more likely to fear the consequences of pain sensations and therefore are more prone to pay attention to pain stimuli and avoid situations that will increase pain. According to this model, anxiety sensitivity acts as a predisposition towards the development of chronicity once an injury is sustained. As this model suggests, pre-existing anxiety sensitivity is critical in development and maintenance of chronic pain. Asmundson’s model is an attempt to account for the fact that not all the individuals who sustain an acute injury fear the associated pain, nor do the majority continue to experience pain once the injury has healed. Asmundson’s model, however, is similar to those previously proposed in considering the role of avoidance in the maintenance and exacerbation of chronic pain.

Although Asmundson et al.’s model (1999) has undergone limited empirical testing to date, the available evidence indicates that it is promising as an explanation of the role of anxiety sensitivity in pain related fear. Recently, Zvolensky, Goodie, McNeil, Sperry, and Sorrell (2001) evaluated the role of anxiety sensitivity in the prediction of pain-related fear and found that anxiety sensitivity significantly predicted variance for fear of pain, in comparison with depression and pain level. These findings are in accordance with Amundson et al.’s (1999) model. However, the correlational methodology of this study means that no causal relationship between anxiety sensitivity and fear of pain can be assumed.
In an investigation on anxiety sensitivity in relation to fear of pain in a healthy group (N=200) of adolescents, Muris, Vlaeyen, and Meesters (2001) found anxiety sensitivity was substantially and positively related to fear of pain. Although these results are consistent with previous findings (Asmundson, 1999), the cross-sectional methodology and use of healthy subjects, means that the findings cannot be generalised to the development of chronic pain in clinical patients. Clearly, further studies are needed to examine these findings in adult chronic pain patients, ideally employing a prospective methodology.

Although there is some evidence of a relationship between anxiety sensitivity and fear of pain in chronic pain patients, it is still unclear whether these findings support the fundamental assertion of Asmundson’s model. That is, Asmundson argues that anxiety sensitivity is a predisposition to the development of chronic pain states. While there is good evidence for the association between anxiety sensitivity, fear of pain and disability, the direction of influence has not been established.

Like previous theories of chronic pain (e.g. Vlaeyen et al., 1995a), Asmundson’s paradigm proposes a vicious cycle starting from injury and ending with chronic pain accompanied by disability and psychological consequences. However, he brings two additional elements to the proposed vicious cycle by Vlaeyen et al. (1995a). Specifically, Asmundson (1999) proposes that anxiety sensitivity is a determining factor, which predicts which individuals will develop fear of pain and subsequently chronic pain. Secondly, as a result of fear, he proposes that hypervigilance allows individuals to over-attend to pain, which in turn fuels the vicious cycle. The following diagram shows the core concepts of Asmundson’s model:
Figure 1.1 Asmundson’s model of anxiety sensitivity and fear of pain

If Asmundson’s model is correct, then it can be concluded that those chronic pain patients who possess higher scores on anxiety sensitivity will follow similar responses in attending to threatening stimuli to those shown in people with anxiety problems. Ascribing a substantial role to vulnerability in responding
anxiously to pain (Asmundson et al., 1999), and being hypervigilant to possible threatening stimuli in order to prevent pain (fear and avoidance) is directly analogues to models of anxiety. This part of Asmundson’s model is the critical point of his conceptualisation, but has yet to be tested fully.

As Sullivan et al. (2001) suggested, both appraisal and schema-activation models (see Barlow, 1991) predict that expectation of threat leads the individuals to focus their attention on pain and preferentially process pain-related information. Therefore, attentional focus on pain may be a critical psychological component playing a role between hypervigilance, catastrophising, and experience of pain. Figure 1.2 pictorially represents the fundamental concepts of contemporary chronic pain models, which are based on anxiety and fear-avoidance construcs (e.g. Lethem et al., 1983; Vlaeyen et al., 1995a; Asmundson et al., 1999).

![Figure 1.2 Contemporary models of chronic pain](image-url)
1.4.3.4 Anxiety sensitivity (AS) and chronic pain

Several studies provide preliminary evidence showing that AS does influence fear and other dimensions of the pain experience (e.g. appraisals) in patients with chronic musculoskeletal pain (Asmundson, Norton, and Veloso, 1999). Asmundson and Norton (1995) assessed 70 chronic back pain patients (32 females and 38 males) using a number of questionnaires including Anxiety Sensitivity Index (ASI), Beck Depression Inventory (BDI), Multidimensional Pain Inventory (MPI), and Pain Anxiety Symptom Scale (PASS)(McCracken et al., 1992). PASS is a 40-item self-report measure designed to elicit information regarding cognitive and physiological anxiety associated with pain, pain-specific escape and avoidance behaviours and general fearful appraisals of pain. The results showed a significant difference between low, medium, and high anxiety sensitivity groups on 4 variables including pain-related cognitive anxiety, fear of the negative consequences of pain, negative affect, and depression. Significant correlations were observed between AS and pain-related cognitive (r=0.62, P<0.001) and physiologic/somatic anxiety (r=0.38, p<0.001), escape/avoidance behaviours (r=0.32, P<0.01), fear of the negative consequences of pain(r=0.48, P<0.001), and negative mood (r=0.41, P<0.001). In general, the results indicated that higher AS scores were associated with higher scores on indices of negative affectivity and pain-specific cognitive anxiety and fearful appraisals in comparison with patients who had medium or low levels of AS. The groups did not differ in pain severity. These findings supported the hypothesis that AS contributes to the fear and avoidance of pain through its effects on the misinterpretation of the harmfulness of anxiety-related bodily sensations. However, the authors did not control for potentially confounding factors, most noticeably depression, which in their study was related to anxiety sensitivity. As a result, the possibility that the observed
relationship between anxiety sensitivity and other constructs is simply reflecting different mood states cannot be excluded.

Asmundson and Taylor (1996) tested the predictions that AS directly exacerbates fear of pain and indirectly, through its effects on fear of pain, increases pain-related avoidance. Participants of this study were 259 patients with musculoskeletal injuries and chronic pain complaints. They predicted that AS will increase fear of pain, and indirectly exacerbate avoidance behaviour through fear of pain, while controlling for the effects of severity of pain and avoidance behaviour (Asmundson and Norton, 1995). Patients completed a battery of questionnaires including ASI, PASS, and McGill Pain Questionnaire (sensory scale), and reported pain duration and consumption of analgesics. They used structural equation modelling to test the predicted relationships. The results revealed that AS accounted for 30% of the variance in fear of pain, which, in turn, accounted for 68% of the variance in pain escape-avoidance behaviour. Further analysis showed that pain severity accounted 13% of the variance in fear of pain in addition to AS, but failed to contribute significantly to escape/avoidance behaviour. The results of this study were consistent with the proposed pathway from AS to avoidance behaviour.

A number of studies using healthy subjects have also investigated the reliability of AS in prediction of fear of pain (Muris, Vlaeyen, and Meesters, 2001; Keogh and Birkby, 1999; Keogh and Manssor, 2001). Muris et al. (2001), for example, investigated the relationship between AS and fear of pain in 200 healthy adolescents using a revised version of AS for children and a revised form of PASS. Consistent with research on adults, it was revealed that anxiety sensitivity is “substantially” and positively related to fear of pain. After controlling for other potential predicting factors for fear of pain (e.g. trait anxiety), anxiety sensitivity
still accounted for a unique proportion of the variance in pain anxiety symptoms. These results are consistent with Asmundson (1999) who argued that anxiety sensitivity mediates fear of pain and contributes to the maintenance of pain.

Overall, there is evidence that anxiety sensitivity is a predisposition factor, which may make people susceptible toward the development and maintenance of anxiety disorders. In chronic pain context, this sensitivity to anxiety symptoms may drive individuals to fear and avoid a variety of stimuli or situations, which are associated with pain.

1.4.3.5 Anxiety Sensitivity and Hypervigilance

The relationship between anxiety and anticipation of threat, hypervigilance, and avoidance of threatening stimuli has been well-investigated and established by research in the anxiety disorders (MacLeod and Mathews, 1991; MacLeod and McLaughlin, 1995; McNally, Riemann, and Kim, 1990; Mogg et al., 1995). It has been suggested that a bias favouring threat cues during perceptual search is an enduring feature of individuals vulnerable to anxiety (Mathews, May, Mogg, Eysenck, 1990). A number of studies have indicated that anxiety is one of the most prevalent emotions associated with chronic pain (Jensen, Turner, and Romano, 1991; Large, 1996; Linton, 2000), suggesting that it is likely for the pain to become chronic through the same mechanism(s) that anxiety in anxiety disorders.

Hypervigilance to threat and the specific content of anxiety is central to theories of anxiety (Mogg and Bradley, 1998). According to Chapman (1978), hypervigilance may be mediated by cognitive factors that lead the person to search for somatic cues. For example, hypochondrical patients monitor their bodily sensations carefully, fearing that every noxious signal is indicative of a
disease (McDermid, Rollman, and MaCain, 1996). The attentional system provides the mechanism for detecting and monitoring environmental and interoceptive stimuli which are relevant to the motivational state of the individual, and facilitates a prompt response to threat cues (Mogg, and Bradley, 1998). Two studies by Ahles, Cassens, and Stalling (1987) demonstrated that young college students with a predisposition towards focusing attention to somatic symptoms reported more areas of pain and rated these sensations as more painful. Interestingly, the most frequently reported pains were in the areas commonly seen in a chronic pain population, such as back pain.

According to cognitive behavioural models (e.g. Vlaeyen et al., 1995a) of chronic pain, patients have a heightened sensitivity to pain due to their increased attention to external stimulation and a preoccupation with pain sensations (Chapman, 1978). Anticipation of pain, as a threatening signal, may force an individual to monitor his/her body to detect pain sensations. Indeed, pain has a high processing priority, and it makes sense that pain sensations take priority in information processing system (Crombez, Eccleston, and Baeyens, 1996). This view is supported strongly by evidence that level of anxiety and fear of pain may determine that to what extent a person is likely to interpret a sensation as painful and react to it (Zvolensky et al., 2001; Plehn, Peterson, Williams, 1998; Crombez, Eccleston, and Baeyens, 1996). There is also evidence that chronic pain patients with higher levels of anxiety are more sensitive and vigilant to bodily sensations (Plehn, Peterson, Williams, 1998). It has been argued that this may explain the presence of non-specific physical complaints in persons with chronic pain (McCracken, Faber, and Janeck, 1998).

Williams et al. (1988) argued that individuals with a permanent tendency to show pre-attentive, automatic vigilance to threat are more susceptible to develop anxiety
disorders when are involved in stressful situations. From this perspective, as the perceived threat increases, people high in anxiety sensitivity become more vigilant to threat. Attentional mechanisms, such as selective attention, are particularly important in the perception of threat (Keogh, et al., 2001a), and have been hypothesised to be important in the maintenance of pain. It has been proposed that anxiety sensitivity as a dispositional tendency may mediate hypervigilance and selective attention to pain cues (Asmundson, Norton and Norton, 1999). However, to date the opinion has been divided with respect to whether anxiety sensitivity adds explanatory power to other measures, such as trait anxiety.

1.4.3.6 Is Anxiety Sensitivity different from Trait Anxiety?
One of the difficulties in determining relationships between different concepts in this area of literature is the degree to which the various constructs overlap both empirically and theoretically. For example, anxiety sensitivity has shown to be moderately correlated with trait anxiety (Taylor, Koch, and Crockett, 1991), which has prompted some authors to question whether anxiety sensitivity adds anything to the concept of trait anxiety (Lilienfeld, 1996a, 1996b). Although the available evidence has generally supported the concept of anxiety sensitivity as a separate concept (e.g. Peterson and Helibronner, 1987; Taylor, Koch, and Crockett, 1991), not all findings have been in agreement (see Lilienfeld et al., 1989, 1993).

McNally (1996) argued that anxiety sensitivity is a specific tendency to respond fearfully to physical sensations associated with anxiety. He contended that anxiety symptoms are not fear provoking by themselves, unless individuals also possess an elevated level of AS. Thus, in individuals who find anxiety to be aversive, AS
is likely to be correlated with trait anxiety (Taylor, Koch, and Crockett, 1991). In other words, from this perspective it is anxiety sensitivity and not trait anxiety that determines how aversive anxiety is to an individual. Since anxiety sensitivity will lead to increased anxiety, it is not unexpected that individuals high in anxiety sensitivity will also be high in trait anxiety.

However, Lilienfeld (1996b) has argued that AS is not an independent construct. Lilienfeld, Jacob, and Turner (1989) claimed that the phenomena attributed to anxiety sensitivity are more parsimoniously explained by trait anxiety itself. Taylor, Koch, and Crockett (1991) proposed three possibilities about the potential relationship between ASI and trait anxiety. Firstly, AS and trait anxiety may measure different constructs. If so, then the items of the relevant questionnaires would be expected to load on separate factors. Secondly, if they are overlapping concepts, a simple structure should emerge with factors from both scales. Thirdly, AS may simply be an alternate measure of trait anxiety, as argued by Lilienfeld. If AS and trait anxiety are indistinguishable then in factor analytic studies one construct should emerge.

To test these hypotheses, Taylor et al. (1991) recruited 142 students with fears of spiders, and 93 psychiatric outpatients. Both the student and psychiatric sample completed measures of anxiety sensitivity and trait anxiety, but the clinical sample also completed a measure of depression. The items of the questionnaires measuring anxiety sensitivity (ASI) and trait anxiety (STAI) were subjected to a component factor analysis with oblique rotation. The best simple solution consisted of two factors that clearly distinguished between anxiety sensitivity and trait anxiety. The correlation between the two factors was .39 for the combined sample. To investigate the source of this correlation, an inter-battery factor analysis was applied. The application of an inter-battery factor analysis allows
representing the domain in common to the two scales. The results showed that anxiety sensitivity and trait anxiety were not correlated because of their item overlap, but because of their similarities in broad features such as worry and concentration difficulties.

More recently, Sandin, Chorot, and McNally (2001) provided more support for the distinction between the anxiety sensitivity construct and trait anxiety. A sample of 390 university students completed the ASI (Anxiety Sensitivity Index) and the Trait Anxiety Scale (STAI-T). The 36 items of both measurements were pooled to a component factor analysis. In accord to the work of Taylor (1996) and Taylor et al. (1991), items loaded on two distinct factors (AS and TA). Further, only two items of ASI were loaded saliently onto the STA-I factors. Sandin et al. (2001) argued that these two items are both related to psychological sensations that are related to signs of psychopathology. This study also indicated that none of the STAI items were loaded onto the ASI factor and it was concluded that ASI and STAI measure two different areas.

McNally and Lorenz (1987) demonstrated in an investigation of agoraphobia that ASI is predictive of variance in fearfulness beyond that predicted by measures of trait anxiety and argued that these measures are conceptually distinct. Asmundson et al. (1996), in their investigation on measurements of fear of anxiety in a sample of people with and without panic attacks, found that correlations between ASI and Trait Anxiety were modest but significant in panic group \( r=0.41, p < 0.01 \). This correlation was not significant in the non-panic group \( r=0.24 \). In this study, STAI-T failed to discriminate significantly between those who had panic disorder and healthy subjects without any history of panic attacks. Anxiety sensitivity was the best predictor, based on a discrimination function analysis using stepwise elimination, which classified panickers from non-panickers. At least in the case of
panic disorder, this study showed that STAI alone is not sufficient to explain the experience of panic. These results lend support to the claim that anxiety sensitivity is a correlated cognitive risk factor for the development of panic disorder (McNally and Lorens, 1987).

Lilienfeld et al. (1993) proposed that the relation between trait anxiety and AS is hierarchical in nature. They concluded that AS was a secondary factor nested hierarchically within a primary order dimension of trait anxiety. In other words they argued that trait anxiety reflects a general tendency to be anxious, whereas AS is a more specific tendency to react anxiously to anxiety sensations. To test this hypothesis, Taylor (1995b) recruited a sample of 100 subjects who completed measures of anxiety sensitivity and trait anxiety. Using factor analysis, all the items measuring anxiety sensitivity and trait anxiety were pooled to a principal factor analysis. Again two factors representing anxiety sensitivity and trait anxiety emerged. By pooling the items of Anxiety Sensitivity Index, Trait Anxiety, Fear of Negative Evaluation Scale, and a measure of Illness/Injury Sensitivity into the confirmatory factor analysis, the hierarchical model was tested and four constructs were developed: AS, TA, fear of negative evaluation, and illness/injury sensitivity. Trait anxiety emerged as the second-order factor, and fundamental fears (anxiety sensitivity, fear of negative evaluation, and illness/injury sensitivity) emerging as first-order factors. Trait anxiety was highly loaded (.85) on the fear of negative evaluation factor, whereas in contrast anxiety sensitivity had the lowest loading (.45) on trait anxiety. These results suggest that fundamental fears may differ in the degree to which they are saturated with trait anxiety. These results are consistent with Watson’s (1999) suggestion that anxiety disorders follow a hierarchical pattern. According to this model, anxiety disorders all contain a shared component, which is described as “anxious apprehension” and
represents the general negative affect associated with these disorders. Watson argued that this affect is common to anxiety disorders and is responsible for the high levels of comorbidity between anxiety disorders and depression. However, while having this common element, each individual disorder has a primary fear that determines the specific symptoms and concerns.

This hierarchical model that has received increasing support in the anxiety disorders literature, is similar to the model proposed by Asmundson et al. (1999). That is, AS is the vulnerability factor that gives rise to fear of pain/(re)injury and subsequent avoidance.

1.4.3.7 Stress-Diathesis Model
Despite the eloquence of the fear of pain/(re)injury models of chronic pain, some authors have questioned the central role given to fear over other emotional constructs, such as negative affectivity or depression (Turk, 2002; Turk and Okifuji (2002). Notably, Turk and Okifuji (2002) argued that different levels of fear may be associated with other factors such as self-efficacy. Self-efficacy was a construct that received considerable research attention historically in the chronic pain literature, but has been relatively neglected in the contemporary models previously reviewed. In response to this, Turk (2002) and Turk and Okifuji (2002) developed their own stress-diathesis model of chronic pain, in which they assign self-efficacy an important role.

Bandura (1977) proposed that a prerequisite for engagement in a given behaviour, is to have sufficient confidence in being able to do it. He argued that it is person’s self-efficacy beliefs which determine whether that behaviour will be initiated and completed. From this perspective, people who feel efficacious are more likely to
persist in the presence of obstacles and aversive consequences than those who possess less self-efficacy (Turk and Okifuji, 2002).

Turk (2002) introduced a stress-diathesis model including actual or perceived trauma which leads to fear of pain, avoidance and subsequent disability. Turk (2002) introduced anxiety sensitivity into the model as a predisposition factor, which perhaps in interaction with trauma may contribute to the fear of pain and also exacerbate the catastrophic response to pain. What is new in Turk’s (2002) model, is the role given to self-efficacy in interaction with catastrophising and disability. That is, self-efficacy interacts with catastrophising, impacts directly escape and avoidance behaviour, and as a results impacts disability. However, Turk (2002) believes that disability may impact self-efficacy as well.

While most of the factors and relationships between these factors are almost same as those proposed by Vlaeyen et al. (1995a) and Asmundson et al. (1999), self-efficacy is new to the model. Similarly, stress-diathesis model hypothesises that premorbid individual differences may modulate between injury and fear of pain. However, a critical role is given to the self-efficacy in the model, which is mediating the effects of catastrophising and escape avoidance behaviour on disability.

There is evidence to support that patients’ anticipation of pain during and following physical tasks interact with self-efficacy. Cioffi (1991) suggested that self-efficacy plays an important role in the perception of pain and its subsequent disability. Cioffi (1991) considered that pain self-efficacy affects response to pain for at least four different reasons. These include decreasing anxiety and
physiological arousal, distracts attention from threatening sensations, standing in
the face of pain, and changing the interpretation of pain.

Converging lines of evidence from investigations of both laboratory and clinically
conducted studies have provided support that self-efficacy has value in limiting
avoidance behaviour after injury and as a result decreasing the likelihood of
disability (Turk, 2002). Lackner, carosella, and Feuerstain (1996) tested the
predictive power of self-efficacy in physical functioning in chronic low back pain
patients. Lancker et al. (1996) reported that the when anticipated pain and re-
injury were controlled, self-efficacy accounted for a substantial proportion of
variance in physical functioning such as lifting, carrying, pulling, and pushing.
Arnstein, caudill, mandle, et al. (1999) found that self-efficacy partially mediates
the relationship between pain intensity and disability. Using a series of regression
analyses, Arnstein et al. (1999) in a sample of 126 chronic pain patients showed
that while pain intensity contribute to disability, lack of self-efficacy in ability to
manage pain may predict the extent to which chronic pain patients become
disabled. These results are consistent with those reported by Dolce et al (1986)
that chronic pain patients with higher levels of self-efficacy were more likely to
enhance their level of physical exercises.

However, despite trait-like factors such as negative affectivity, and anxiety
sensitivity, self-efficacy is not thought to be static, but rather modifiable in the
light of experience (Bandura, 1977). Hence, it seems that self-efficacy instead of
being considered as an etiological factor, might be better understood as a mediator
variable (Asghari and Nicholas, 2001), which could prevent an acute pain from
being translated into a chronic pain. In addition, enhancement of self-efficacy as a
result of cognitive-behavioural pain management programs has been shown (e.g.
Biller et al., 2001; Williams et al., 1996; Nicholas et al., 1992).
1.4.4 Conclusion and overview of the thesis

In conclusion, despite the fact that there are currently a number of competing theories that attempt to account for the development and/or maintenance of chronic pain, there is considerable agreement between the models. All models include an important role for fear, fear of pain/(re)injury, and subsequent avoidance behaviour. Many of the theories also posit a pre-disposing factor, such as negative affectivity, anxiety sensitivity, or self-efficacy, although the nature of the variable differs between the models.

Given the similarities between the models, it is hardly surprising that much of the literature supports various components of each model. However, in the literature, to date, no empirical study has tested between the models in a large sample of chronic pain patients to determine which of the prevailing contemporary theories, best accounts for disability associated with chronic pain.

Specifically, the question of whether the fear of pain models (Lethem et al., 1983; Vlaeyen et al., 1995a; Asmundson et al., 1999) are better supported than the model proposed by Turk and Okifuji (2002), which added self-efficacy, has not been examined. This is the aim of the first study as presented in chapter two.

In chapter 3 a critical review of the role of cognitive processing in the development of chronic pain in presented. While the content of cognitions has been investigated extensively using validated and reliable questionnaires, it is argued that the genuine underlying mechanism of information processing has been less well investigated. Chapter 3 argues that the investigation of cognitive bias in a large sample of chronic pain patients is a significant contribution to the literature.
In chapters 4, 5, and 6, selective attention to pain-related stimuli is investigated. Using a sub-set of patients described in chapter 2, who agreed to do the dot-probe test in addition to self-report questionnaires, the existence of attentional biases to pain-related words in comparison with matched non-pain words has been investigated in chapter 4.

In chapter 5, a group of healthy subjects recruited through advertising, and matched with pain-patients in terms of age, sex, and education from the sample presented in chapter 4, the clinical group and non-pain group are compared on responses to pain and non-pain related stimuli.

The aim of the study reported in chapter 6 is to examine whether attentional biases in chronic pain patients are modifiable through a CBT program. A sub-set of patients reported in chapter 4, who participated in the CBT program were followed from baseline at the beginning of the treatment to the end, and one month after the termination of treatment. Chapter 6 is designed to investigate the treatment-related changes in attentional biases over time.

Chapter 7 presents a general discussion of the findings, according to theoretical foundations, and considering the limitations that further studies would need to deal with.
Chapter Two

Structural evaluation of the contemporary psychological models of chronic pain

2.1 Introduction

As described in chapter one, the construct of fear of movement/(re)injury in chronic pain as described by Vlaeyen, Kole-Snijders, Boeren, and Van Eek (1995) has been a useful explanatory model for the etiology and maintenance of many of the common features of disabling chronic pain (see Figure 2.1). Drawing on the work of researchers and clinicians such as Lethem et al. (1983), and Waddell et al. (1993), Vlaeyen et al. (1995) argued that fear of activities perceived as pain-provoking and therefore harmful could act to inhibit a person from engaging in those activities. Successful avoidance of an activity minimises the chances of discovering that continued pain does not necessarily mean more damage and lessens the chance of disconfirming unrealistic beliefs or expectations about pain (Philips, 1987). As a consequence, avoidance of those activities temporarily reduces pain, is reinforced and contributes to increased disability. Vlaeyen et al. (1999) postulated that this response pattern was more likely in those people who were susceptible to interpreting their pain experience in a catastrophic or overly alarmist way.
In support of their model, Vlaeyen et al. (1995) cited evidence that disability and work-time lost were better predicted by fear-avoidance beliefs than biomedical factors such as pain severity or anatomical pathology (e.g., Waddell et al., 1993). However, as the relationship between fear of pain/(re)injury and disability is only moderate ($r = 0.34$, $p<.05$) (Crombez et al., 1999), which implies that fear of pain, by itself, is able to account for only a proportion of the features of those with chronic pain-related disorders. More recently, Vlaeyen and colleagues (Vlaeyen & Crombez, 1999; Vlaeyen et al., 1999; Vlaeyen & Linton, 2000) have expanded on their earlier model by including additional mediator and moderator factors, such as negative affectivity, information processing, and threatening appraisals of pain. The fearful appraisal of pain (anticipation of pain) has recently been demonstrated to be a strong predictor of engagement in physical activity (Crombez et al., 1998; Crombez et al., 1999; Al-Obaidi et al., 2000). There is also strong evidence that those pain patients who engage in catastrophic or overly alarmist styles of thinking are more likely to be distressed and disabled by their pain (Linton, 2000).
However, it is not clear why some people, but not all, develop a fear of pain when it is acute (Turk, 2002; Turk and Okifuji, 2002). A recent review by Vlaeyen and Linton (2002) listed a number of pre-existing characteristics that may account for the development of disability in response to a painful injury, including anxiety sensitivity and hypervigilance to bodily sensations. Anxiety sensitivity has been defined as a personality trait of people who are more sensitive or reactive than others to aversive sensations typically associated with anxiety (Reiss and McNally, 1985). Asmundson (1999) suggested that anxiety sensitivity might predispose individuals to developing a fear of pain and, hence, indirectly predict fear/avoidance behaviour (Asmundson & Taylor, 1996). Similar arguments have been posited for the personality construct of neuroticism (eg. Asghari and Nicholas, 1999).

Using structural equation modelling, Asmundson and Taylor (1996) found that anxiety sensitivity was a strong direct predictor of fear of pain. Fear of pain, in turn, predicted pain-related escape and avoidance behaviours better than other factors, such as pain severity. However, close inspection of the methodology employed by Asmundson and Taylor (1996) suggests that alternative explanations for their findings cannot be excluded. Firstly, Asmundson and Taylor (1996) used three subscales of the Pain Anxiety and Symptoms Scale (PASS) (McCracken et al., 1992, 1993) as indicators of the latent variable of fear of pain. The latent variable of anxiety sensitivity was indicated by subscales of Anxiety Sensitivity Index (ASI) (Peterson and Reiss, 1992). PASS subscales and the ASI are known to be highly correlated and have similar items (r= .69) (Greenberg and Burns, 2003), which makes it likely that the prediction from AS to fear of pain is at least partially due to the multicolinearity between these scales, designed to measure
related constructs. Secondly, the latent factor of avoidance comprised three variables namely analgesic use, change in lifestyle, and the escape and avoidance subscale of PASS. The avoidance construct loaded most highly on the PASS subscale (.80) relative to the two other indicators (analgesic use [.40], and change in lifestyle [.40]). Since PASS was developed to measure related constructs and to be internally consistent, the subscales of PASS are, by definition, highly intercorrelated. Hence, it remains possible that the results reported by Asmundson and Taylor (1996) are due to these high correlations and are artefacts of the method of measuring these constructs. Nonetheless, despite these potential problems this study remains as the only one, which has examined the fear of (re)injury model in musculoskeletal chronic pain patients using Structural Equation Modelling.

While Asmundson and Taylor (1996) included a number of relevant variables in their model, some constructs were notable for their absence, including depression and self-efficacy. Turk (2002) and Turk and Okifuji (2002) in their diathesis-stress model of chronic pain argued that the construct of self-efficacy might be another intervening variable, which may explain why some people become fearful and disabled by their experience of pain but not others. In their model, Turk (2002) and Turk and Okufuji (2002) suggest that once pain has become chronic, those individuals who have a low level of self-efficacy are less likely to approach activities, because they believe that they are unable to do so. As a result, those who also fear pain are unlikely to disconfirm unhelpful beliefs that pain means harm and continue to avoid, hastening the vicious cycle between pain and disability.
Self-efficacy, as proposed by Bandura (1977), is a personal belief in one’s ability to do something despite apparent obstacles. There is evidence that pain self-efficacy beliefs predict future avoidance behaviours. Asghari & Nicholas (2001), using a prospective design, reported that pain self-efficacy beliefs predicted self-reported avoidance behaviour over a period of 9-months even after controlling for the possible effects of demographic variables, pain severity, depression, disability, and catastrophising. More recently, Rudy et al. (2003) reported that self-efficacy beliefs were the strongest predictor of performance outcomes in a sample of chronic pain patients. Specifically, those who believed they could perform activities such as lifting and push-pull tasks were able to do these tasks for a longer period relative to those who did not share these beliefs. Overall, the findings from self-efficacy research with chronic pain patients suggests that self-efficacy beliefs are important. Turk (2002) argues that these beliefs may mediate the relationships between fear of movement/(re)injury, anxiety sensitivity, catastrophising and emotional distress in disabling chronic pain. However, this proposition has yet to be adequately tested.

In summary, the current literature suggests that the model of disabling chronic pain described by Vlaeyen et al. (1995) is broadly supported by evidence from clinical samples. However, the central role of fear of pain/(re)-injury beliefs in this model when a number of other cognitive and mood factors are included remains untested. Except for the study by Asmundson and Taylor (1996), the findings of most existing studies have been limited by restricted statistical power due to their small sample sizes. Associated with the use of small samples, most studies in this area have examined only a restricted range of variables in their attempts to test the Vlaeyen model. This in turn has meant they have been unable
to simultaneously examine the relationships between the variables that Vlaeyen’s theoretical model incorporates. Furthermore, the stress-diathesis model proposed by Turk (2002), which differs largely from Vlaeyen’s model in the role that it ascribes to self-efficacy has not been tested in modelling studies of chronic pain. Indeed, no study that has investigated the role between different fear-related variables in chronic pain has included self-efficacy.

Hence, the present study has two aims. Firstly, to test the Vlaeyen et al.’s (1995) model of fear of movement/(re)injury by using a range of variables that have all been shown singly, or in various combinations, to predict pain-related disability in chronic pain patients. Further, this study aims to test the role ascribed to self-efficacy in Turk’s (2002) diathesis-stress model. That is, the present study will test the proposition that self-efficacy mediates the relationship between avoidance and disability according to the guidelines of mediation conditions described by Baron and Kenny (1986). This study employed Structural Equation Modelling (SEM) to enable the simultaneous examination of interactions between the variables of interest as predictors of avoidance behaviour and pain-related disability. In addition, it will allow the relative merits of two competing models, namely the fear of pain/(re)injury model and the diathesis-stress model to be compared.

2.2 Method

*Structural Equation Modelling (SEM).* Structural Equation Modelling (SEM) was applied to evaluate Vlaeyen et al.’s (1995) and Turk’s (2002) theoretical models of chronic pain. SEM is based on covariances and is a technique recommended for large sample studies with 200 cases required for adequate analysis (Tabachnick &
SEM is a very appropriate method to examine theories or models (MacCallum & Austin, 2000) especially when researchers are dealing with a complex set of data (Kristen, 2000).

SEM is a combination of multiple regression and confirmatory factor analyses (Tabachnick & Fidell, 1996). This combination gives SEM several advantages over other standard analyses such as ANOVA, multiple regression, or factor analysis. SEM allows the relationship between multiple dependent or outcome variables to be examined; accepts mediating variables in the same single model as predictors; and has the capability to use latent constructs. Having these advantages, SEM produces loading weights and regression coefficients. SEM also involves an estimation procedure, which estimates the weight of parameters resulting in a covariance matrix to find the best solution for the hypothesised model. This solution gives a single number (fit index) indicating the degree of correspondence between the implied and observed covariance matrix. Closer matrices mean a better fit of the data to hypothesised model. The most common index of fit is the Chi-square goodness-of-fit test. A significant Chi-square means that there is a significant discrepancy between the implied and the observed covariance matrix, and in other words “poor fit” of the model to the data. Since Chi-square is very sensitive to sample size (especially large samples) and non-normality of the data, other fit indices have been introduced (Hoyle, 1995). However, all goodness of fit indices are a function of Chi-square. Given the sensitivity of the Chi-square to sample size, in addition, we will as also report other fit indices, which are less dependent on sample size including GFI (Goodness of Fit) (Joreskog and Sorbom, 1989), TLI (Tucker-Lewis Index) (Tucker and Lewis, 1973), and CFI (Comparative Fit Index) (Bentler, 1989). A value of at least .90 is required for the model to be accepted, while values over .95
are judged as “good” (Hox and Bechger, 1998). In addition to the mentioned fit indices, a relatively modern index called Root Mean Square Error of Approximation (RMSEA) have been recommended to be reported for evaluation of the model in terms of model misspecification (MacCallum and Austin, 2000). RMSEA is the most conservative fit index (MacCallum and Austin, 2000) and required to be less than .05. However, there is no general consensus on cut-off points or best index of fit, and to judge the degree of fit of a proposed model, the researcher is advised to consult different fit indices (Hoyle, 1995). Following the guidelines suggested by Hoyle (1995), the first step in SEM is to specify a model based on theory to be estimated by the available data. These data might be observed or latent variables and/or both. SEM demonstrates the hypothesised relationship between the exogenous (independent) and endogenous (dependent) variables, and these relationships might be an association, a direct, or an indirect effect. While an indirect effect describes the effect of an independent variable on a dependent variable through another mediating variable, a direct effect is a direct relationship between two variables. However, a dependent or endogenous variable may play the role of an independent variable in relationship with another variable in the model. Analysis of Moment Structure (AMOS 4.) (Arbuckle, 1999) was used to perform SEM.

2.2.1 Measures

Participants completed a battery of questionnaires.

2.2.1.1 Fear of Pain Questionnaire – III (FPQ-III) (McNeil and Rainwater, 1998)

FPQ-III consists of 30 items assessing fear of pain (FOP) on a 1-5 rating scale, with a total number between 30 and 150. FPQ-II has good reliability and validity
in pain management settings (Turk and Melzack, 2001). This questionnaire consists of three subscales including minor fear of pain, severe fear of pain, and fear of medical procedures. While other validated existing measures for fear of pain such as Pain Anxiety Symptoms Scales (PASS) (McCracken et al., 1992) exist, these measures are designed with the assumption that the individual is actively experiencing pain (McNeil and Rainwater, 1998). This orientation makes PASS inappropriate if pain free participants are compared to chronic pain patients in the same study. In addition, FPQ is shorter than PASS with forty items, and is designed to assess pain-related fear specific to particular painful situations or stimuli (McNeil and Rainwater, 1998). For the present study’s proposes, we used the total score of FPQ-III.

2.2.1.2 Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992)

The ASI is a 16-item questionnaire, rating from 0-4 with a total score from 0 to 64, which measures sensitivity to anxiety-related sensations. The factorial structure of ASI has been examined extensively, and despite a number of extracted factors in different studies (see Taylor, 1999, for a full review), the most frequently replicated factor structure consists of the following three subscales: fear of physical sensations; psychological sensations, and publicly observable sensations (Cox et al., 2001). High levels of internal consistency and good test-retest reliability have been reported (Peterson and Reiss, 1992, Asmundson, 1999). Despite the three factors of the ASI constituting different subscales, it is recommended that total scores are used rather than subscale scores due to the small number of items in each subscale (Cox et al., 2001).
2.2.1.3  Depression, Anxiety, and Stress Scale (DASS) (Lovibond & Lovibond, 1995)

Although anxiety and depression are two different concepts, quantifying these constructs using questionnaires has resulted to a high degree of overlap. Such findings have led investigators to develop measures, which are more reliable in discriminating these two domains (Brown et al., 1997). The DASS is a 42-item measure of depression, anxiety, and stress using a 0-3 scale with a range of 0 to 42 for each subscale. Normative data for DASS both in clinical samples and normal population has been published, and the reliability and validity of DASS are well established (Lovibond and Lovibond, 1995; Brown et al., 1997; Crawford and Henry, 2003). Lovibond & Lovibond (1995) have reported the Cronbach’s alpha for each subscale: anxiety = 0.84, depression =0.91, and stress = 0.90. Since subscale of stress has been shown highly correlated with both depression and anxiety subscales (.72 and .71 respectively; Crawford and Henry, 2003), in this study we used only subscales of depression and anxiety. A recent study, comparing other measures of depression, such as the Beck Depression Inventory (BDI), the Hospital Anxiety and Depression Scales and the Centre for Epidemiology Scale – Depression (CES-D), found the DASS to be the most reliable measure of depression in a chronic pain sample (Crawford, and Henry, 2003). The authors argue that this was because the DASS does not rely predominantly on somatic items, and is less likely to be inflated in a chronic pain setting.

2.2.1.4  Tampa Scale of Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990)

TSK is the most common scale for measuring pain-related fear of movement, and is suitable to be used in chronic pain population (Swinkels-Meewisse et al., 2003).
TSK consists of 17 items rated on a 4-point (1-4) Likert-type scale. Higher scores (ranges from 17 to 68) indicate more fear of pain and movement. TSK as a measure of specific fear of movement has shown a good validity and reliability with the internal consistency ranging from alpha= 0.68 to 0.80 (Vlaeyen et al., 1995). Items 4, 8, 12, and 16 of the scale need to be reversed. It is suggested that if these reverse items are deleted, the internal consistency increases slightly (Swinkels-Meewisse et al., 2003).

2.2.1.5 Pain severity

The *West Haven-Yale Multidimensional Pain Inventory* (MPI) (Kerns, Turk, & Rudy, 1985) is a multifactorial instrument designed to assess the broad domain of psychosocial variables relevant to the chronic pain experience. MPI has three sections including 12 subscales. Section one measures five important chronic pain experiences on five scales (i.e. interference, significant others, pain severity, life-control, and affective distressed). For the proposes of this research, we used the pain severity subscale of MPI to assess severity of pain, which includes three items on a seven-point scale. MPI pain severity subscale has psychometric advantages over single item indices of pain intensity, and also is a well-validated and established measurement in chronic pain literature (Melzack and Turk, 2001).

2.2.1.6 Roland and Morris Disability Questionnaire (RDQ) (Roland & Morris, 1983)

RDQ is a 24-item checklist to determine the degree to which a person is limited by pain in completing daily activities. This measure covers a range of daily activities perceived by the patients to be limited due to their pain. Total score ranges from 0 (no disability) to 24 (severe disability). In this study, we modified the wording from “my back pain” to “my pain” to cover a heterogeneous sample of chronic pain patients. Psychometric properties of RDQ after this slight wording
change have been reported (Asghari and Nicholas, 2001). In the present study Cronbach alpha was calculated at 0.92, indicating excellent internal reliability, which is similar to those reported for original version (Roland and Fairbank, 2000). RDQ has good psychometric properties (Roland and Fairbank, 2000).

2.2.1.7 The Pain Responses Self Statements (PRSS) (Flor et al, 1993).

PRSS, an 18-item questionnaire, assessing active coping with pain and pain catastrophising (nine items each), was included to assess catastrophic pain related cognitions. Psychometric properties of PRSS have been established (Flor et al., 1993). While there are other measures of catastrophising (e.g. catastrophising sub-scale of Coping Strategies Questionnaire; Rosentiel & Keefe, 1983), they do not differentiate between cognitive schemata and automatic thinking that are assumed to underlie emotional disorders (Flor et al., 1993). PRSS is developed to assess situation-specific cognitions that either promote or hinder attempts to cope with pain.

2.2.1.8 Pain self-efficacy Questionnaire (PSEQ) (Nicholas, 1989)
PSEQ was administered as an index of patients’ self-efficacy measuring their level of confidence that they are able to do a range of daily activities despite the pain. The PSEQ contains 10 items rated on a 7-point rating scale where zero equals “not at all confident” and 6 equals “completely confident”. Scores on the PSEQ may range from 0 to 60, with higher scores indicating stronger self-efficacy beliefs. The reliability and validity of the PSEQ are good (Asghari & Nicholas, 2001). Sensitivity of the PSEQ to treatment has been reported by Nicholas et al., (1992) and Williams et al.(1996) and it remains the only validated measure of self-efficacy in a chronic pain population.
2.2.2 Subjects

Over a period of nine months, 232 consecutive eligible chronic musculoskeletal pain patients referred to the Pain Management and Research Centre at the Royal North Shore Hospital, University of Sydney were approached and invited to participate in the study. Of the 232 eligible patients, 217 agreed to participate in the present research. Six patients later declined, and 4 patients returned incomplete questionnaires and so 207 (recruitment rate: 89%) patients are included in the present study.

To be eligible, patients had to be over 18 years of age, in pain for at least 3 months, have sufficient command of English to read and answer the questionnaires appropriately. Patients were excluded if they had severe psychological disorders (such as psychoses), and drug or alcohol problems. Patients were approached during their initial assessment at the pain clinic, and informed of the research proposal. Those who agreed to participate in the study signed the consent form.

2.2.3 Structural Models

Model 1. We proposed a model based on Vlaeyen et al.’s fear of pain/(re)injury model as depicted in Fig. 2.2, to test the role of negative affectivity and pain-related fear and avoidance as predictors of disability in chronic pain patients. We hypothesised two latent variables (LVs, shown by ellipses) as the constructs of fear of pain/avoidance, and negative affectivity. Derived from the Vlaeyen model, three measured variables (MVs, shown by rectangles) were considered to reflect the construct of fear/avoidance. They were: fear of movement (TSK), fear of pain (FPQ-III), and catastrophising (PRSS.cat). Another latent variable labelled
negative affectivity was constructed by three indicating variables measured by anxiety (DASS-A), anxiety sensitivity (ASI), and depression (DASS-D). Two paths were considered from the latent variable of negative affectivity to pain avoidance, and to disability. Another path also was proposed from pain avoidance to disability. The measured variable of pain severity was considered to predict directly the variable of disability. Therefore, a path from “pain” to “disability” was considered. However, since pain severity has been suggested itself to be affected by so-called predisposition factors such as negative affectivity and anxiety sensitivity (Asmundson et al., 1999; Vlaeyen et al., 1999), another path from negative affectivity to pain severity was introduced into the model. Variables labelled r1-r8 are residuals corresponding to the variance in the MVs not accounted for by LVs. Z1 is the equivalent of the constant in regression analysis.

Figure 2.2 Structural model as predicted based on Vlaeyen et al. (1995) Theory. Paths marked with “1” are fixed to identify the model. Neg/aff: Negative affectivity (LV);
Avoidance: Fear avoidance (LV)
Duration of pain was not included in the model because it has been argued that demographic factors including duration of pain, when patients have been in pain for a long time, is not a significant factor (Turk and Okifuji, 2002).

**Model 2.** Based on the proposition that high self-efficacy can mediate the effects of anxiety, and enable a person in pain to perceive a potentially threatening physiological sensation in a less fearful way, and prepared to confront (or engage in) activities expected to be painful (Turk, 2002; Turk and Okifuji, 2002; Cioffi, 1991; Philips and Jahanshahi, 1986), we developed a model to test the possible mediating role of pain self-efficacy on pain-related fear/avoidance behaviour, consistent with Turk’s diathesis-stress model of chronic pain. In this model we did not include negative affectivity for two reasons. First, Turk and Okifuji (2002) did not include it explicitly in their model, and secondly, the argument is that those people who have higher self-efficacy cope with pain better than others, even when other factors are controlled (Asghari and Nicholas, 2001, 2003).

As described in model 1, a path from the latent variable of pain fear/avoidance, as indicated by measured variables of FPQ, TSK, and PRSS.cat., was hypothesised to predict the dependent variable of disability (RDQ). In addition, the independent variable of pain self-efficacy (PSEQ) was hypothesised as a predictor of both avoidance behaviour and predictor of perceived disability. Figure 2.3 shows the hypothesised model. Variables indicated as R1-R4 are residuals of measured variables, and Z1 is the equivalent with constant in regression analysis.
Figure 2.3 depicts predicted relationships based on model 2. Paths marked with “1” are fixed to identify the model. R1-R4: residuals; Z1: equivalent with constant in regression equation; FPQ: Fear of pain questionnaire; TSK: Tampa Kinesiophobia Scale; PRSS-CAT: catastrophising subscale of PRSS; Avoidance: Fear/avoidance of pain (LV); PSEQ: Pain self-efficacy questionnaire; RDQ: Roland & Morris disability checklist.

2.3 Results

2.3.1 Participants

Participants were 207 chronic musculoskeletal pain patients (110 females, 53.1%). The sample was heterogeneous, with a majority of patients experiencing chronic low back pain (34.3%), lower limbs (15.2%), or upper shoulders/upper limbs (14.6%). The average duration of pain in the sample is 75 months (SD: 99.4), and all participants were taking analgesic medication. The mean age of the sample was 45.2 (SD: 13.6), with 26% having university degree, 35.4% were educated for at least 11 years or more, and 38.6% had less than 11 years education. However, 51.3% of the patients were unemployed, and only 18.3% were working full time. A majority of the patients were receiving financial compensation (44.4%), mostly married (56.6%), followed by 22.2% who were single, and the remainder were
separated, divorced, or widowed. Patients’ characteristics are similar to previous reported samples by other studies at the same setting (Sharp and Nicholas, 2000; Asghari and Nicholas, 2001) and also in international research (e.g. Turner et al., 2000; McCracken, Gross, and Eccleston, 2002).

2.3.2 Self-report data

The average scores on the DASS indicated that, as in other samples of chronic pain patients, our sample presented with mild to moderate depression, anxiety, and stress as compared to a healthy population (Crawford and Henry, 2003), and were clinically distressed (Brown et al., 1997). On average, this sample reported a moderate level of pain severity, and moderate levels of disability. The scores of self-report measures for the sample are shown on table 2.1.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disability (RDQ)</td>
<td>11.45 (5.5)</td>
</tr>
<tr>
<td>Pain severity (MPI)</td>
<td>4 (1.1)</td>
</tr>
<tr>
<td>Catastrophising (PRSS)</td>
<td>2.64 (1)</td>
</tr>
<tr>
<td>Pain self efficacy (PSEQ)</td>
<td>26.2 (13.4)</td>
</tr>
<tr>
<td>Fear of movement (TSK)</td>
<td>39 (8.5)</td>
</tr>
<tr>
<td>Anxiety sensitivity (ASI)</td>
<td>18.8 (11.4)</td>
</tr>
<tr>
<td>Depression (DASS D)</td>
<td>15 (12)</td>
</tr>
<tr>
<td>Anxiety (DASS A)</td>
<td>9.2 (8.2)</td>
</tr>
<tr>
<td>Fear of pain (FPQ)</td>
<td>67 (18.4)</td>
</tr>
</tbody>
</table>

Table 2.1 shows the means and (SDs) for self-report questionnaires
A correlational analysis was conducted to investigate the inter-correlation between measures used in the specified models (Table 2.2).

<table>
<thead>
<tr>
<th></th>
<th>MPI</th>
<th>PRSS</th>
<th>PSEQ</th>
<th>TSK</th>
<th>ASI</th>
<th>FPQ</th>
<th>DASS-D</th>
<th>DASS-A</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDQ</td>
<td>.56**</td>
<td>.36**</td>
<td>-.60**</td>
<td>.46**</td>
<td>.25**</td>
<td>.09</td>
<td>.46**</td>
<td>.40**</td>
</tr>
<tr>
<td>MPI</td>
<td>.37**</td>
<td>-.47**</td>
<td>.37**</td>
<td>.23**</td>
<td>.09</td>
<td>.43**</td>
<td>.49**</td>
<td></td>
</tr>
<tr>
<td>PRSS</td>
<td>-.45**</td>
<td>.48**</td>
<td>.42**</td>
<td>.28**</td>
<td>.60**</td>
<td>.49**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSEQ</td>
<td>-.53**</td>
<td>-.22**</td>
<td>-.04</td>
<td>-.58**</td>
<td>-.43**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSK</td>
<td></td>
<td></td>
<td>.31**</td>
<td>.27**</td>
<td>.42**</td>
<td>.37**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td></td>
<td></td>
<td></td>
<td>.41**</td>
<td>.48**</td>
<td>.59**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FPQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.14</td>
<td>.18*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASS-D</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.64**</td>
<td></td>
</tr>
</tbody>
</table>

Table 2.2 Inter-correlation between measurements

FPQ: Fear of pain total score; ASI: Anxiety Sensitivity Index; DASS-D Depression, DASS-A Anxiety, TSK Tampa Kinesiophobia, Catastrophising (PRSS), PSEQ: Pain self-efficacy Questionnaire, MPI: MPI subscale of pain severity, RDQ: Roland & Morris Disability Questionnaire. *p<05; **p<01

In first phase, model 1 was tested. This model resulted in a significant chi-square ($\chi^2$ [15]= 32, P=.004), however, other fit indices suggested a goodness-of-fit index (GFI) of .96, Tucker-Lewis Index (TLI) of .95, Comparative Fit Index (CFI) of .97, and RMSEA .07. In order to improve the model, we removed the path from
negative affectivity to disability, which was not significant in the original model. In addition, we had to let the residual of ASI be correlated with residuals of MPI-pain severity, FPQ, and DASS-A; also the residual of DASS-D needed to be correlated with PRSS-CAT. MacCallum (1995) and Hoyle (1995) recommend that any modification should be theoretically justified. These modifications do not violate the theoretical foundation of the model, since Vlaeyen et al. (1995) did not propose a direct relation between negative affectivity and disability, although statistically measures of negative affectivity have been found associated with disability. In addition, the residuals of variables of anxiety sensitivity and severity of pain were allowed to be correlated, because of the somatic-related elements of both measures and also possible conceptual relations anxiety and pain. These modifications improved the model and decreased the chi-square although it remained significant ($\chi^2 [14]= 24.6, p=.04$). Other fit indices also improved indicating an “acceptable” to “good” model fit with GFI=.97, TLI=.96, CFI=.98, and RMSEA =.06 (Figure 2.4). All path coefficients were significant at the level of .001 or greater. According to the final model depicted in figure 4, it can be seen that 1 SD increase in negative affectivity was associated with a .80 SD increase in pain fear/avoidance. Also 1 SD increase in pain fear/avoidance results in an increase of .38 SD in disability. It is indicated by the model that 1 SD increase in pain severity is associated with .37 SD increase in disability. In addition, this model suggested that the report of the level of pain severity is associated with the level of negative affectivity; so that a 1 SD increase in negative affectivity, increases the pain severity by .59 SD. Based on the model, pain fear/avoidance and pain severity accounted for 42% of variance in disability. Negative affectivity accounted for 65% of variance in pain fear/avoidance, and 35% of variance in pain severity.
Figure 2.4 Shows the standardised path coefficients for the variables in the model 1. (all coefficients are significant, p<.001). R1-R8: residuals; Z1: equivalent with constant in regression equation; FPQ: Fear of pain questionnaire; TSK :Tampa Kinesiophobia Scale; PRSS-CAT: catastrophising subscale of PRSS; Avoidance: Fear/avoidance of pain (LV); PSEQ: Pain self-efficacy questionnaire; RDQ: Roland & Morris disability checklist. Pain: MPI pain severity subscale; ASI: Anxiety sensitivity scale; DASS-A & DASS-D: Anxiety and depression subscales of DASS respectively.

In phase 2, model 2 derived from Turk’s diathesis-stress model was tested (as shown in Figure 2.3). The chi-square statistic of this model indicated rejection ($\chi^2[4]= 15.5, p=.004$), as were TLI=.89, and RMSEA=.11; but GFI =.97 and CFI=.96 were “good”. Inspection of regression weight revealed that FOP relative to the...
other two indicator variables in the latent variable of pain fear/avoidance was not sufficiently strong to be retained within the model. Therefore, we removed the fear of pain (FPQ) variable from the model.

The final model consisted of two indicator variables for pain fear/avoidance as shown in Figure 5.5 Fit indices for this model were excellent with all fit indices showing a good degree of fit to the model: chi-square (1) = .20, p=.66; GFI=1; TLI=1; CFI=1; and RMSEA=.00. Overall, the final model accounted for 41% in disability, and pain-self efficacy accounted for 63% of the variance in pain fear/avoidance variable. This model also suggests that elevated pain self-efficacy is associated with decreased pain-related avoidance. Indeed, 1 SD increase in pain-self efficacy is associated with .70 SD decrease in pain-related fear and avoidance. A similar association was revealed between pain self-efficacy and disability. One SD increase in pain self-efficacy results in .36 SD decrease in disability.
Figure 2.5 shows the structural model of pain fear/avoidance and disability and the impact of pain self-efficacy (All coefficients are significant, p<.001).

We tried to include self-efficacy in Vlaeyen’s model to test a combination of both models to determine whether additional variance in disability could be accounted for in a more comprehensive model of chronic pain. Hence, a third model was developed including two latent factors and eight observed variables. The structure of the negative affectivity (neg/aff) latent factor is built on the findings of model one and the structure of the avoidance latent factor is extracted from model two. Based on the findings from second model, pain self-efficacy was included with the same paths to avoidance and disability. Figure 2.6 reflects the path coefficients for the variables in model three. The name of the variables and residuals are the same reported in models one and two. The chi-square statistic of this model indicated rejection ($\chi^2 [14]=84; p=0.0001$), as RMSEA indicated (0.15) as well. However, other fit measures were acceptable (CFI= 0.98, TLI= 0.95).
Modification indices suggested correlating PSEQ with observed variables of disability, depression, and anxiety sensitivity, which while improving model, violates the theoretical assumptions. Hence, model was reported without further modifications.
Figure 2.6 reflects a model based on the combination of models one and two.
2.4 Discussions

The present study was designed to test two models of chronic pain. The fear of pain and movement/(re)injury model developed by Vlaeyen et al. (1995) and the diathesis-stress model proposed by Turk (2002). The results of the study largely supported both models.

As Vlaeyen et al. (1995) have postulated, the experience of pain in conjunction with negative affectivity is associated with maladaptive avoidance of pain and movement/(re)injury, which was, in turn, associated with increased disability. More specifically, distress and negative affectivity predicted pain-related avoidance behaviour. That is, when residuals were allowed to be correlated, model 1 supported two latent variables of negative affectivity and avoidance. Negative affectivity was comprised of anxiety sensitivity, anxiety and depression. Fear/avoidance was comprised of TSK, FPQ and catastrophising. The model suggested that negative affectivity was associated with avoidance, which was in turn associated with disability. While negative affectivity had only an indirect relationship with disability, fear/avoidance accounted for 42% of variance in disability directly. The only other variable to show a direct relationship with disability was pain severity. This largely supports the central tenets of Vlaeyen et al.’s (1995) model.

This finding is consistent with the results of Asmundson and Taylor (1996), who using a similar methodology, showed that negative interpretation of painful sensations predicts further fear of pain, which indirectly contributes to pain-related avoidance. However, the present study added to the model in examining
the relationship between fear avoidance and disability. Our results confirm that high levels of avoidance behaviour are associated with increased disability. In addition, while “pain severity” directly impacts upon disability, pain severity, as predicted, is associated with distress and negative affectivity. These results are consistent with the literature. McCracken et al. (1998) found that physical pain complaints are manifestations of pain-related distress. Specifically they found that, in addition to depression and anxiety, physiological symptoms of pain-related anxiety were significant predictors of physical complaints. This is consistent with the argument that painful movement develops due to anticipation that pain facilitates fear of anxiety symptoms (Greenburg and Burns, 2003). In general, the finding that there is a mutual relationship between sensitivity to pain and reported pain severity is now well established (Hadjistavropoulos, Asmundson, LaChapelle, and Quine, 2002; Eccleston, Crombez, Aldrich, and Stannard, 2001; Crombez et al., 1999).

Given the important role that negative affectivity plays in both perception of pain, and pain-related avoidance, it was interesting that the present results indicated no direct effect from the latent factor of negative affectivity on disability. Indeed, it was found that negative affectivity contributes to the development of disability through increasing pain perception and avoidance behaviour. These findings are in agreement with the role of negative affectivity as a possible dispositional factor in developing pain-related fear/avoidance as argued by Asmundson et al. (1999) and facilitating avoidance of painful movements (Vlaeyen et al., 2002).

The second model tested the mediating role of pain self-efficacy on the association of avoidance and disability as proposed by Turk (2002). That is,
higher levels of self-efficacy are associated with decreased avoidance and report of disability. Accordingly, the latent variable of fear/avoidance, comprised of fear of movement and catastrophising, directly predicted disability. When self-efficacy was introduced to the model, the level of association between avoidance and disability decreased, although all three paths from self-efficacy to avoidance, from avoidance to disability, and from self-efficacy to disability were significant, fulfilling the requirements for partial mediating role suggested by Baron and Kenny (1986). As mentioned before, Vlaeyen’s model (1995) is not clear enough in explaining why some fearful chronic pain patients approach painful activities despite pain. Pain self-efficacy might be a potential factor, which is claimed to modify the interaction between fear/avoidance and disability (Turk and Okifuji, 2002). These results provide further evidence for the fact that despite being anxious about engagement in physical activities, people who believe that they can control their pain are better able to tolerate and cope with pain that occurs as a result of activity (Bandura, 1992). Even in the absence of negative affectivity, fear of movement/(re)injury and self-efficacy accounted for almost same amount (41%) of the variance in disability.

Indeed, in the present data set, Turk’s (2002) model has advantages over Vlaeyen’s model. Firstly, while accounting for a similar proportion of variance, Turk’s model includes fewer variables and as such is more parsimonious. In addition, the model did not need to correlate the residuals in order for it to be accepted. Therefore, Turk’s model is also statistically superior. One explanation for the superiority of Turk’s model over Vlaeyen’s model is the degree to which each model emphasises the development and/or maintenance of chronic pain. The focus of Vlaeyen’s model is on the development of chronic pain. That is, it
provides an explanation for the transition from acute pain to chronic pain. On the other hand, Turk’s model describes the maintenance of chronic pain. They argue that patients already in pain with high self-efficacy are able to resume activities despite pain. As a result, there is a significant decrease in avoidance behaviour and subsequently disability. Since the current sample, was a sample of patients already in chronic pain, this may be the reason that Turk’s (2002) model was a better fit with the data than Vlaeyen’s model which may have more relevance earlier in the development of chronic pain.

The results of the study supports the fact that in chronic pain samples, pain self-efficacy is a mediating factor that may impact upon fear of movement and pain, and the level of pain-related avoidance. That is, higher levels of self-efficacy may decrease avoidance and disability. These results are consistent with studies indicating that pain self-efficacy is a stronger predictor of measures of physical functioning relative to other psychological factors, such as anxiety (Lackner et al., 1996, Lackner and Carosella, 1999, and Arnstein et al., 1999).

Turk (2002) and Turk & Okifuji (2002) argued that although being fearful of pain may increase the likelihood of the development of physical disability and further psychological distress, it does not necessarily imply that this will happen (Lackner, Carosella, and Feuerstein, 1996). It has been suggested by research that being fearful of pain is not necessarily equivalent to avoiding painful activities. Further, it has been argued that patient’s level of function (e.g. lifting) can be more accurately determined by functional self-efficacy rather than perceived pain or anxiety (Lackner and Carosella, 1999). The present results, which assessed both self-efficacy and pain fearfulness are consistent with these results. Fear of
pain/movement may determine who avoids activity and becomes disabled. However, once disability has developed (as in chronic pain samples) patients’ self-efficacy becomes more important. Those who feel confident that they are able to “keep going” despite pain, perhaps are more likely to accept pain and manage it.

The present study has a number of limitations that should be considered when interpreting the results. Although we applied structural equation modelling for analyses, due to the cross-sectional design of the study, the direction of causality can only be inferred from paths in the model. In addition, as a result of shared variance between measures, we had to allow some of the residuals in the first model to be correlated, which some of the experts would not agree with. This is an issue that is typically neglected in the literature and is rarely reported. While some view correlating residuals as unacceptable due to conceptual similarities. Further, given the possibility of shared items, especially in measures of mood and anxiety related constructs, overlap between the residuals is not surprising (G. Fogarty; personal communication). However, in the second model, negative affectivity measures were not introduced into the model. In this model, residuals did not need to be correlated, allowing the model to be accepted without violating statistical assumptions.

A further limitation of the study is that we tested the role of self-efficacy in a separate model from negative affectivity. It was decided to test the two models separately. Vlaeyen’s model (1995) does not include self-efficacy as a variable, and it was not obvious where to place it. Similarly, Turk’s model does not explicitly refer to negative affectivity. Therefore, the present study provided a test
of two competing models. Nonetheless, we tried to test the combined model and it was not supported. It seems that the conceptual overlap of self-efficacy with disability (as measured by PSEQ and RDQ) as suggested by the modification indices, is the most important problem that has to be resolved in future studies. Further studies using different measures of pain self-efficacy and disability might be a suitable approach to examine the issue. Hence the present study indicated that combining concepts (notably negative affectivity and self-efficacy) from both models would not explain more of the variance than either did when tested in isolation. This is most likely due to the high inter-correlations between measures of negative affectivity (notably anxiety and depression) and self-efficacy. However, the results do support the major tenets from two similar, but differing models.

Acknowledging the mentioned limitations, this study using an adequate sample, well-validated measures, and a methodology, which has advantages over regression analysis, provided further support for the role of fear/avoidance in chronic pain as conceptualised by Vlaeyen et al. (1995). In addition, in a tentative modelling, suggested self-efficacy as a factor that may explain how some people may take a way out of the vicious cycle of injury –disability, as suggested by the diathesis-stress model (Turk, 2002). Future research needs to replicate the study with combining both models in a single model. To do this, we would suggest choosing measures, which have the less possible item overlap. A preliminary component factor analysis might be helpful to clarify underlying factor(s) of each measure to prevent the problem of shared items. In addition, to be able to infer causal relationships through modelling, these models should be tested using prospective studies in both acute and chronic pain populations. It may be that the
factors associated with disability in an acute pain sample differ from those in a chronic sample. Such studies will determine their relative strengths in terms of development and/or maintenance of chronic pain.

Although the present study confirmed the importance of anxiety and fear-related factors (e.g. fear of pain, anxiety sensitivity), and self-efficacy in chronic pain, the mechanism(s) through which fear and avoidance impacts on disability were not addressed. Vlaeyen et al (1995a) suggest that fear, as in anxiety disorders, promotes hypervigilance towards pain, which in turn, increases avoidance and subsequent disability. Similarly, Turk (2002) suggested that anxiety and fear of pain might facilitate attending to pain through sensitisation. However, people with higher levels of self-efficacy may manage to confront pain despite the existing sensitivity. In both models, hypervigilance and sensitisation to pain are mentioned as possible mechanisms that mediate the relationship between fear of pain and avoidance and subsequent disability. In the three following chapters, the role of hypervigilance and attentional bias to pain-related information as a possible mechanism for the maintenance of pain will be addressed.

Contemporary models of chronic pain, with slight differences, recognise the role of cognitive vulnerabilities in development of chronic pain, and argue that the experience of pain affects the cognitive processes in favour of processing pain-related information (Crombez, Eccleston et al., 1996). Fearing pain is expected to result in hypervigilance to pain and giving priority to process pain-related stimuli at the expense of non-pain-related stimuli. In the ensuing chapters this phenomenon will be examined both in chronic pain patients and the pain-free population. In a further step, since it has been found that cognitive distortions can
be treated using cognitive behavioural techniques, the effect of CBT on attentional biases in chronic pain patients is addressed.
Chapter Three

Cognitive biases
The role of information processing in maintenance of emotional disorders has been the subject of a number of psychological theories. According to both Bower’s (1981) network model and Beck’s (1997) schema model, anxiety and depression should be associated with emotionally congruent biases in all aspects of information processing, including memory and attention. There is a good deal of evidence to support the existence of these information processing biases in anxious and depressed people (Mogg, Miller, and Bradley, 2000; Dalgleish et al., 2001; Heinrichs and Hofmann, 2001; Wells and Matthews, 1996; McNally, 1995; Williams, Mathews, and MacLeod, 1996; Mathews, Mogg, Kentish, and Eysenck, 1995; Bradley et al., 1998).

Recently these paradigms have been utilised in attempts to explain aspects of chronic pain. Although current theories of chronic pain differ slightly in terms of the factors they focus on, all recognise the role of cognitive vulnerability factors in development of chronic pain. Cognitions such as catastrophising (Turner et al., 2000), hypervigilance (Turk and Okifuji, 2002), and attention to pain (Eccleston, 1995), are the most commonly cited cognitive mechanisms for maintenance of disability and distress associated with chronic pain. These cognitions have been extensively investigated using validated and reliable questionnaires (Turk and Melzack, 2001). However, whether cognitive vulnerabilities can be measured by self-report questionnaires has been questioned (Pincus and Morley, 2002). Contextual issues often surround psychological assessment of chronic pain patients. For example, patients may be involved in a Workers' Compensation claim (Robinson et al., 1997). As a result, there might be motivation to respond to self-report instruments that is influenced by more than nociception. Self-report
measures may be considered to reflect the “product” of cognitive bias rather than the genuine underlying mechanism of information processing (Pincus and Morley, 2002). This product may be the subject of distortion through selectively processed information, semantic memorising, and interpretations that are in agreement with the pre-existing cognitive schema. Hence, Pincus and Morley (2002) argued for measures of cognitive information processing rather than content that minimise such distortions. They suggested that self-reported questionnaires are limited by allowing access to only the conscious level of information, and not to the schematic level, which is automatic (Pincus and Morley, 2002).

3.1 Selective memory in chronic pain

It has been argued that the experience of pain affects the cognitive processes in favour of processing pain-related information (Crombez, Eccleston et al., 1996). Current theoretical models propose that individuals with chronic pain will give preference to the processing of pain-related information over information that is not pain-related.

Edwards, Pearce, Collett, and Pugh (1992) hypothesised that like the mood congruity effects demonstrated in affective disorders, a subgroup of chronic pain patients may exhibit similar biases. They studied selective memory for sensory and affective pain-related information in chronic pain patients with and without depression, depressed psychiatric patients, and a healthy control group. A recall test consisting of sensory pain, affective pain, and neutral words were selected and matched for frequency. This test was followed by a recognition task, where the words of the recall test were randomised with an equal number of new adjectives matched for word type and frequency. Comparisons revealed specific recall biases for sensory adjectives in non-depressed chronic pain patients compared to the
non-pain control groups. Further, recall biases for both sensory and affective words in depressed chronic pain patients compared to the other groups were observed. These results suggested that the experience of pain is associated with memory biases for sensory pain words only. However, the experience of pain and depression is associated with an additional bias towards pain affective words.

Edwards, Pearce, and Beard (1995) in a further investigation on the source of the selective memory in chronic pain patients, found that biased information processing in this group is more likely to be a function of pain intensity than emotional state (i.e. depression and anxiety). Utilizing a longitudinal design, twenty-four women with chronic pelvic pain undergoing hysterectomy and oophorectomy were recruited the day before the operation. They were reassessed two and six months later. The patients completed a memory recall task, the Beck Depression Inventory (BDI), and pain intensity ratings on a Visual Analogue Scale. On each occasion four different lists of words were aurally presented to patients, including sensory, affective, neutral, and gardening words matched for frequency and length. Significant reductions were reported in pain intensity between pre- and post-treatment as a result of surgery. Recall time for each word type was recorded. Despite recalling more pain-related words before surgery, six months after surgery patients recalled fewer pain-related words and more neutral words in comparison with pre-surgery interview. However, this difference failed to reach statistical significance, which most likely reflects the lack of statistical power. Since this operation results in a high probability of becoming pain-free within a few weeks after surgery, the authors argued that these results are inconsistent with the hypothesis that processing of pain-related information is a cognitive vulnerability toward developing chronic pain, and suggested that the experience of pain itself plays a key role in memory of pain.
Although Edwards et al. (1995) discussed the relationship between memory and depression and measured level of depression, they did not control for the baseline level of depression. In addition, the sample size was small, especially at follow up, which limited the statistical power of the study. Further, lack of a control group, which did not have the operation, means the possibility that the changes observed for memory bias reflect the process of time could not be excluded. Whether these findings can be generalised to musculoskeletal pain, especially where no physically identifiable problem has been found, is also unclear (Rode, Salkovskis, and Jack, 2001). Recently, Wells, Pincus, and McWilliams (2003) provided evidence that receiving a diagnosis in chronic pain patients predicted a recall bias away from depression adjectives relative to chronic pain patients who did not have a diagnosis. Suggesting that the vagueness of chronic pain, by itself may contribute to cognitive bias, at least in those chronic pain patients who have not received any clear explanation about their pain.

Pincus, Pearce, McClelland, and Isenberg (1995) compared the responses of depressed pain patients, non-depressed pain patients and pain-free control subjects on a free recall task. Depression-related words, pain-related words, and neutral words were presented in both positive and negative valance, and as either self-referent or other-referent. The self-referent pain-related words but not depression self-referent words were recalled selectively by depressed pain patients, suggesting that the primary issue might be “pain” rather than mood.

Overall, the association between recall biases and self-report measures has generally been supported. However, Pincus and Newman (2001) in a recent study investigated the relationship between biased recall and costs in chronic pain in back pain patients. They hypothesised that patients who recall more pain-related stimuli would be more likely to use more health care facilities. Using a similar
paradigm to Pincus et al. (1995), the percentage of recalled pain-related words relative to total words was calculated as the measure of biased recall. A significant correlation between total cost of back pain and recall bias was found. Hierarchical regression revealed that recall bias significantly predicted health care utilisation. The relationship of a bias measure and objective measures of health care utilisation is particularly impressive as health care use reflects a behaviour that might not be expected to be predicted by a measured cognitive bias.

In general, Research conducted to date supports the view that chronic pain patients show recall biases to pain-related words, but the pattern of these biases is not consistent. Pincus et al. (1993) concluded that these biases are evident mostly in self-referential conditions. They showed that pain patients recall more sensory words relative to neutral words when the words were presented in a self-referential (e.g. referring to yourself) rather than other-referential condition (e.g. referring to your friends). Similar findings have been reported among those rheumatoid arthritis patients who were depressed when compared with non-depressed RA patients or healthy controls (Clemmey and Nicassio, 1997). Pincus and Morley (2001) suggest that a history of depression prior to the development of pain is a factor that should be considered in research on recall biases in chronic pain patients. Although most of the research on selective memory in pain patients has found a relationship between pain and memory for pain-related stimuli, it remains unclear whether these biases are a result of pain or have some etiological significance. Pincus and Morley (2001) argued that cognitive biases in chronic pain would be better explained in the form of an enmeshment model comprising variables of pain, illness, and self. This model is well consistent with findings of research on memory biases in chronic pain patients.
While the studies reviewed above support a memory bias in chronic pain patients for pain sensory words, the source of memory biases and their theoretical significance is unclear. Memory is highly state dependent (Williams et al., 1997). That is, we recall information consistent with our emotional state at the time we learned the information. Only Edwards et al. (1995) have designed research, which allows some understanding of the direction of causality by the suggestion that as pain disappears the observed recall biases may decreased as well. Their results favoured the interpretation that it is the presence of pain, which is associated with memory bias, at least in people with chronic pelvic pain.

The other issue with the memory bias literature, is that it is unclear exactly which cognitive biases are involved. That is, a memory bias can result if a person (a) gives a stimulus more attention leading to better encoding; (b) has a more elaborated schema relating to that information; and/or (c) whether the information is more easily retrieved. The mechanisms through which memory biases develop have both theoretical and/or clinical implications. Theories of chronic pain argue that cognitive biases represent hypervigilance towards pain. That is they predict that the cognitive bias is at the attentional phase of processing. Accordingly, existence of pain may prioritise attending to pain-related information, and contribute to memorising and retrieving pain-related stimuli at the expense of ignoring non-pain-related information. therefore it is of more theoretical interest whether chronic pain patients demonstrate selective attention to pain-related stimuli, given the central role of pain-related fear and subsequent hypervigilance in current theories of chronic pain.
3.2 Selective attention

Cognitive theorists have postulated that people with anxiety possess fear-related cognitive structures which enhance their attention to fear-related information (Williams et al., 1997). These structures are argued to increase the attention to fear-related information at the expense of other information. Such a pattern of information processing has been well documented and clearly shows that anxious people exhibit attentional bias towards the source of perceived threat (Williams et al., 1997). Chronic pain patients frequently report anxiety. This co-existence of pain and anxiety, together with recent theories that emphasise the role of fear in the development of pain-related disability (Lethem et al., 1993; Vlaeyen et al., 1995a), has led some authors to conclude that similar attentional biases might be important in disabling chronic pain (Asmundson et al, 1997; Asmundson et al., 1999; Pincus and Morley, 2001; Turk, 2002).

Many studies in the field of chronic pain have supported the idea that people who are fearful about their pain relative to non-fearful patients, report higher levels of pain severity (Zvolensky et al., 2001; Vlaeyen and Linton, 2000). Such cognitions are believed to be responsible in the maintenance of illness through misinterpretation of physical sensations as catastrophic and threatening (Hadjistavropoulos et al., 2000; Hadjistavropoulos et al. 2002). Researchers have applied mainly self-report questionnaires to study these cognitions (Pincus & Morley, 2001). As noted earlier, a problem with these methods is that they are subject to strategic cognitive processing rather than automatic cognitive processing (Pincus and morley, 2001; Mogg et al. 2000; Labus, Keefe, and Jensen, 2003). To date, only seven published studies have addressed pain-related attentional biases in chronic pain patient population. The primary hypothesis of all
the studies is that chronic pain patients show biased attention towards pain-related stimuli in comparison with non-related or neutral stimuli, and non-pain patients.

3.2.1 Stroop Task

The most frequently used technique for demonstrating attentional biases has been the emotional Stroop task (Stroop, 1935) (Williams et al., 1997). This task requires the subject to name the colours of words while ignoring their meanings (Wells and Mathews, 1996). The interference of meaning in colour naming is called Stroop task interference and was originally argued to be an index of attentional bias. Many investigators have applied this paradigm to a range of psychological disorders and confirmed strong and consistent attentional biases towards salient stimuli (e.g. GAD, PTSD, and Panic)(see Williams et al., 1997; Martin, Williams, and Clark, 1991 for a review).

In the first study to apply the Stroop task to chronic pain patients, Pearce and Morley (1989) hypothesised that chronic pain patients would show greater interference on pain related words. Four different Stroop tasks were administrated: colour naming, general negative emotional words, sensory pain words, and affective pain words. In general, the results revealed that relative to healthy subjects, chronic pain patients had significantly delayed colour-naming latencies for both sensory and affective cues of pain but not for negative emotional words. This pattern of results suggests that chronic pain patients have a priority to process sensory and affective components of pain but not negative emotional ones. One problem with this study was their use of non-computerised presentation and time recording, which casts doubt on the reliability of the reaction times reported.
Pincus, Fraser, and Pearce (1998) attempted to replicate Pearce and Morley’s (1989) work. Twenty chronic pain patients (12 female) from a pain clinic, and 20 age-matched volunteers (12 female) were recruited. Scores of depression and anxiety (HADS), pain intensity at the present time and at its worst on that week, and pain duration were recorded. Three categories of emotional words including positive, sensory, and affective words were presented. Results failed to support Pearce and Morley (1989) findings. Pincus et al. (1998) did not find any evidence that chronic pain patients differed from controls in their processing of sensory or affective words on the Stroop task. Controlling for the confounding effects of depression and anxiety did not change the pattern of results. The results of a recall task on completion of Stroop test revealed that pain patients recalled significantly more sensory words than controls. No other significant effects were found between or within groups.

Pincus et al. (1998) conducted a second experimental experiment designed to overcome the problems of the earlier study. They introduced computerised administration and controlled for the effects of anxiety and depression. Seventeen chronic pain patients (12 female) and same number healthy controls (11 female), matched for age, were recruited. Pain (MPQ and PPI), anxiety (STAI), and depression (BDI) were assessed. Eight categories of words were applied: classic Stroop, colour naming, sensory pain words, affective words, positive words, physical threat words, household words, social threat words. According to the results, there was no significant interaction between group and word condition. However, the difference between reaction times to affective words and the household category words in pain patients approached to significant level, but not in controls. However, when depression was controlled for, this difference no longer approached significance. Significant correlations were found between trait
anxiety and sensory pain words, and between depression and affective pain words, which led the authors to conclude that response latencies to pain-related stimuli are related to anxiety and depression rather than the experience of chronic pain.

Overall, Pincus et al.’s study (1998) failed to support a Stroop effect in chronic pain patients. However, the authors suggested that information biases might be more pronounced at early stages of pain when pain is more associated with anxiety. They claimed that over time, adaptation to pain may decrease sensitivity to pain cues (sources of threat or danger). However, Turk (2002) has suggested that some chronic pain patients despite their duration of pain, behave as if they are suffering from acute pain. These patients are vigilant and focus on their bodily sensations searching for more information indicating further harm. This argument suggests that future research needs to control for duration of pain and investigate whether people with acute pain differ from those who have been in long term pain.

Crombez, Hermans, and Adriaensen (2000) suggested that Stroop effects might be sensitive to the words, which are being chosen as stimuli. They argued that to result in an attentional bias, the stimuli must be representative of the core concerns of the pain patient. Crombez et al. (2000) argued that this aspect had been neglected in previous research. Since pain patients vary in symptoms, the sensory quality for different chronic pain problems is likely to be different. Crombez et al. (2000) investigated this question while taking care that the sensory and affective pain words were relevant for the pain population that they assessed. They selected a homogeneous group of chronic pain patients (only low back pain), and the most frequent words used by an independent group of LBP patients based on MPQ were selected and applied as target words. A set of words referring to back pain pathology (e.g. hernia and lumbago) was selected. Familiarity of the
patients with the experimental words, their degree of pain-relatedness, and pleasant or unpleasantness utilising a Likert type rating scale were measured. Based on fear of (re)injury model (Vlaeyen et al., 1995a), it was predicted that patients who are sensitive to their bodily sensations would have attentional bias to these words. Twenty-five chronic LBP patients after rating their current pain intensity (VAS and NRS) completed the modified Stroop task and then were requested to fill a battery of questionnaires.

The Stroop task consisted of five word categories: sensory pain words, affective pain words, specific back disorder words, other pain disorder word, and general negative valence words. Words were presented by computer and reaction times were recorded. Inspection of familiarity of patients with words revealed that sensory, affective, and back disorder words were significantly more related to pain and were reported as more unpleasant than the other word groups.

Crombez et al.’s (2000) results revealed no significant difference in reaction time between experimental word groups. However a pair-wise comparison indicated that patients were significantly slower in naming the colour of the sensory words in comparison with their matched control words. A multiple regression analysis revealed that only pain intensity was a significant predictor of colour naming for sensory pain words. This study indicated that reaction to sensory words was slower than reaction to matched words, but not differ from other experimental categories. This failure to find a robust effect of cognitive bias in the study led the researchers to argue that selected words (especially affective words) are not representative of core of concerns among chronic low back patients. This study found evidence of attentional biases to some categories of pain words (i.e. sensory) but not to others (i.e. affective or back injury words). This supports the
claim that word characteristics are important determinants of the responses obtained.

In a more recent article, Snider, Asmundson, and Wiese (2000) argued that, in addition to other limitations mentioned before, previous studies either with Stroop or Dot probe, have assessed cognitive biases at strategic (conscious) level, and have not addressed automatic information processing in chronic pain patients. Differentiation between automatic and strategic information processing is suggested to have etiological value in the development of pain as it has been shown in anxiety literature (e.g. McNally, 1995). Beck and Clark (1997) defined automaticity as effortless, mostly outside of consciousness, difficult to control, and consuming minimal attention. In contrast, they defined strategic processing as effortful, conscious, slow, absorbing considerable attention, and involve semantic analysis and synthesis. Snider et al. (2000), based on Pearce and Morley’s (1989), study developed 4 sets of words (covering the categories of: sensory pain, affective pain, physical, social), and matched control words. Every word was presented in two conditions, masked and unmasked. In the unmasked condition the presented word remained on the screen until the participant named the word colour. In the masked condition, a word was presented for 14.3 milliseconds and it was then replaced by a mask, consisting of random letters and similar in length to the replaced word, that remained on the screen until the participant responded.

Thirty-three chronic pain patients and same number of healthy controls, matched for age and sex, participated in the study. At the time of testing, the chronic pain patients described their pain as ranging from discomforting to distressing, based on the MPQ, and rated their pain severity from moderate to severe. On completion of the Stroop task self-report questionnaires, including BDI, BAI, ASI, STAI, and Pain Anxiety Symptoms Scales (PASS), were completed by all subjects.
Primary analyses failed to show any between group significant differences. However, further investigation of the results, controlling for depression scores (as a covariate), revealed a significant main effect for the masking condition. Further analyses showed that response times to sensory and affective words were significantly longer than those for neutral words in the pain group compared to controls, but only in the unmasked condition. These findings suggest that there is a difference between chronic pain patients and controls in cognitive responses to both sensory and affective pain-related visual stimuli. To determine the source of the observed effects, regression analyses revealed that only the cognitive anxiety subscale of PASS and ASI were significant predictors in the model. Contrary to expectations, lower levels of anxiety sensitivity were found to be related to longer colour-naming latencies to affective pain words. Snider et al. (2000) attributed this finding to levels of depression, which varied substantially within the patient group, and partly to individual differences over and above levels of depression. None of the self-reported indices were predictive of longer colour-naming latencies for sensory words.

Beck, Freeman, Shipherd, Hamblen, and Lackner (2001) studied the generality and specificity of attentional biases. Three samples of comorbid PTSD/chronic pain patients (n=28), chronic pain patients without symptoms of PTSD (n=26), and patients with motor vehicle accident-related injuries who reported neither PTSD nor pain (n=21) were compared on response latencies to four groups of words. Word types were accident-related, pain-related, positive, and neutral. All words were presented by computer. Groups were significantly different in their reaction times to the experimental words. The comorbid PTSD/chronic pain group showed attentional bias for both accident and pain-related words relative to positive and neutral ones. Chronic pain patients, however, only showed selective
attention to pain-related words, and both groups were significantly slower, relative to no pain/no PTSD group, across all word types. Controlling for mood, age, and duration of suffering from pain or PTSD, did not change the pattern of the results. The interesting point revealed by the study, is the specificity in response bias. That is, patients demonstrated biases to those words that where relevant to their condition (i.e. pain or threat). The findings of this study suggest that to capture attentional biases in chronic pain patients it might be necessary to apply more specific pain-related words.

While the study of Beck et al. (2001) seems promising, two very recent studies have questioned the validity of the Stroop task as an index of attentional bias. Roelofs, Peters, and Vlaeyen (2003) divided a sample of 36 chronic low back pain patients into low and high fear of pain groups (18 patients in each), and compared their response latencies as measured by the Stroop test to a matched control group, but no between group differences were found. These results are consistent with those of Moss-Morris and Pertie (2003) who used the Stroop in patients with Chronic Fatigue Syndrome (CFS). In support of the study’s hypothesis, it was found that that CFS patients had more somatic biased interpretation of ambiguous word cues relative to healthy control group. However, the results of the Stroop test were not consistent with the interpretative results collected through questionnaires. No between group interactions with word type were found, suggesting that none of the word types (somatic or depressed) were superior in interfering with response latencies. However, it was found that regardless of word type, CFS patients were slower on all somatic, somatic control words, depressed, and depressed control words, in comparison to the control group. The results of the study, however, are limited by applying a manual presentation of words on cards and manual timing. This general slowness in cognitive processing has been
reported by others (e.g. Duckworth et al., 1997). Duckworth, Iezzi, Adams, and Hale (1997) used a computerized version of the modified Stroop task to assess selective attention in two groups of patients including 9 chronic pain patients with numerous somatic symptoms, 10 chronic pain patients with few somatic symptoms, and a group of 10 healthy volunteers. Target words were divided into 3 groups including pain-related, depression-related, and neutral words.

Results indicated that relative to patients with few somatic symptoms and healthy volunteers, patients with numerous somatic symptoms exhibited delayed colour-naming response latencies for all word types tested, but did not show a selective bias for pain words. Duckworth et al. (1997) suggested that the observed pattern of results was likely the product of impaired stimulus filtering. This latter explanation would mean that chronic pain patients, at least those high on somatization, have a general attentional problem and are not able to divide their attention efficiently between relevant and irrelevant cues. However, the conclusions of this study are limited by low statistical power and reliance on questionable measures of somatizing. Nevertheless, support for such a general slowing Stroop response has been provided by Grisart and Plaghki (1999), who reported chronic pain patient are slow in cognitive processing regardless of the type of presented stimuli.

In addition to published research, the review by Pincus and Morley (2001) included unpublished but examined studies. They reported an unpublished Doctorate dissertation (Boissevan, 1994), which found that patients with pain show slower colour-naming on the Stroop task. Boissevan (1994) used the Stroop task in 20 chronic pain patients and 20 healthy controls to test the influence of pain and depression on information processing. Boissevan (1994) found that patients were slower on colour naming of sensory pain-related words but not on
pain-affect words. These results are partially consistent with findings by Pearce and Morley (1989), and consistent with Crombez et al. (2000) that chronic pain patients may respond to sensory pain-related stimuli slower than other word types.

In summary, the literature on selective attentional bias in chronic pain patients reveals mixed results. There is some evidence of biases for the sensory pain words (e.g. stabbing, burning, cramping) (e.g. Snider et al., 2000). However, other studies failed to confirm these (Pincus et al., 1998; Crombez et al., 2000, Roelofs, 2003). While there are methodological differences between studies that may account for the different findings, it is clear that if a Stroop effect exists, it is less robust than that observed in the anxiety disorders literature (Pincus and Morley, 2001). Roelofs et al. (2003) concluded that the Stroop test might not be an appropriate measure of attentional biases in chronic pain patient samples.

MacLeod and Mathews (1991) has criticised the Stroop task paradigm, arguing that the Stroop does not provide an unambiguous measure of attentional functioning. The observed Stroop effects may stem from other cognitive processes such as competition, either in responding to or after attending to other stimuli. Task interferences might be a result of semantic activation of the word content, diverting attention from detecting the ink colour (competition at the input stage) to the meaning of the word, or it may occur in those processes involved in selecting and generating the response. MacLeod and Rutherford (1992) argued that although the Stroop task has been traditionally viewed as the prototype of automatic processing, its adequacy as a measure of automaticity is under question because stimulus presentation times of words also permit strategic processing to occur. Similarly, Asmundson et al. (1997) have also suggested that delayed colour naming may result from rumination over word meaning and/or the effort to produce appropriate verbal responses. Dalgliesh et al. (2001) also argued that
interference on the Stroop task might be a response bias measure rather than an attentional bias (see also Fox, 1993). Thus, the design of the task does not rule out that the response biases are responsible for the observed latency in colour-naming. We cannot necessarily attribute task interference to an attentional bias, hence, even if consistent Stroop effects were observed, it would still be unclear whether reflected attentional biases opposed to response biases.

<table>
<thead>
<tr>
<th>Study</th>
<th>Stroop Type</th>
<th>participants</th>
<th>N</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearce &amp; Morley (1989)</td>
<td>card</td>
<td>Chronic pain patients Healthy controls</td>
<td>16</td>
<td>Sensory&amp; affective &gt; negative, &amp; neutral</td>
</tr>
<tr>
<td>Pincus et al. (1998)</td>
<td>card</td>
<td>Chronic pain patients Healthy controls</td>
<td>20</td>
<td>Positive= sensory= affective= neutral</td>
</tr>
<tr>
<td>Pincus et al. (1998)</td>
<td>computer</td>
<td>Chronic pain patients Healthy controls</td>
<td>17</td>
<td>Sensory= affective= positive= psychological threat= Social threat= neutral</td>
</tr>
<tr>
<td>Snider et al. (2000)</td>
<td>computer</td>
<td>Chronic pain patients Healthy controls</td>
<td>33</td>
<td>Sensory&amp; affective&gt; neutral</td>
</tr>
<tr>
<td>Crombez et al. (2000)</td>
<td>computer</td>
<td>Chronic pain patients</td>
<td>25</td>
<td>Sensory&gt;neutral, but =affective &amp; Negative</td>
</tr>
<tr>
<td>Study</td>
<td>Method</td>
<td>Condition 1</td>
<td>N</td>
<td>Condition 2</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>--------</td>
<td>----------------------------------</td>
<td>-------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>Duckworth et al. (1997)</td>
<td>computer</td>
<td>Chronic pain patients</td>
<td>19</td>
<td>Healthy controls</td>
</tr>
<tr>
<td>Beck et al. (2001)</td>
<td>computer</td>
<td>PTSD/PAIN patients</td>
<td>10</td>
<td>Non PTSD/PAIN patients</td>
</tr>
<tr>
<td>Moss-Morris et al. (2003)</td>
<td>card</td>
<td>Chronic fatigue patients</td>
<td>25</td>
<td>Healthy controls</td>
</tr>
<tr>
<td>Roelofs et al. (2003)</td>
<td>computer</td>
<td>Low back pain patients</td>
<td>36</td>
<td>Healthy controls</td>
</tr>
</tbody>
</table>

Table 3.1 summarises the studies used Stroop task in chronic pain population

Criticisms logged against the Stroop task as a measure of selective attention could be summarised as follows:

- The Stroop task does not provide an unambiguous measure of attentional functioning.

- In the case of threat-related biases, the source of interference remains unknown.
Since the participant is requested to ignore some information (words) in favour of others (colours), the responses may be just the result of trying to give the correct answer rather than an attentional bias.

3.2.2 Dot Probe Paradigm

To overcome the above mentioned shortcomings of the Stroop task, MacLeod, Mathews, and Tata (1986) developed the dot probe paradigm. In dot probe task, two words are presented briefly on the computer screen, one above the other. One of the words usually has emotional valance while the other one is neutral. As words disappear, a dot appears on the same special place of one of the words randomly. The subject is requested to press a key as fast as possible when see the dot on the screen. According to MacLeod et al. (1986), people who are preoccupied cognitively by emotional words will detect the dot faster than those who show no preferential attention to none of them. Significant differences between response times to dots at the same special place of emotional words and neutral ones, is taken as an index of attentional bias.

The strength of the dot probe task is that a neutral response is given to a neutral stimulus. That is, the participant is requested to press a key in response to a dot that does not have any meaningful valence (MacLeod et al., 1986). This paradigm has been endorsed by many researchers as the most appropriate technique to assess attentional biases (Williams, Watts, MacLeod, and Mathews, 1997).

Several modified versions of the dot probe paradigm have been developed and applied to different disorders, such as General Anxiety Disorder (Bradley et al., 1999), and panic disorder (Asmundson et al., 1994). Patients with these disorders have been found to selectively attend to threat stimuli relevant to their disorder. However, the dot probe has rarely been applied to chronic pain population.
Asmundson, Kuperos, and Norton (1997) conducted the only reported study utilising dot probe task to address the question of selective attention in chronic pain patients. In that study nineteen patients with chronic musculoskeletal pain and 22 healthy control participants were assessed using the dot probe test. Thirty-two words, 16 related to pain and 16 related to injury, were matched with neutral words within the dot probe paradigm.

Asmundson et al. (1997) did not found any significant interactions between words, probe position and/or target position. Depression was found to be unrelated to attentional bias. Overall, the results of this study failed to demonstrate evidence for an attentional bias in patients with chronic pain. However, post-hoc analyses revealed that chronic pain patients who were low on anxiety sensitivity shifted their attention away from pain-related cues compared to those with high anxiety sensitivity. This suggests that those low in anxiety sensitivity might learn to ignore pain stimuli, such that people high in anxiety sensitivity show a relative bias towards pain words rather than a general bias.

Replication of Asmundson et al. (1997) study when other theoretically related concepts, such as pain severity, catastrophising, self-efficacy, and fear of pain are measured, would allow the relative contribution of different factors to be assessed. Importantly, conclusions regarding these results reported by Asmundson et al. (1997) are limited by their small sample size which decreases the statistical power of the studies. Since chronic pain patients are not a homogenous group, a larger sample would seems to be needed for investigating attentional biases in chronic pain population.

Taken together, the results of the literature suggest that there may be selective attentional biases in at least some patients with chronic pain conditions. Results
seem to vary between studies and may reflect differences in words, paradigms, or sample characteristics. In general, the literature has been limited by the size of sample that have limited power and this may contribute to the mixed results. The Asmundson et al.’s (1997) study remains the only study to divide their patients into different levels of anxiety sensitivity, theoretically a predispositional factor. However, this manipulation was conducted as post-hoc analysis and the findings are therefore to be seen as exploratory only, and require confirmation through an appropriately designed study.

The inconsistency of results in research on cognitive biases among chronic pain patients in comparison to research on anxiety disorders might be the result of chronic pain is a more complex and heterogeneous disorder (e.g. Turk, 1996). The multidimensional nature of chronic pain may make it different from more discrete psychological conditions as phobias or panic disorder. As such, hypervigilance in chronic pain patients might be mediated or moderated by other psychological factors frequently reported by research on this population including anxiety, anxiety sensitivity, fear of pain, catastrophising, self-efficacy, pain severity, pain intensity, depression, physical activity, duration of pain, or socio-economic factors. Many of these factors have been suggested as important variables in the transition from acute pain to chronic pain and/or in the maintenance of pain in a number of empirical studies by contemporary cognitive-behavioural theories (Vlaeyen et al., 1995b, Vlaeyen and Linton, 2000; Asmundson et al., 1999; Turk, 2002). As Asmundson et al. (1997) showed, chronic pain patients with low anxiety sensitivity (i.e. fear of pain) may take their attention away from pain stimuli or may not particularly attend to pain stimuli. In contrast, studies using different paradigms have found that people with higher levels of health anxiety tend to focus their attention on bodily sensations, possibly to detect information
related to their pain (Turk, 2002; Hadjistavropoulos, Hadjistavropoulos, and Quine, 2000). Further evidence for the importance of anxiety-related factors in attention towards pain-related stimuli has been found in healthy people.

Using a modified dot probe task, Keogh et al. (2001a) assessed the reaction time of seventy-four healthy first year university students to words related to pain, social threat, and positive. On completion of dot-probe task, participants completed a battery of questionnaires. Keogh et al. (2001a) reported that for pain-related material, a significant difference in response time was found between the low and high pain fearful groups, and between the medium and high pain fearful groups. Specifically, those in the high pain fearful group exhibited a selective attentional bias towards pain-related material compared to those with a low or medium fear of pain. Surprisingly, no difference was found on ASI scores between three groups and anxiety sensitivity failed to be consistently related to dot-probe responses. The other interesting findings were the significant negative correlations between both stress and anxiety subscales of DASS with the attention bias towards pain-related material. This finding suggests that general mood state and specific fears may be related to different types of bias. It is possible that specific measures are needed to detect specific pain-related biases, which may explain inconsistencies in previous research. In addition, these results indicate that attentional processing of pain-related material is not just a product of chronic pain states, but may also be observed in a healthy group who are not currently in pain.
The results of the study confirmed that people who are more fearful of pain attended selectively to pain-related stimuli. In addition, since these results were found in a healthy group, it suggests that selective attention to pain may not be simply a consequence of pain. Recent psychological theories of chronic pain suggest that anxiety sensitivity or fear of pain may predispose healthy people to show chronic pain if they become injured (Asmundson et al., 1999; Vlaeyen and Linton, 2000; Turk, 2002). However, further research is needed to confirm that the hypothesis that those individuals who selectively attend to pain are more likely to develop chronic pain conditions in the presence of injury. In general, the Keogh et al.’s (2001a) study confirmed that fear of pain mediates the selective attention to pain-related cues, at least in healthy controls. More recently, Keogh, Thompson, and Hannent (2003) reported that healthy university students with low levels of fear of pain showed attention away from pain-related stimuli at unmasked (conscious) condition but not at masked (unconscious) condition; whereas those high in fear of pain did not show such effect in none of the conditions. Keogh et al. (2003) concluded that high fearful people might lack the ability to consciously orient their attention away from pain, and as a result, are vulnerable to develop chronic pain. The results are generally consistent with those of Asmundson and Taylor (1996) and Asmundson et al. (1997), but suggest that the specific fear of pain rather than anxiety sensitivity may be the relevant factor. Further investigation is needed that would combine the designs of Asmundson et al.’s (1997) study and that of Keogh et al.’s (2001a) study, including a large sample of chronic pain patients and a matched healthy control group.

3.3 Summary of limitations of the research to date

Methodological shortcomings such as non-computerised or semi-computerised assessment of attention (e.g. Pearce and Morley, 1989; Pincus et al, 1998,
Experiment 1); small sample sizes, with 36 subjects as the largest sample (e.g. Roelofs et al., 2003); lack of healthy controls (e.g. Crombez et al., 2000); and using all patients with pain (e.g. rheumatoid arthritis, chronic fatigue syndrome) as the same limits the findings of the reviewed studies. Failure to include other psychological variables, which have been found important in accounting for maintenance of pain and might play a role in attention to pain (e.g., catastrophising, pain severity, pain intensity, self-efficacy), is another limitation of the literature in general.

In addition, Pincus and Morley (2001) have pointed to variability in number of colours and presentation (cards versus computer) in the Stroop test, poor matching of selected words, and the validity of words in activating the attention of chronic pain patients. Nevertheless, the same sample sizes and procedures have differentiated people with anxiety problems from healthy people in terms of the selective attention to emotional stimuli. Hence, it seems that there is a difference between chronic pain patients and people with anxiety disorders in their propensity to selectively attend to relevant threatening stimuli.

It is possible that the role of attentional bias in chronic pain is different from the role of selective attention in the anxiety literature. Support for this comes from Keogh et al.’s (2001a) study with healthy subjects which showed a robust effect which none of the studies with chronic pain patients showed. Chronic pain populations are heterogenous and chronic pain patients vary substantially across psychological variables. While it is possible to classify people with anxiety disorders based on a fundamental psychological criterion, there is not a single but shared factor across all chronic pain patients, reflecting the complexity of chronic pain. The existence of a physical damage and its functional consequences might
be another crucial difference between chronic pain and anxiety problems (Eccleston and Crombez, 1999).

Over all, high variability in duration of pain in patients who participated in previous research, is a factor that has not received any attention in terms of its relationship with the selective attention. Geisser, Haig, and Theisen (2000) reported that longer duration of pain was associated with better performance on a special lifting index. They argued that it might be a result of adjustment. If patients with shorter duration of pain are more sensitive to pain, then attentional biases may be shown by these patients rather than those who have been in pain for a long time. These issues are important gaps in present literature, which have not yet been addressed.
4 Chapter Four

Selective attention to pain-related information in chronic musculoskeletal pain patients

This chapter has been published as follows:

Mohsen Dehghani, Louise Sharpe, Michael K. Nicholas (2003). Selective attention to pain-related information in chronic musculoskeletal pain patients; Pain, 105 (Issue 1-2): 37-46

4.1 Introduction

Theories of chronic pain highlight the role of fear of pain/(re)injury in the development and maintenance of chronic pain (e.g. Lethem et al., 1983; Vlaeyen et al., 1995a, 1999; Asmundson et al., 1999). Accordingly, people who are fearful, worry about the consequences of pain and respond with increased hypervigilance to pain. Hypervigilance promotes avoidance, leading to physical deconditioning and disability (Vlaeyen et al., 1999).

Contemporary theories draw on theories of anxiety disorders (Sharp, 2001). In anxiety disorders, fear leads to selective processing of fear-related information, which leads to avoidance and increased disablement (Eysenck, 1992). Researchers have developed experimental paradigms to examine selective attention. There is strong support for anxiety being associated with selective attention towards anxiety-related stimuli (Mogg and Bradley, 1998). The effect is robust, found in
clinical and non-clinical samples and using different methodologies (e.g. Williams et al., 1997; Matthews & Wells, 2000; Dalgleish et al., 2001; Heinrich & Hofman, 2000).

These paradigms have been applied to chronic pain. There are nine published articles on selective attention in chronic pain and eight have used the modified Stroop paradigm. The results of these studies have been inconsistent (Pincus & Morley, 2001, 2002). Only three found that chronic pain patients selectively attended to pain-related stimuli (Pearce & Morley, 1989; Snider et al., 2000, Beck et al., 2001). In the most recent study, Beck et al. (2001) found that PTSD/pain patients showed a significant difference in responding to both pain and accident-related words relative to positive and neutral ones, whereas pain patients/no PTSD were slow only in responding to pain-related words but not other words. Although Beck et al. (2001) found these biases in chronic pain patients with and without PTSD, other studies found a bias only in sub-groups of pain patients, such as depressed (e.g. Pincus, Fraser & Pearce, 1998) or anxious patients (Crombez, Hermans & Andriansen, 2000, Snider et al., 2000).

The modified Stroop task has been criticised for being ambiguous and open to response-bias artefact (MacLeod et al., 1986). To overcome this problem, MacLeod et al. (1986) developed the dot-probe task. In this task, two words (experimental and control) are presented, one above the other. Their presentation is followed with a probe and the participant responds with a key press. If attention is drawn to the experimental stimuli, reaction times are faster when the probe appears in the location of the experimental word. Only one published study has used the dot-probe task in chronic pain patients. Asmundson and colleagues
(1997) failed to find selective attention amongst chronic pain patients, however, found that individuals low in anxiety sensitivity demonstrated a bias away from pain stimuli.

These mixed findings have led some authors to question the role of attentional biases in chronic pain, but methodological limitations warrant caution in accepting this conclusion. Pincus and Morley (2001) noted that only one study considered the influence of specificity of the stimuli on response and found the type of pain word influences bias (Crombez et al., 2000). Indeed, there is evidence of differences between responding to sensory and affective pain words (Crombez et al., 2000; Snider et al., 2000). Studies examining only pain and control words may have missed an effect present for a subset of stimuli.

Studies of selective bias have also relied on small samples (range: 16-40). These sample sizes are sufficient to detect large effect sizes, but smaller effect sizes would be missed, producing a Type II error. The median effect size for available studies is 0.23 (Pincus and Morley, 2001), which would require a sample of 64 to find this effect (Cohen, 1992).

Another problematic issue is the relationship between emotional state and selective attention. Keogh et al. (2001a) found that healthy subjects high in fear of pain demonstrated a selective bias towards pain words, whereas, those low in fear of pain attended away from pain stimuli. Perhaps only those chronic pain patients high in fear of pain may demonstrate the predicted bias. Recently, Keogh and Cochrane (2002), using a cold pressor task, found that healthy participants with high anxiety sensitivity showed greater interpretive biases towards negative pain information. However, these biases were found through self-report and were not observed in the dot probe task also administered.
The aim of the present study was to investigate selective attention in a large sample of patients, sufficient to detect a moderate effect size with an alpha level of 0.05 and 95% power. Pain stimuli were grouped into four categories (sensory, affective, threat and disability) to assess differences between types of pain words. All participants completed questionnaires assessing variables that may influence attentional responses.

We hypothesized that chronic pain patients would demonstrate faster responses to the probe when presented in the same location as the pain-related word. Further, we predicted that individuals high in fear of pain would demonstrate more attentional bias than those with moderate fear of pain, who would in turn demonstrate greater attentional bias than those low in fear of pain.

4.2 Method

4.2.1 Design

A 3 (fear of pain) x 4 (word type) x 2 (probe location) x 2 (target location) mixed model design was employed. The between-group factor was fear of pain (high, medium, low) and within-group factors were word type (affective, threat, sensory, disability), probe location (upper, lower) and target location (upper, lower). The dependent variable was response latency.

4.2.2 Participants

The study was conducted at a Pain Management Centre in Sydney, Australia. Inclusion criteria were being over 18 years, having constant pain for at least 3 months (IASP, 1986) and English literacy. Exclusion criteria were not being able
to use both hands, head injury, serious mental illness or current alcohol or drug abuse. Between January and September 2002, 231 consecutive patients referred to the pain clinic were invited to take part. Of those approached, 204 patients were eligible. One hundred and seventy-six∗ patients agreed to participate (87%). The study was approved by The University of Sydney and Hospital Ethics Committees.

The mean age of the sample was 44.5 (SD: 13.4; range: 18-80) and the average pain duration was 74 months (SD: 97.15). Sixty-one percent (n=103) were married. English was the native language of 93.5% of the sample. Only 40 participants were educated to University level (23%), with 50 completing high school (28%), and the remainder having less than ten years of education (49%). The distribution of pain problem by pain site was as follows (Merskey & Bogduk, 1994). Sixty-six participants had chronic low back pain (37.5%), 26 reported upper limb pain (15%), 24 had lower limb pain (n=24,14%) and 6% had cervical pain (n=11). Fifty four percent were unemployed, 18.5% worked full-time, 9.5% part-time, 11.9% retired, 4.25% casual, and 1.8% were studying. Seventy-five patients (43%) were receiving workers’ compensation. Eight patients were excluded due to incomplete questionnaires (n=6), brain injury (n=1), and technical problems (N=1). Data related to 168 patients was available for the analyses.

4.2.3 Measurements

Participants completed a battery of questionnaires.

4.2.3.1 Fear of Pain Questionnaire – III (FPQ-III) (McNeil and Rainwater, 1998)
FPQ-III consists of 30 items assessing fear of pain fear of pain. FPQ-II has good reliability and validity in pain management settings (Turk and Melzack, 2001).

∗ These 166 patients are from the original large sample of 207 reported in chapter two.
Participants were divided into three groups based on the FPQ-III scores: Low fear of pain scored in the lowest quartile (<52, N=42), people in the upper quartile (>80, N=42) were considered high fear of pain, and the remainder (N=84) medium fear of pain. An average score of 79 has been reported for normal samples (McNeil and Rainwater, 1998) and it is noteworthy that our high fear of pain group have scores just above the population average (i.e. > 80). Although we used the same procedure as Keogh et al. (2001a) to classify subjects into three fear-of-pain groups, our three groups had lower fear of pain scores than the university students in that study. We investigated using Keogh et al.’s (2001a) cut-off points, but only 11 of the current sample (6.5%) would have been classified as high in fear of pain according to those criteria. Such a small sub-sample would have precluded appropriate statistical analyses and hence we reverted to the quartile split procedure adopted by Keogh et al. (2001a) as the basis of the fear of pain groups.

4.2.3.2 Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992)
The ASI is a 16-item questionnaire measuring sensitivity to anxiety-related sensations. High levels of internal consistency and good test-retest reliability have been reported (Peterson and Reiss, 1992, Asmundson, 1999).

4.2.3.3 Depression, Anxiety, and Stress Scale (DASS) (Lovibond & Lovibond, 1995)
The DASS is a 42-item measure of depression, anxiety, and stress. The DASS does not rely predominantly on somatic items, and is less likely to be inflated in a chronic pain setting. The reliability and validity of DASS are well established. Lovibond & Lovibond (1995) have reported the Cronbach’s alpha for each subscale: anxiety = 0.84, depression =0.91, and stress = 0.90.
4.2.3.4 Tampa Scale of Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990)
TSK consists of 17 items rated on a 4-point Likert-type scale. TSK is a measure of fear of movement and has been shown to have a good validity and reliability (Vlaeyen et al., 1995a).

4.2.3.5 Pain severity
The pain severity subscale of West Haven-Yale Multidimensional Pain Inventory was included to assess severity of pain (Kerns, Turk, & Rudy, 1985). This subscale has 3 items on a seven point scale.

4.2.3.6 Roland and Morris Disability Questionnaire (RDQ) (Roland & Morris, 1983)
RDQ is a 24-item checklist to determine the degree to which a person is limited by pain in completing daily activities. The RDQ has good psychometric properties (Roland and Fairbank, 2000). In this study, we modified the wording from “my back pain” to “my pain”.

4.2.3.7 The Pain Responses Self Statements (PRSS) (Flor et al, 1993).
PRSS, an 18-item questionnaire, was included to assess catastrophic pain related cognitions. Psychometric properties of PRSS have been established (Flor et al., 1993).

4.2.3.8 Pain self-efficacy Questionnaire (PSEQ) (Nicholas, 1989)
PSEQ was administered as an index of patients’ self-efficacy. The PSEQ contains 10 items rated on a 7-point rating scale, with higher scores indicating increased self-efficacy. The reliability and validity of the PSEQ are good (Asghari & Nicholas, 2001).
4.2.4 Dot probe task

A modified dot-probe program was developed specifically for this research using Visual Basic Programming. All the stimuli were presented on the monitor of an IBM 600X LAPTOP, Intel Inside, Pentium III. A fixation point “.” appeared on the centre of the monitor for 500 ms and was then replaced by a pair of words, one above and the other below the fixation point. The words remained for 500ms and were then replaced with the fixation point. The fixation point was subsequently replaced with the letter “p” or “q”, which appeared randomly in the same location as one of the words. Subjects were requested to press “p” or “q” as quickly as possible when either appeared. These cues disappeared when the subject pressed the key or after 1500ms. Each pair of words was presented four times and each word/target combination occurred in random order. The time between the appearance of “p” or “q” and the participant’s key press was recorded as the dependent variable. A one-minute break was given to participants after the presentation of 40 words, giving three breaks in total. Five pairs of practice words were presented at the beginning of the task and two more practice words were presented at the start of each set.

Four sets of stimuli related to different aspects of pain including affective, sensory, disability, and threat words were selected from words on the McGill Pain Questionnaire (Melzack, 1987) and those used in previous research. Ten words were allocated to each category (table 1) and matched for length and frequency with a neutral word (Kucera and Francis, 1967). Forty pairs of target/neutral words comprised the final set of stimuli (Table 4.1).

* We used “p” and “q” instead of “•” to enhance the face validity of the task, and increase attention to the task.
<table>
<thead>
<tr>
<th>Affective/Neutral</th>
<th>Disability/Neutral</th>
<th>Sensory/Neutral</th>
<th>Threat/Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vicious/lessons</td>
<td>vulnerable/afterwards</td>
<td>flickering/waterfalls</td>
<td>rushing/elephant</td>
</tr>
<tr>
<td>Annoying/chivalry</td>
<td>suffering/expensive</td>
<td>throbbling/sailboat</td>
<td>fearful/tribune</td>
</tr>
<tr>
<td>Miserable/undertake</td>
<td>disable/semester</td>
<td>shooting/drinking</td>
<td>rightful/flowering</td>
</tr>
<tr>
<td>Troublesome/restraining</td>
<td>paralysed/detectors</td>
<td>boring/swivel</td>
<td>terrifying/innovative</td>
</tr>
<tr>
<td>Unbearable/metabolite</td>
<td>unhealthy/timetable</td>
<td>drilling/whirling</td>
<td>killing/crystal</td>
</tr>
<tr>
<td>Cruel/drums</td>
<td>ill/joy</td>
<td>sharp/items</td>
<td>suffocating/advertisers</td>
</tr>
<tr>
<td>Tiring/cotton</td>
<td>sick/rear</td>
<td>burning/moment</td>
<td>scared/garage</td>
</tr>
<tr>
<td>Exhausting/blackberry</td>
<td>inactive/flooring</td>
<td>stiff/skirt</td>
<td>danger/waited</td>
</tr>
<tr>
<td>Punishing/advocates</td>
<td>chronic/grasped</td>
<td>tugging/refresh</td>
<td>harmful/airmail</td>
</tr>
<tr>
<td>Discouraging/subcommittee</td>
<td>injury/mirror</td>
<td>pinching/postmark</td>
<td>threat/golden</td>
</tr>
</tbody>
</table>

**Table 4.0.1** Word pairs used in the Dot-probe Task
4.2.5 Procedure

The dot probe task was administrated at the initial assessment for the Pain Clinic. Instructions were given verbally, and presented on the monitor, as follows: “This is an attention task. First, you will see a ‘.’ in the centre of the screen. Focus your gaze on the dot when it appears. After the dot, you will be shown a pair of words, one above the other. Read the words silently. The words will be followed by the letters ‘p’ or ‘q’. Press the ‘p’ key as fast as you can when you see ‘p’. Press the ‘q’ key as fast as you can when you see ‘q’. The computer will record your responses.”

4.2.6 Analyses

All analyses were conducted using SPSS 11.0. Preliminary analyses involved one-way ANOVAs with the between groups factor being FOP to determine which other variables differed between groups. Correlations between those variables that were different and indices of attention bias were conducted to determine which variables, if any, should be included as covariates.

Indices of attention bias were calculated for each word set using the following formula:

\[ \text{Index} = \frac{(\text{tulp} - \text{tupl}) + (\text{tlpu} - \text{tupu})}{2}; \]

\( t = \) target word, \( p = \) probe, \( u = \) upper location, and \( l = \) lower location.
For example, “tupl” for sensory/neutral word pairs indicates a trial where a target word appeared in the upper location, followed by a probe in the lower location. A positive score indicates selective attention towards the location of the target, and a negative score indicates avoidance of the stimulus (Keogh et al., 2001b).

A mixed model 3 (fear of pain groups) x 4 (word type) x 2 (word position) x 2 (probe position) MANOVA was conducted. In addition, a one-way repeated measures MANOVA was conducted for word type, using the indices of attention bias.

4.3 Results

4.3.1 Participant Characteristics

Consistent with other studies of chronic pain as reported in chapter 3, participants reported moderate levels of pain, disability, depressive and anxiety-related symptomatology and severe levels of stress (See Table 4.2 for means).
There were no significant differences between fear of pain groups on any of the DASS sub-scales, although anxiety approached significance \( F(2,162)=2.75, p<.06 \). The fear of pain groups were different on ASI scores \( F(2,167)=15.41, p<.0001 \). Patients with differing levels of fear of pain groups

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Low</th>
<th>Medium</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPQ</td>
<td>67.30(18.99)</td>
<td>42.50(7.03)</td>
<td>68.5 (7.50)</td>
<td>90.80(11.03)</td>
</tr>
<tr>
<td>ASI</td>
<td>8.63(11.87)</td>
<td>12.42(9.78)</td>
<td>18.22(10.03)</td>
<td>25.66(13.58)</td>
</tr>
<tr>
<td>TSK</td>
<td>39.14(9)</td>
<td>34.85(8.82)</td>
<td>40.27(8.17)</td>
<td>41.07(9.55)</td>
</tr>
<tr>
<td>DASS-A</td>
<td>8.82(7.89)</td>
<td>6.52(5.82)</td>
<td>9.09(7.92)</td>
<td>10.50(9.13)</td>
</tr>
<tr>
<td>DASS-D</td>
<td>14.96(12.39)</td>
<td>12.97(11.84)</td>
<td>14.32(11.92)</td>
<td>18.09(13.44)</td>
</tr>
<tr>
<td>DASS-S</td>
<td>7.27(11.22)</td>
<td>14.05(10.41)</td>
<td>18.23(10.88)</td>
<td>18.50(12.25)</td>
</tr>
<tr>
<td>Catastrophising</td>
<td>2.60(1.07)</td>
<td>2.07(1.06)</td>
<td>2.63(.99)</td>
<td>3.02(1.07)</td>
</tr>
<tr>
<td>PSEQ</td>
<td>26.23(13.71)</td>
<td>24.31(13.18)</td>
<td>26.79(13.52)</td>
<td>27.07(14.74)</td>
</tr>
<tr>
<td>MPI-sev.</td>
<td>4.07(1.13)</td>
<td>3.91(1.23)</td>
<td>4.06(1.14)</td>
<td>4.25(1.01)</td>
</tr>
<tr>
<td>DQ</td>
<td>11.51(5.58)</td>
<td>11.15(5.59)</td>
<td>11.45(5.62)</td>
<td>12 (5.58)</td>
</tr>
<tr>
<td>Age</td>
<td>44.56(13.40)</td>
<td>47 (13.61)</td>
<td>43.21(12.79)</td>
<td>44.83(13.40)</td>
</tr>
</tbody>
</table>

**Table 4.0.2 Characteristics of participants**

FPQ, Fear of Pain Questionnaire; ASI, Anxiety Sensitivity Index; TSK, Tampa Scale for Kinesiophobia; DASS anxiety, depression, and stress scales; PRSS, catastrophising; PSEQ, Pain Self-Efficacy Questionnaire; MPI, Multidimensional Pain Inventory-severity scale; DQ, Roland & Morris Disability Questionnaire.
also exhibited significant differences for fear of movement $[F(2,161)=6.57, p<.002]$ and catastrophizing $[F(2,155)=8.19, p<0.001]$. A series of independent t-tests were conducted to compare the responses of men and women. Males scored significantly higher for fear of movement/(re)injury scores ($t(160)=2.49, p<.01$), and lower on pain self-efficacy ($t(1,158)=-2.38, p<.01$). No gender differences were found on fear of pain scores, mood-related measures or indices of attentional bias.

Since the dot probe task involves language skills, the impact of education level was examined. There were no significant correlations between education level and attentional bias and hence, education was not considered in further analyses. Correlations were conducted between attentional bias indices and FPQ-III scores, ASI, catastrophising, DASS-anxiety, DASS-depression scales and fear of movement (See Table 4.1). No significant correlations between the anxiety-related constructs, depression and attentional bias were observed, but significant correlations emerged between the disability attentional bias index, TSK scores and catastrophising. A significant correlation was also found between the attentional bias index for threat words and TSK scores.

To ensure that these correlations did not affect the results, the analyses were conducted with catastrophizing and TSK scores as covariates. However, the results of the MANCOVA did not affect the pattern of the results. As such, only the results of the MANOVA are reported below.
Table 4.0.3 Inter-correlation between questionnaires

<table>
<thead>
<tr>
<th></th>
<th>FPQ</th>
<th>ASI</th>
<th>DASS-A</th>
<th>DASS-D</th>
<th>TKS</th>
<th>CATAS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASI</td>
<td>.45**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASS-A</td>
<td>.26**</td>
<td>.60**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASS-D</td>
<td>.18**</td>
<td>.49**</td>
<td>.57**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSK</td>
<td>.29**</td>
<td>.34**</td>
<td>.37**</td>
<td>.46**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CATAS.</td>
<td>.36**</td>
<td>.43**</td>
<td>.37**</td>
<td>.65**</td>
<td>.53**</td>
<td></td>
</tr>
<tr>
<td>Aff.indx</td>
<td>.05</td>
<td>.03</td>
<td>.01</td>
<td>.00</td>
<td>.03</td>
<td>.01</td>
</tr>
<tr>
<td>Dis.indx</td>
<td>-.15</td>
<td>-.05</td>
<td>-.06</td>
<td>-.12</td>
<td>-.18*</td>
<td>-.19*</td>
</tr>
<tr>
<td>Sen.indx</td>
<td>.07</td>
<td>-.08</td>
<td>-.03</td>
<td>-.05</td>
<td>.00</td>
<td>-.10</td>
</tr>
<tr>
<td>Thr.indx</td>
<td>.07</td>
<td>-.07</td>
<td>-.10</td>
<td>-.10</td>
<td>.18*</td>
<td>-.05</td>
</tr>
</tbody>
</table>

FPQ: Fear of pain total score; ASI: Anxiety Sensitivity Index; DASS-D Depression, DASS-A Anxiety, TSK Tampa Kinesiophobia, Catastrophising (PRSS), Affective, Disability, Sensory, and Threat Indexes. (2-Tailed) *p<0.05; **p<0.01

4.3.2 Selective attentional bias

Individual reaction times for each patient were collected and grouped by word type, word location and probe position. All responses less than 200 ms or above 1000 were removed as outliers (Keogh et al., 2001b). Inspection of responses revealed that false/no responses accounted for only 6% of responses and were removed and not further considered.

A mixed-group ANOVA was conducted with fear of pain (high vs. medium, vs. low) as the between-group factor and word type (threat vs. disability vs. sensory vs. affective), probe location (upper vs. lower), and target word location (upper vs. lower) as within-group factors. Correction for the multiple tests was carried
out. A main effect was found for probe location \([F(1,165)=7.62;p<0.006]\) and word type \([F(3,165)=3.15;p<0.02]\), but not for word position or fear of pain group (\(p > 0.30\)). These results suggested that participants responded more quickly to probes located in the upper location and more slowly to sensory/neutral word pairs than other pairs (See Table 4.4).

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Low</th>
<th>Medium</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. TUPU</td>
<td>565(87)</td>
<td>578(73)</td>
<td>559(91)</td>
<td>589(88)</td>
</tr>
<tr>
<td>A. TUPD</td>
<td>575(88)</td>
<td>567(79)</td>
<td>567(83)</td>
<td>597(101)</td>
</tr>
<tr>
<td>A. TDPU</td>
<td>579(86)</td>
<td>566(75)</td>
<td>573(82)</td>
<td>604(98)</td>
</tr>
<tr>
<td>A. TDPD</td>
<td>591(86)</td>
<td>578(73)</td>
<td>590(86)</td>
<td>608(96)</td>
</tr>
<tr>
<td>D. TUPU</td>
<td>573(84)</td>
<td>559(69)</td>
<td>566(82)</td>
<td>601(95)</td>
</tr>
<tr>
<td>D. TUPD</td>
<td>576(93)</td>
<td>569(79)</td>
<td>572(85)</td>
<td>590(119)</td>
</tr>
<tr>
<td>D. TDPU</td>
<td>571(77)</td>
<td>566(74)</td>
<td>565(71)</td>
<td>587(91)</td>
</tr>
<tr>
<td>D. TDPD</td>
<td>581(89)</td>
<td>567(77)</td>
<td>579(85)</td>
<td>599(105)</td>
</tr>
<tr>
<td>S. TUPU</td>
<td>568(83)</td>
<td>553(78)</td>
<td>566(76)</td>
<td>599(94)</td>
</tr>
<tr>
<td>S. TUPD</td>
<td>590(88)</td>
<td>580(71)</td>
<td>581(86)</td>
<td>615(103)</td>
</tr>
<tr>
<td>S. TDPU</td>
<td>582(82)</td>
<td>571(75)</td>
<td>574(86)</td>
<td>608(90)</td>
</tr>
<tr>
<td>S. TDPD</td>
<td>574(89)</td>
<td>562(85)</td>
<td>573(86)</td>
<td>587(98)</td>
</tr>
<tr>
<td>T. TUPU</td>
<td>579(84)</td>
<td>557(74)</td>
<td>576(83)</td>
<td>606(90)</td>
</tr>
<tr>
<td>T. TUPD</td>
<td>575(85)</td>
<td>566(82)</td>
<td>575(81)</td>
<td>587(96)</td>
</tr>
<tr>
<td>T. TDPU</td>
<td>565(91)</td>
<td>545(76)</td>
<td>560(88)</td>
<td>597(115)</td>
</tr>
<tr>
<td>T. TDPD</td>
<td>572(92)</td>
<td>563(78)</td>
<td>568(85)</td>
<td>588(115)</td>
</tr>
</tbody>
</table>


Table 4.0.4 Mean reaction times (ms) of each word group (affective, disability, sensory, and threat), word location (upper/lower), and probe location (upper/lower) for total sample and fear of pain groups.

There was a significant two-way interaction between word type and word position \([F(1,165)=9.12;p<0.005]\). Contrasts revealed that when the target word was presented in the upper half, participants responded more slowly for threat words.
whereas the reverse was true of disability \([F(1,165)=26.22;p<0.005]\) and affective words \([F(1,165)=3.82;p<0.05]\), but not sensory words \([F(1,165)=1.46;p=0.23]\). The two-way interaction between probe position and fear of pain group \((1,165)=3.50;p<0.03\) was also significant. Post-hoc comparisons showed that in trials where affective words appeared in the lower location and the probe in the upper location, the high fear of pain group were slower than the low fear of pain group \((p<0.04)\). For disability words, people high in fear of pain, reacted slower to probes in the same location as the target relative to the other fear of pain groups \((p<0.02)\). Reaction time to sensory words also revealed that people with high fear of pain were slower than those in low \((p<0.04)\) and medium fear of pain groups \((p<0.03)\). Similarly, for threat words, the high fear of pain group was significantly slower than low \((p<0.008)\) or medium fear of pain \((p<0.03)\) in reaction to probes following threat words. Figures 4.1-4.4 shows the mean reaction time for fear of pain groups across difference word groups. None of the other two-way interactions reached significance \((p>0.13)\).
Figure 4.2 Mean reaction times for disability words by fear of pain group

Figure 4.3 Mean Reaction times for affective words by fear of pain group
Of the three-way interaction effects, only the word-type by word position by probe position interaction reached significance [$F(6,165)=11.55; p<0.001$]. Contrasts revealed that participants responded more quickly to sensory words appearing in the upper location that were replaced by the probe in comparison to threat words [$F(1,165)=25.66; p<0.001$], disability words [$F(1,165)=25.661, p<0.001$] or affective words [$F(1,165)=15.40; p < 0.001$]. There were no significant differences between responses to the other words depending on the position of the target and probe ($p > 0.22$). The four-way interaction of FOP, word type, probe position, and word position was not significant [$F(6,164)=.86, p>.52$].
In order to interpret these results in the framework of attentional bias, the formula (described above) was used to calculate indices of attentional bias for each group of words (Keogh et al., 2001a). A two-way ANOVA was conducted with type of bias indices as within subject variables (affective vs. disability vs. sensory vs. threat) and fear of pain (high vs. medium vs. low) as the between group factor. Consistent with the previous analyses, a significant within subject effect for word type was revealed (F(3,165)=11.82, p<.0001). Pair-wise mean comparisons using bonferroni corrections indicated that participants showed a positive bias towards sensory-pain stimuli relative to affective-pain (F(3,165)=11.82, p<.0001), disability-pain (F(3,165)=11.82, p<.0001), and threat-pain stimuli (F(3,165)=11.82, p<.0001) (see figure 4.5). The interaction of bias indices and FOP was not significant (F(6,165)=0.82, p>0.05).
Figure 4.5 Pattern of attentional biases for different type of words.

0 = no selective bias (i.e. responses are equal for control and experimental stimuli); a positive score = bias towards experimental stimuli, and a negative score = biased attention away from experimental stimuli.

Since anxiety sensitivity has been argued to underlie pain-related cognitive biases, patients were also divided into three groups according to their scores on the ASI in the way that the fear of pain groups had been. The same 4 x 3 x 2 x 2 MANOVA was performed with anxiety sensitivity as the between group factor, which revealed no significant effects.
4.4 Discussion

We hypothesized that chronic pain patients would demonstrate an attentional bias towards pain-related words. The results provide strong evidence of selective attention for chronic pain patients as a group. However, this bias was limited to sensory pain words and was not observed for disability-related words, affective words or threat-related words. There were no effects of anxiety sensitivity on selective attention, confirming Keogh et al.'s (2001a) finding that anxiety sensitivity is not associated with selective attention to pain words. In addition, the hypothesis that participants with high levels of fear of pain would show an increased attentional bias was not confirmed. In fact, patients with high fear of pain were slower in responding to stimuli than the low or medium fear of pain groups. Moreover, the slowing of responses was evident not only for sensory words, but also disability and threat-related stimuli but not affective pain words. The attentional bias towards sensory words found in the present study was observed in all three fear of pain groups.

The finding that the bias demonstrated in the present study was specific to sensory words is consistent with the literature. Crombez et al. (2000) found that the specific nature of stimuli was an important determinant of attentional biases. Further, studies investigating the recall of pain-related information have consistently found recall biases for sensory pain information (e.g. Johnson & Spence, 1997, Edwards et al, 1992). However, Edwards et al. (1992) failed to find the predicted biases for affective pain words. The present study supports the view that affective pain words are processed differently to sensory pain words. This explanation is theoretically appealing, since theories suggest that it is fear of
(re)injury that sensitises patients to pain-related information (Vlaeyen et al., 1995a). Hence, any survival benefit of hypervigilance, would be related to attending to pain itself (i.e. the sensory aspect) rather than the emotional consequences of pain. This interpretation also concurs with results suggesting that cognitive biases towards negatively valanced health words (similar to pain affective words) occur only in depressed pain patients (Clemmey & Nicassio, 1997, Pincus et al., 1995). These results support Pincus and Morley’s (2001) proposition that there are two separate schema relevant to chronic pain: pain schema (i.e. response to sensory pain stimuli) and illness schema (response to affective pain words, disability and threat words).

While the general selective attentional bias found in this study is consistent with the literature, the results with regard to fear of pain are not. Keogh et al. (2001a) found that high FOP individuals responded more quickly to targets when preceded by pain-related words than medium or low fear of pain groups. Our results contradict this finding and demonstrate slower responding amongst highly pain-fearful participants. The task used in this study was based on Keogh et al.’s methodology (2001a). Therefore, methodological differences are unlikely to account for the differences in results. The major difference between the two studies is the sample employed. Keogh et al. (2001a) used healthy undergraduate students, in contrast to our sample of chronic pain patients. It may be that in the absence of pain, high fear of pain predisposes individuals to selectively attend to pain-related stimuli. However, in the presence of pain, people high in fear of pain have difficulty disengaging from competing tasks. This might apply especially to those seeking help for their pain, as in the present sample.
An alternative explanation for the conflicting findings is the lower levels of FOP observed in our chronic pain sample in comparison to Keogh et al.’s (2001a) sample. Only 6% of our sample would have fallen into the high FOP group, had we used the same criterion. Therefore the majority of those deemed in the present study to be high in FOP would have fallen in the moderate FOP group in Keogh et al.’s (2001a) study. These differences may be responsible for the lack of differences on attentional bias found between fear of pain groups in our study.

The finding that chronic pain patients have lower fear of pain scores than undergraduates was unexpected in itself and deserves comment. It may be that because our chronic pain group are in constant pain, they habituate to anxiety associated with pain and hence become less fearful of the pain. The nature of fears may, as a result shift, to fear of (re)injury (for example) and hence fear of pain, per se, becomes less theoretically important once pain becomes chronic. These results suggest that the natural development of fear of pain in acute and chronic pain states warrants future research.

Nonetheless the finding of slowed response amongst the relatively high fear of pain group is interesting and potentially of theoretical importance. Similar findings have been reported in the investigation of attentional biases in social anxiety. Fox (2002) found that under conditions of social threat, those high in social anxiety respond more slowly to threatening facial expressions. The present findings are similar. That is, those high in fear of pain, all of whom are constantly in pain, responded more slowly to stimuli. This is a post-hoc interpretation of an unexpected finding and should be interpreted cautiously. However, this explanation is consistent with the literature demonstrating that chronic pain patients have problems with attention and concentration (e.g. Eccleston &
Crombez, 1999). That is, if patients attend to pain and cannot disengage from it, then attention to pain will compete with other attentional demands, resulting in general problems with attention and concentration.

The present study found no evidence of an effect of anxiety sensitivity on cognitive biases. Asmundson et al. (1997) found that anxiety sensitivity was associated with biases in chronic pain patients, whereas Keogh et al. (2001a) found no association between anxiety sensitivity and cognitive biases. Our results suggest that, it is the specific fear of pain, and not anxiety sensitivity, that is relevant to attention amongst chronic pain patients and support the fear of (re)injury model (Vlaeyen et al., 1995a).

It must be noted that the present study cannot determine whether the attentional biases are the cause of chronicity or the consequence. However, in a recent study using university students, Roelofs et al. (2002) failed to find predicted Stroop effects when they experimentally induced fear and pain levels and suggested that attention to pain-related stimuli may be a response to pain, rather than a cause. Future research with individuals with acute injuries is needed to determine causality. Nonetheless, the present results suggest that attentional bias may be one factor that contributes to the maintenance of chronicity in chronic pain.

The present study was designed to overcome the methodological problems of previous research. Specifically, the strengths of this study include a large sample of consecutive patients, the use of a modified dot-probe task, the inclusion of different pain-related word categories and the inclusion of a range of
questionnaires. Nonetheless, some methodological limitations should be acknowledged. Firstly, the present study did not include a healthy control group. Comparisons reported here indicate that there is a bias towards sensory pain material relative to other pain-related material. However, we cannot conclude that a healthy control group would not demonstrate the same bias. In fact, control groups drawn from hospital staff have been found to show a bias, which is assumed to relate to a frequency effect from exposure to pain-related words in the course of work (Wells et al., 2003). Nonetheless, since other studies have failed to find such biased information processing amongst controls (e.g. Herbert, 1992, Pincus et al., 1998) this explanation is unlikely, but inclusion of well-matched controls would provide more confidence in the results.

A further limitation is that most participants completed the questionnaires before the dot-probe task. It is possible that the questionnaires about pain primed participants. While this explanation cannot be excluded, this should have affected other pain-related words equally, particularly since sensory words occurred less frequently than other pain-related words in the questionnaires.

Despite these limitations, the present study provides strong support for an attentional bias towards sensory pain words in a large, representative sample of chronic pain patients. These results confirm that the nature of pain-related stimuli influences participants’ responses. In the present study, fear of pain increased latency of response, which may suggest highly pain-fearful patients, currently in pain, not only attend to pain-related stimuli, but have difficulty disengaging from that stimuli (Van Damme, Crombez, and Eccleston, 2002). These results have implications managing chronic pain problems. If patients’ selective attention to
painful sensations maintains chronic pain, then interventions that reduce hypervigilance should be helpful. Cognitive-behavioural interventions have such an aim and future research should determine whether or not these biases are changed following intervention.
Chapter Five

Is selective attention to sensory pain-related words limited to chronic pain patients?

5.1 Introduction

A substantial body of research suggests that chronic pain patients show cognitive bias towards pain-related information at the expense of processing non-pain-related stimuli (Crombez et al., 1999). However, while there is good evidence for memory and interpretation biases in comparison to healthy controls (Edwards et al., 1995; McKellar et al., 2003; Moss-Morris and Petrie, 2003), studies on attentional biases have produced conflicting results (Pincus and Morley, 2001, 2002). In relation to selective attention, there is some evidence that chronic pain patients are hypervigilant to sensory-pain type information (Pearce & Morley, 1989; Crombez et al., 2000; Snider et al., 2000) in comparison to healthy control subjects. Although recently, other studies have failed to replicate these findings (e.g. Pincus et al., 1998) and it has been demonstrated that observed effects were not as robust as those found in anxiety disorders. However, the previous chapter has reported a robust sensory attentional bias in a large sample of chronic pain patients relative to other pain-related words and matched neutral words. Nevertheless, as the authors acknowledged, due to lack of healthy-control subjects, it would not be concluded that these biases were specific to people with chronic pain.
This caution is important for at least two reasons. Firstly, although Pearce and Morley (1989), Snider et al. (2000), and beck et al. (2001) found attentional biases in chronic pain patients relative to controls, others have found chronic pain patients to be equivalent to controls (Pincus et al., 1998; Duckworth et al., 1997; and Asmundson et al., 1997; Roelofs et al., 2003). Secondly, Wright and Morley (1995) have suggested that theoretically, priority processing of pain-related information may play a vital role in the survival of an organism. Hence, there may be survival benefit in hypervigilance to sensory pain stimuli, which might explain the lack of significant differences between chronic pain patients and controls. For example, Koutantji et al. (1999) compared memory for pain-related words in children with arthritis and healthy matched controls. Both groups exhibited a memory bias for pain sensory words but not for affective or neutral words. These findings suggest that, although chronic pain patients may have an attention bias to sensory pain-related words, it does not necessarily rule out the possibility that healthy pain free people also demonstrate similar biases.

The aim of the present study is to determine whether the attentional biases towards pain-sensory words are also evident in a healthy control group. If pain-related biases are evident in both groups, this would be consistent with Wright and Morley’s (1995) view that all people have biases to pain-related information which could be explained by evolutionary advantage. Alternatively, if these biases are not observed in the control group, this would favour theoretical models suggesting that hypervigilance to pain has a role in the etiology and/or maintenance of chronic pain.
5.2 Method

5.2.1 Design

A mixed-model 2 (chronic pain patients vs. healthy controls) x 4 (affective-pain vs. disability vs. Sensory-pain vs. pain-threat words) ANOVA was used for the analysis.

5.2.2 Subjects

Thirty-five healthy people, who reported experiencing no pain during the previous 3 months, and no history of chronic illness, volunteered to participate in the study as a healthy control group. The control participants were recruited through advertisements at a public hospital, a university campus, and a community library. Volunteers were interviewed to ascertain they did not have a current drug or alcohol problem, and were fluent in English, but no one needed to be excluded on the basis of these criteria.

A consecutive sample of 168 chronic pain patients (87% recruitment rate) had been recruited from a tertiary pain management centre, at the Royal North Shore Hospital, as reported in chapter 4. Data from the attentional biases identified in the entire sample have been reported (chapter 4). For the purposes of the present study, 35 of the original 168 patients were matched to the control group according to for gender, age (± 2 years) and as closely as possible for education (tertiary degree, completed high school, more than 10 years education, less than 10 years).

5.2.3 Measures

All participants completed the following measures.
5.2.3.1  Anxiety Sensitivity Index (ASI)
Anxiety Sensitivity Index (Peterson and Reiss, 1992), measures fear of anxiety-related symptoms (Asmundson, 1999). The ASI has 16 items with a total score ranging from 0 to 64. Participants rate the degree to which each statement describes them on a 5-point rating scale. Higher scores mean that the person is more fearful of anxiety sensation. Validity and reliability of ASI have been established and reported (Peterson and Reiss, 1992).

5.2.3.2  Fear of Pain Questionnaire-III (FPQ-III)
Fear of pain was measured using FPQ-III developed by McNeil and Rainwater (1998). FPQ-III includes 30 items resulting in a total score ranging from 30-150. Reliability and validity of FPQ-III has been reported (McNeil and Rainwater, 1998; Turk and Melzack, 2001).

5.2.3.3  Depression, Anxiety, Stress Scales (DASS)
Depressed mood and negative affectivity have been suggested as confounding variables in studies of cognitive bias (Pincus et al., 1998). The DASS (Lovibond and Lovibond, 1995) has shown reliability to differentiate depression, anxiety, and stress (Crawford and Henry, 2003). It contains 42 items scoring from 0 to 3, with a total score for each subscale between 0-42.

5.2.3.4  Selective attention measure
The dot probe task is identical to that described in chapter four. In this program, a fixation point appeared on the monitor for 500ms, and then a pair of words was presented one above the other for 500ms. The fixation point reappears following presentation of the words for a further 500ms in order to facilitate concentration. When the fixation point disappears, the letters “p” or “q” are presented in random
order. Subjects are required to respond by pressing either the “p” or “q” button as quickly as possible after the target has appeared. As soon as the response is made, the letter disappears. If the patient makes no response in 1500ms, the next item is automatically presented. The words that constituted the stimuli in the present study are presented in table 5.1.

Table 5.1 Word pairs used in the Dot-probe Task

<table>
<thead>
<tr>
<th>Affective/Neutral</th>
<th>Disability/Neutral</th>
<th>Sensory/Neutral</th>
<th>Threat/Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vicious/lessons</td>
<td>vulnerable/afterwards</td>
<td>flickering/waterfalls</td>
<td>rushing/elephant</td>
</tr>
<tr>
<td>Annoying/chivalry</td>
<td>suffering/expensive</td>
<td>throbbing/sailboat</td>
<td>fearful/tribune</td>
</tr>
<tr>
<td>Miserable/undertake</td>
<td>disable/semester</td>
<td>shooting/drinking</td>
<td>frightful/flowering</td>
</tr>
<tr>
<td>Troublesome/restraining</td>
<td>paralysed/detectors</td>
<td>boring/swivel</td>
<td>terrifying/innovative</td>
</tr>
<tr>
<td>Unbearable/metabolite</td>
<td>unhealthy/timetable</td>
<td>drilling/whirling</td>
<td>killing/crystal</td>
</tr>
<tr>
<td>Cruel/drums</td>
<td>ill/joy</td>
<td>sharp/items</td>
<td>suffocating/advertisers</td>
</tr>
<tr>
<td>Tiring/cotton</td>
<td>sick/rear</td>
<td>burning/moment</td>
<td>scared/garage</td>
</tr>
<tr>
<td>Exhausting/blackberry</td>
<td>inactive/flooring</td>
<td>stiff/skirt</td>
<td>danger/waited</td>
</tr>
<tr>
<td>Punishing/advocates</td>
<td>chronic/grasped</td>
<td>tugging/refresh</td>
<td>harmful/airmail</td>
</tr>
<tr>
<td>Discouraging/subcommittee</td>
<td>injury/mirror</td>
<td>pinching/postmark</td>
<td>threat/golden</td>
</tr>
</tbody>
</table>

5.2.4 Procedure

Participants were given a subject information sheet about the study to read, and then signed a consent form. Demographic information was collected from participants. Participants were then asked to complete the battery of questionnaires. Following their completion, the dot probe task was presented. The
study was approved by the Human Ethics Committees at the Hospital and The University of Sydney.

### 5.2.5 Analyses

All data analyses were conducted using SPSS 11. Indices of attentional bias were computed for each group of words using the following formula, frequently applied in attentional bias research:

\[
\text{Index} = \frac{(tupl - tlpl) + (tlpu - tupl)}{2}
\]

T= target word, p= probe, u= upper location, and l= lower location.

The product of this formula is an index of the interaction of word type (experimental vs. neutral), word position (upper vs. lower location), and probe position (upper vs. lower location). A positive index indicates attentional bias towards the stimuli, while a negative index means attentional bias away from stimuli (Keogh et al., 2001b).

### 5.3 Results

#### 5.3.1 Demographic features

The sample for the present study comprised 35 control participants and 35 matched chronic pain patients. Twenty of each group were male (57%) and the mean age of the samples was 41.5 years (SD = 15.2, range 23 – 75). Forty percent of the patients and 69% of the healthy control sample had a tertiary degree; 36.4% of the patients and 23% of the controls having completed high school; while 22.9% of patients and 8% of controls had ten years of formal education or less. Chi-square analysis revealed no differences between chronic
pain patient group and healthy controls in terms of education $[\chi^2 (4) = 4.87, p = 0.3]$ or marital status $[\chi^2 (5) = 7.07, p = 0.21]$, however, groups were significantly different in work status $[\chi^2 (5) = 27.14, p < 0.001]$ with 48.6% of the patient group currently unemployed versus only 2.9% of controls. Demographic features are presented in Table 5.2.

Table 5.2 Presents the demographic characteristics of experimental groups.

<table>
<thead>
<tr>
<th></th>
<th>Pain patients</th>
<th>healthy controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>Mean 42; SD 14.9</td>
<td>Mean 41.5; SD 15.2</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degree</td>
<td>16 (40%)</td>
<td>24 (69%)</td>
</tr>
<tr>
<td>&gt;=11 years</td>
<td>12 (36.4%)</td>
<td>8 (23%)</td>
</tr>
<tr>
<td>&lt;=10 years</td>
<td>8 (22.9%)</td>
<td>3 (8%)</td>
</tr>
<tr>
<td><strong>Work status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full-time</td>
<td>8 (22.9%)</td>
<td>22 (65%)</td>
</tr>
<tr>
<td>Part-time</td>
<td>4 (11%)</td>
<td>5 (15%)</td>
</tr>
<tr>
<td>Retired</td>
<td>5 (14%)</td>
<td>2 (5.7%)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>17 (49%)</td>
<td>1 (2.9%)</td>
</tr>
<tr>
<td>Others</td>
<td>1 (2.9%)</td>
<td>4 (11%)</td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>9 (28%)</td>
<td>14 (41%)</td>
</tr>
<tr>
<td>Married</td>
<td>17 (50%)</td>
<td>17 (50%)</td>
</tr>
<tr>
<td>Defacto</td>
<td>3 (8.8%)</td>
<td>3 (8.8%)</td>
</tr>
<tr>
<td>Separated/Widow/divorced</td>
<td>6 (17%)</td>
<td>1 (2.9%)</td>
</tr>
</tbody>
</table>
5.3.2 Self-report measures

Means and Standard deviations (SD) of self-report measures for each questionnaire are presented in table 3 for the two experimental groups. A series of t-tests were conducted to examine group differences across all self-report measures. Highly significant differences were found for all subscales of DASS as follows: depression \([t(1, 66)= 4.46, p<.001]\); anxiety \([t(1,66)=4.12; p<.001]\); stress \([t(1,66)=3.6; p<.001]\). There was a trend for groups to differ on ASI scores, but this did not reach statistical significance \([t(1,68)=1.86; p<.07]\), but no differences were observed on scores of the FPQ \([t(1,68)=2.2; p=0.8]\). All differences indicated that the patient group experienced significantly more symptoms of distress.

Table 5.3 Means (SDs) of the chronic pain patients and healthy controls on self-reported measures.

<table>
<thead>
<tr>
<th>Measures</th>
<th>Patients</th>
<th>Controls</th>
<th>Ps</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASI</td>
<td>18 (11.5)</td>
<td>13 (8.1)</td>
<td>.07</td>
</tr>
<tr>
<td>FPQ</td>
<td>66 (17)</td>
<td>67 (18.3)</td>
<td>.8</td>
</tr>
<tr>
<td>DASS-D</td>
<td>15.6 (12.8)</td>
<td>4.3 (7.2)</td>
<td>.001</td>
</tr>
<tr>
<td>DASS-A</td>
<td>9 (8)</td>
<td>2.8 (3.6)</td>
<td>.001</td>
</tr>
<tr>
<td>DASS-S</td>
<td>17.8 (10.7)</td>
<td>8.9 (9.2)</td>
<td>.001</td>
</tr>
</tbody>
</table>

ASI: anxiety sensitivity index, FPQ: Fear of pain Questionnaire, DASS: Depression, Anxiety, Stress Scale
5.3.3 Dot probe task

Individual reaction times for each patient were collected and grouped by word type, word location, and probe position. All reaction times faster that 200ms and slower than 1000 were considered as outliers and removed (Keogh et al., 2001b). Applying the attentional bias index formula as described before, attentional indices for both groups were calculated.

Since experimental groups differed significantly on mood-related measures (i.e. anxiety, stress, and depression), it was decided to control for the possible effects of mood on attentional bias (Pincus and Morley, 2001). Questionnaire measures of anxiety, stress, depression and anxiety sensitivity were highly correlated (i.e. $r = 0.52 – 0.84$, $p < 0.05$). In order to determine the possible influence of mood on attentional bias, inter-correlations between the questionnaire measures and attentional indices were conducted. None of the measures of mood, according to the DASS, were associated with attentional bias scores ($r < .20$, $p > 0.05$). Neither was anxiety sensitivity significantly correlated with bias scores ($r < .09$, $p > 0.05$). However, fear of pain was significantly associated with sensory pain index ($r = 0.26$, $p < 0.05$), but not other indices of selective attention ($r > .07$, $p > 0.05$). Since fear of pain was not significantly different between the groups, there seemed no reason to control for its effects in the subsequent analyses. It was also considered unnecessary to control for each of the DASS measures in the analyses, since they were strongly inter-related and not associated with the dependent variable. Anxiety was more consistently related to each of the other DASS measures (i.e. $r = 0.68-.69$), and ASI ($r = 0.69$). Therefore to ensure that the
differences between groups could not influence the results of the analyses, scores of anxiety were included as a covariate in the following analyses. Hence, a 2x4 mixed model ANCOVA was conducted.

A significant main effect was found for word type indices \( [F(3,63)=6.68, p<.001] \), but there was no significant main effect for anxiety as covariate \( [F (3,63)=.83, p>.05] \). The interaction effect of groups (patients vs. controls) by attentional bias indices (affective vs. disability vs. sensory vs. threat) was also not significant \( [F(3,66)= .24, p>.05] \). Post hoc analyses revealed that both groups selectively attended to sensory-pain words compared to affective-pain words \( [F(1,68)=5.8, p=.019] \), disability words \( [F(1,68)=26.9, p=.001] \), and pain-threat words \( [F(1,68)=11.5; p=.001] \). There was no significant effects for affective vs. threat indices \( [F (1,68)= 3.25, p>.05] \) or disability vs. threat \( [F(1,68)=.4, p>.05] \). The contrast between affective and disability indices was significant \( [F(1,68)=10.3, p=.002] \). Figure 5.1 depicts the pattern of attentional biases in both experimental and control groups.
5.4 Discussion

The aim of the present study was to examine whether attentional biases to pain-related information were specific to chronic pain patients or were also present in a healthy population. Chronic pain patients were compared with matched healthy controls on their reaction times to affective, disability, sensory, and threat word types. The results suggested that the healthy control group exhibited a similar pattern of attentional biases to those observed in chronic pain patients. That is, both healthy people and those in chronic pain show a significant attentional bias towards sensory pain-related information in comparison to other types of pain words.

Despite the similarities in attentional biases between the chronic pain group and the healthy controls, the (non-significant) differences were in the predicted direction. In fact, the effect size calculated in the present study is 0.28, which is consistent with the median effects sizes (0.23) reported by Pincus and Morley...
(2001) in their review of the literature. In that sense, the present results could also have been consistent with the conclusion drawn by Pincus and Morley (2001) that chronic pain patients show a relative selective attentional bias towards sensory pain-related stimuli in comparison to healthy controls. However, as the differences are modest, a larger sample of participants (at least 64 in each group) would be required for that difference to reach statistical significance (Cohen, 1992).

Despite the modest difference between chronic pain patients and healthy controls in the extent to which the preferentially attend to sensory related pain stimuli, the present results suggest that both groups show a clear bias towards sensory related pain words, as opposed to the other stimuli tested. It is interesting to consider why a healthy control group would selectively attend to one set of pain-related words and not other types of pain-related words.

Wright and Morley (1995) have suggested that there might be an evolutionary advantage to preferential processing of pain-related stimuli. That is, if an organism perceives a threat in the form of a painful injury and responds quickly, the chance of that injury being compounded through continued use of the injured area may be reduced. Hence, there could be a survival advantage to the organism of being hypervigilant towards painful sensations.

The differential effect between sensory pain words and the other pain words studied (affective, disability, and threat-related words), is consistent with earlier findings comparing sensory pain words and affective pain words (Pearce and Morley, 1989; Pincus et al, 2000; Denton et al., in press). Pincus and Morley (2001) have conjectured that this differential effect may be related to the potential threat value of these different classes of pain words. Thus, sensory words might
be seen to reflect a close relationship to injury, while affective and disability pain words might be seen as associated with more distant consequences of injury and therefore less immediate as a threat.

This interpretation would be consistent with findings reported by Snider et al. (2000) who showed that those who worried more about pain were more likely to demonstrate attentional biases than those who worried less about their pain. Similarly, in healthy populations, those high in fear of pain have been found to selectively attend more to pain-related stimuli than those who score low or medium in fear of pain (Keogh et al., 2001a).

Pincus and Morley (2001) suggest that sub-groups of chronic pain patients, such as those who are depressed, anxious or fearful of pain, might show selective attention towards pain-related information relative to chronic pain patients in general. In the present study, no differences were found between the pain and healthy groups for fear of pain, although highly significant differences between groups were found on all other psychological measures. At the same time, inter-correlations between the various psychological measures and attentional bias for sensory pain-related words revealed that the only significant relationship was between fear of pain and selective attention. The finding that the two groups studied did not differ in fear of pain might explain the lack of significant differences between the groups for attentional bias.

While this explanation would be consistent with Keogh and colleagues (2001a) and Snider and colleagues (2000) in a healthy group and a chronic pain group,
respectively, the explanation is inconsistent with the findings from chapter 4. The earlier results failed to demonstrate that fear of pain differentiated between a large sample of chronic pain patients in attending to pain-related words. In fact, there was some evidence to suggest that those who were highly fearful of pain had longer latencies in responding to targets preceded by pain stimuli. Nonetheless, as in the present study, the fear of pain level in the chronic sample was unexpectedly low, which may have affected the results. Future research should investigate both healthy subjects and chronic pain subjects who demonstrate high and low levels of fear of pain and the degree to which they selectively attend to pain-related information.

If attentional biases towards pain cues are in fact ‘normal’, then one might question whether the construct of hypervigilance has any theoretical or practical interest in understanding the etiology, maintenance or management of chronic pain. Wright and Morley (1995) have argued that attention towards pain may have survival benefit to healthy people. The question remains whether this is also the case for chronic pain patients. Selective attention might be helpful for an acute injury, where identification of the injury and its source and successful avoidance of danger is adaptive. In chronic pain, it is generally accepted that avoidance of pain-provoking activities is not adaptive and may contribute to the development of disability (Vlaeyen & Linton, 2002). That is, many chronic pain patients, despite the duration of their pain, still seem to behave as though their pain is acute (Turk and Okifuji, 2002). It may be that sensitivity to pain stimuli has adaptive value for healthy people, however, when pain becomes chronic, sensitivity to pain is unhelpful (Janssen, 2002). Indeed, Pincus and Morley (2001) suggested that
studies of those with very recent injuries are needed to determine the actual role of attentional bias in the development of chronic pain.

Despite attention to methodological rigor, certain methodological constraints of the present study warrant discussion. Firstly, chronic pain patients who participated in the study were all taking medication, whereas none of the control group were. Medication use, especially long-term use, might be a confounding factor in comparing chronic pain patients with healthy controls in terms of cognitive functioning. A variety of medications used by chronic pain patients may impact on patients’ cognitive processes (Duckworth et al., 1997). Although any patients who were judged by the investigators to be markedly affected by medication were excluded from this study, the chronic pain sample used may nonetheless have been affected by analgesia and/or antidepressants in a way that was not directly observable but did influence their response times. Although this may have affected the latency of responses, one might expect it to affect response latencies equally across all word types and positions. The use of the bias index, rather than raw reaction times, should have minimized the impact of medication differences between the groups.

The second limitation of the present study is that both chronic pain patients and healthy subjects completed the questionnaires before completing the dot probe task. This was unavoidable in the chronic pain group due to the nature of their clinical assessment, and therefore we also administered the questionnaires first for control participants to keep the procedure standardized. Nonetheless, the potential for a priming effects exist. It is also possible in the case of chronic pain patients that the presence of pain had already primed pain-related information. However, for healthy controls, the questionnaires may have primed a response that would
not otherwise have been present. Future research should, if possible, administer selective attention tasks before presenting questionnaires.

Despite these methodological limitations, the results of the present study show that chronic pain patients and healthy controls both exhibit attentional biases to sensory pain stimuli in comparison to other types of pain words and neutral words. However, there is some evidence that this bias might be more pronounced for chronic pain patients. Further, we found that this attentional bias is independent of mood. While the chronic pain group reported higher levels of distress, such as depression and anxiety, neither mood state was associated with attentional bias index. Contrary to expectations, both groups were almost identical in scores on fear of pain. Indeed, fear of pain was the only self-report measure associated with selective attention variable. The lack of differences in fear of pain may have contributed to the lack of differences between the groups.
Chapter Six

Modification of attentional biases in chronic pain patients: a preliminary study

This chapter has been submitted to the European Journal of Pain as follows:


6.1 Introduction

Recent models of chronic pain emphasize specific fear of (re)injury in the maintenance of chronic pain and associated disability (Vlaeyen et al., 1995b). Specifically, when people become injured, those who are highly fearful of pain may focus more on the pain and interpret it as harmful. In turn, this group may respond by avoiding many daily activities. In the short-term, this strategy may provide relief from pain (Phillips, 1987). However, in the longer term, this strategy can lead to increased disability and the development of a vicious cycle between attention to pain, pain-related fear and activity avoidance.

This model has played an influential role in the development of cognitive-behavioural approaches (CBT) to the management of pain. One aim of CBT is to modify unhelpful cognitions about pain, such as pain signifies damage. Additionally, CBT encourages patients to increase and perform previously avoided physical activities, through engagement in exercise programs and other activities. Typically CBT is conducted in a multi-disciplinary setting and includes skills, such as activity pacing and goal setting, exercise, medication reduction, problem solving, cognitive restructuring and relaxation training. There is
considerable evidence that CBT is an effective treatment for chronic pain patients (Morley, Eccleston, and Williams, 1999; Guzman et al., 2001).

Although the effectiveness of CBT has been established, there is considerable debate about the mechanisms of change. McCracken and Turk (2002) reviewed the prediction of outcome literature for CBT and BT in chronic pain. Their review suggested that a range of different variables were associated with treatment-related changes, depending on the outcome measure used. Studies have used a variety of outcomes (e.g. pain, disability or distress) with differing results, making the mechanisms of change more difficult to understand. Nonetheless some factors, such as beliefs about pain, coping strategies, or emotional responses, have been cited more consistently than others as associated with treatment change. McCracken & Turk (2002), concluded there was consistent evidence pointing to anxiety-related constructs as important factors in mediating the response to treatment. This was illustrated with a recent well-controlled prediction of outcome study that found changes in pain-related anxiety during CBT were associated with improvement in a variety of measures, including pain-severity, physical activity, and depression (McCracken, Gross, and Eccleston, 2002).

The fear of pain/(re)injury model of chronic pain (Vlaeyen et al., 1995a) suggests that the mechanism through which fear increases disability is hypervigilance to painful sensations. This model predicts that fear of pain/(re)injury causes attentional biases towards pain which leads to avoidance of pain-related activities and disability. However, Turk (2002) and Turk and Okifuji (2002) argued that self-efficacy may mediate between fear avoidance and disability.
Research supports the presence of cognitive biases towards threat-related information in anxiety disorders (Williams et al., 1997), however evidence of pain-related biases in chronic pain patients, in the chronic pain literature has less consistent (Pincus & Morley, 2001). Pearce and Morley (1989) and Snider, Asmundson, and Weise (2000) found attentional biases towards pain-related words, but others failed to find evidence of the predicted biases (Pincus, Fraser & Pearce, 1998; Crombez, Hermans & Andriansen, 2000, Asmundson, Kuperos, Norton, 1997). Pincus & Morley (2001) suggested that methodological limitations in these studies may account for some of these different findings. For example, the paradigm employed by most studies (i.e. the Stroop) is problematic as attentional biases cannot be differentiated from response biases (MacLeod, Mathews & Tata, 1986). Similarly, different studies used different words with little attempt to demonstrate the relevance of the stimuli to the sample. Importantly, sample sizes in most studies have been small and lacking statistical power (n=16-42).

In chapter four we reported a study using a large (n=168), consecutive sample of chronic pain patients. The results demonstrated evidence of attentional bias specifically for sensory pain words, but not other pain-related words. This finding is consistent with the two other studies (Pearce and Morley, 1989; Snider, Asmundson, and Weise, 2000) that have found similar biases towards sensory pain words. Both studies that found significant biases employed sensory pain words. Indeed, a recent meta-analysis found that chronic pain patients are biased towards sensory pain stimuli more than other pain words (Roelofs et al., 2002). Therefore, it seems likely that chronic pain patients do selectively attend to
sensory pain-related stimuli but the effect sizes are smaller than those observed in anxiety disorders.

Cognitive biases, when present, have been found to predict patient functioning. Pincus and Newman (2001) showed that cognitive biases significantly predicted future health care utilization, increased pain and relapse. Such findings support the argument that changes in cognitive biases may be an important target in treatments (Pincus and Morley, 2001).

It might be expected that CBT could change cognitive biases associated with chronic pain. Changing maladaptive cognitions is one of the major aims of CBT. It is assumed that by changing fear of pain, individuals will attend less to pain, approach activity more and begin the process of recovery. Further, in anxiety disorders, cognitive biases have been demonstrated to change following CBT (Mathews et al., 1995). Keogh et al. (2001a) argued that, in a similar way, if pain-related fear mediates attention to pain sensations, then interventions that target these unhelpful thoughts should change selective attention towards pain-related stimuli. To date, however, no study has investigated whether CBT produces change in attentional biases in chronic pain patients.

The present study aimed to address the following question: Does an intensive, multi-disciplinary cognitive-behavioural pain management program lead to changes in selective attention towards pain-related stimuli? We hypothesized that CBT will result in cognitive change not only at strategic thinking level (i.e. types of beliefs endorsed by the patient), but at an automatic level (i.e. selective attention to pain-related information). Further, we predicted that changes in fear of
pain/movement following treatment would be associated with subsequent changes in cognitive biases towards pain.

6.2 Method

6.2.1 Participants

The sample was recruited from patients attending a CBT program for chronic pain in Sydney, Australia. Between January and August 2002, chronic pain patients participating in six consecutive Pain Management Programs were approached and asked to participate in the study. Patients were included in the study if they were over 18 years, in constant pain for at least 3 months (IASP, 1986) and literate in English. Exclusion criteria were being unable to use both hands, a history of head injury, serious mental illness or having a current alcohol or illicit drug problem.

Sixty patients took part in these programs and 52 were eligible to participate. Four patients were under eighteen, one was unable to use both hands and one had had a head injury. Forty-five patients volcanereed for the study (86.5%). Three patients were later excluded due to head injury not diagnosed at the initial assessment (n=1) and incomplete data (n=2). Five patients withdrew during treatment (n=38) and 3 patients withdrew by follow up (n = 35). The study was approved by The University of Sydney and Hospital Ethics Committees. Participants gave written consent.

The mean age of the sample was 42 (SD:9.9) and the average duration of pain was 59 months (SD:63.8). Twenty-seven (64.3%) patients were married and 22 (52.4%) were female. English was the native language of 95.2% of the sample. Only seven (16.7%) were educated to University level, with 13 (31%) completing

* This sample is part of the sample reported in chapter four.
high school, and the remainder (52.3%) having less than twelve years of education. The distribution of pain by site was as follows. Sixteen (38.1%) had chronic low back pain, five (12%) had upper limb pain, five (12%) had pain in the lower limbs and five (12%) had pain in the cervical region. Most (64.3%) patients were unemployed, with 23.80% working full time, 9.5% part time and 2.4% retired. Thirty three (78.6%) were receiving workers’ compensation.

6.2.2 Procedure

Participants completed a battery of questionnaires (detailed below) prior to commencing the program, at the end of the program and one-month later. In addition, they undertook a computerised version of dot probe task on each occasion.

The Pain Management Program is a three-week intensive, multi-disciplinary, day-patient program, and patients attend a booster session four weeks later. During the three weeks, patients attend 5 days a week from 9a.m-5p.m. The content included education about pain, graduated exercises, applied relaxation training, training in pacing and goal setting, problem solving and cognitive restructuring. Medication reduction was also an important element of the program. One aim of CBT is to educate participants about physiological mechanisms and the consequences of inactivity. The emphasis is on disconfirming the beliefs that physical activity is harmful and encouraging patients to gradually resume avoided activities. Providing patients with an opportunity to disconfirm their fearful beliefs about movement plays a key role in CBT.
6.2.3 Measurements

6.2.3.1 Fear of Pain Questionnaire – III (FPQ-III) (McNeil and Rainwater, 1998)

FPQ-III is a 30-item questionnaire that measures fear of pain. Good psychometric properties have been reported (Turk and Melzack, 2001).

6.2.3.2 Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992)

The ASI is a 16-item questionnaire measuring sensitivity to anxiety-related sensations. High levels of internal consistency and good test-retest reliability have been reported (Peterson and Reiss, 1992, Asmundson, 1999).

6.2.3.3 Depression, Anxiety, and Stress Scale (DASS)

The DASS (Lovibond & Lovibond, 1995) is a 42-item measure of depression, anxiety, and stress. The DASS does not rely predominantly on somatic items, and is therefore less likely to be artificially inflated in a chronic pain setting. The reliability and validity of DASS have been well established (Lovibond & Lovibond, 1995).

6.2.3.4 Tampa Scale of Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990)

TSK consists of 17 items and each is scored on a 4-point Likert scale. TSK is a measure of specific fear of movement and has shown good validity and reliability (Vlaeyen et al., 1995b).
6.2.3.4 Pain severity
The pain severity subscale (3-item) of West Haven-Yale *Multidimensional Pain Inventory (WHYMPI)* (Kerns, Turk, & Rudy, 1985) was included to assess severity of pain. This instrument is a widely used and reliable research tool for assessing patients' responses to pain disorders.

6.2.3.5 Roland and Morris Disability Questionnaire (RDQ) (Roland & Morris, 1983)

RDQ is a 24-item check-list to determine the level of disability. The RDQ has good psychometric properties (Roland & Fairbank, 2000). In this study, we employed a modified version of the RDQ that is appropriate for patients with all pain sites (Asghari and Nicholas, 2001).

6.2.3.7 The Pain Responses Self Statements (PRSS) (Flor et al, 1993).

The PRSS was included to assess catastrophic pain-related cognitions. It consists of 18 items and psychometric properties of PRSS have been established (Flor et al., 1993).

2.3.7 Pain self-efficacy Questionnaire (PSEQ) (Nicholas, 1989): The PSEQ was administered to assess patients’ self-efficacy beliefs. Higher scores indicate higher self-efficacy. Good reliability and validity of the PSEQ has been reported (Asghari & Nicholas, 2001).
6.2.4 Dot probe task

A modified dot-probe program (as described in chapter 4 and 5) was developed using Visual Basic Program. All stimuli were presented on the monitor of an IBM 600X LAP TOP, Intel Inside, Pentium III. A fixation point “.” appeared on the centre of monitor for 500 ms and was replaced by a pair of words, one in the upper half and the other below the fixation point. The words remained for 500ms and were then replaced with another fixation point. The fixation point was replaced with either the letter “p” or “q”, which appeared randomly in the location of one of the words. Subjects were requested to press the “p” or “q” button as quickly as possible when they appeared. These cues disappeared when the subject pressed the key or after 1500ms if no response was given. Each word pair was presented on four occasions, so that each word/target combination occurred randomly. The time between the appearance of “p” or “q” and the participant’s key press was the dependent variable. A one-minute break was given to participants after 40 presentations, resulting in three breaks. Five pairs of practice words were presented before the task and two more were presented before each set.

The experimental stimuli were drawn from the earlier study reported in chapter 4 and are available in Table 4.1. The stimuli consisted of forty target/neutral words, which were divided into four sets to represent the following pain-related categories: sensory (e.g. shooting), affective (e.g. cruel), disability (e.g. paralysed) and threat (e.g. harmful).
6.2.5 Analyses

6.2.5.1 Self-report measures.

To investigate the treatment outcomes, a one-way repeated measures ANOVA (pre, post, and follow-up) was used to determine changes across time.

6.2.5.2 Attentional bias index.

Indices of attention bias were calculated for affective, disability, sensory, and threat words using the same formula presented in chapter 4 and 5. A positive score indicates selective attention towards the location of the target word, and a negative score indicates avoidance of the stimulus (Keogh et al., 2001b).

A repeated measures ANOVA with word type as the repeated measure was conducted on each assessment occasion to investigate differences in cognitive bias. In addition, a repeated measures ANOVA with time (pre-, post- and follow-up) as the repeated measure was conducted for each set of words.

6.2.5.3. Prediction of Outcome

Correlations between change in self-report measures during treatment and subsequent changes in the sensory attentional index were conducted. Variables correlated with change in sensory attentional index (as the dependent variable) were entered into a multiple regression analysis as predictors.
6.3 Results

6.3.1 Participants characteristics

The current sample had been in pain for at least 2 years (mean=5 yrs; SD= 5.4 yrs). Participants reported, on average, a moderate level of disability, pain, fear of movement, fear of pain and self-efficacy. However, patients reported high levels of stress and the average for depression was in the clinical range. In contrast, levels of anxiety and anxiety sensitivity were relatively low. (See Table 6.1).

Table 6.1 Psychological profile of patients at pre, post, and follow up sessions. Means (SD)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre</th>
<th>Post</th>
<th>F/up</th>
</tr>
</thead>
<tbody>
<tr>
<td>DQ</td>
<td>12 (5.4)</td>
<td>11 (5.6)</td>
<td>11.4(6.5)</td>
</tr>
<tr>
<td>MPI-sev</td>
<td>4.25(1)</td>
<td>3.85(1.1)</td>
<td>3.75(1.2)</td>
</tr>
<tr>
<td>Catas.</td>
<td>2.98(1)</td>
<td>2.53(1)</td>
<td>2.46(1.1)</td>
</tr>
<tr>
<td>PSEQ</td>
<td>24.3(13.8)</td>
<td>33.4(15.4)</td>
<td>29 (15.7)</td>
</tr>
<tr>
<td>TSK</td>
<td>39.6(10)</td>
<td>34.5(10)</td>
<td>36.6 (10)</td>
</tr>
<tr>
<td>ASI</td>
<td>23 (11.4)</td>
<td>22.6(12)</td>
<td>23 (15)</td>
</tr>
<tr>
<td>FPQ</td>
<td>71 (19.5)</td>
<td>68.5 (22)</td>
<td>66.9 (26)</td>
</tr>
<tr>
<td>DASS-A</td>
<td>11.2 (8.2)</td>
<td>12 (9.7)</td>
<td>9.8 (9.4)</td>
</tr>
<tr>
<td>DASS-D</td>
<td>20.4 (11)</td>
<td>16 (13.2)</td>
<td>16.4 12.6</td>
</tr>
<tr>
<td>DASS-S</td>
<td>22.4</td>
<td>17.6 (11)</td>
<td>16 (9.3)</td>
</tr>
</tbody>
</table>

DQ Disability Questionnaire; MPI- severity subscale; PRSS catastrophising subscale; PSEQ Pain Self-efficacy Questionnaire; TSK Tampa Scale of Kinesiophobia; ASI Anxiety Sensitivity Index; FPQ Fear of Pain Questionnaire; DASS anxiety, depression, and stress subscales. SDs in parentheses.

A repeated measures ANOVA was conducted to investigate the change from pre- to post-treatment and follow-up for each outcome measure. The results revealed...
significant reductions in pain severity ($F(2,31)=5.62, p=.008$), catastrophizing ($F(2,31)=6.22, p=.005$), pain self-efficacy ($F(2,31)=9.57, p=.001$), fear of re-injury ($F(2,31)=5.99, p=.006$), depression ($F(2,31)=6.65, p=.004$) and stress ($F(2,31)=7.56, p=.002$). There was no significant change over time in fear of pain, anxiety or anxiety sensitivity scores. Surprisingly, there was also no significant change in disability over treatment.

The failure of a well-validated CBT program to produce significant changes in disability was of concern. Inspection of scores indicated that a sizeable minority of this sample reported low levels of disability at pre-treatment ($n=16, 38.1\%$, DQ=$<10$) (vs. the average for the program which is 14). These low scores suggested that the analyses might be affected by a floor effect. To examine this possibility, the sample was divided into a low disability group ($<12$) and a high disability group ($>12$), using a median split. A mixed model 2x2 ANOVA was conducted with time (pre-treatment vs. post-treatment) as the within group factor, and disability level (low vs. high) as the between group factor. Results revealed a significant interaction effect between disability and time ($F(1,35)=4.34, p=.04$). Examination of the means suggested that the low disability group did not change their level of disability over treatment, whereas those high in disability improved significantly.

6.3.2 Attentional bias analysis

All reaction times below 200 ms and over 1000 ms were removed as outliers, as suggested by Keogh et al. (2001a). False/no responses constituted 5% of all responses and were removed from further analyses.
A repeated measures ANOVA was conducted with word type as the within-subjects variable (sensory, affective, disability, threat). A significant main effect was found for word type at pre-treatment \((F(3,39)=4.32, p=.01)\). Post-hoc analyses of mean differences revealed that chronic pain patients were positively biased towards sensory-pain stimuli relative to affective \((F(1,41)=12.06, p<.001)\), disability \((F(1,41)=9.12, p<.004)\) and threat-related pain words \((F(1,41)=5.64, p<.02)\).

Table 6.2 shows raw scores on dot probe at three occasions:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre-Test</th>
<th>Post-Test</th>
<th>Follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.TDPT</td>
<td>623(97)</td>
<td>578(99)</td>
<td>552(97)</td>
</tr>
<tr>
<td>A.TDPT</td>
<td>596(93)</td>
<td>568(102)</td>
<td>548(94)</td>
</tr>
<tr>
<td>A.TTPD</td>
<td>584(93)</td>
<td>562(92)</td>
<td>546(83)</td>
</tr>
<tr>
<td>A.TTPTD</td>
<td>570(100)</td>
<td>552(102)</td>
<td>538(92)</td>
</tr>
<tr>
<td>D.TDPT</td>
<td>599(87)</td>
<td>579(99)</td>
<td>555(90)</td>
</tr>
<tr>
<td>D.TDPT</td>
<td>580(85)</td>
<td>560(96)</td>
<td>541(93)</td>
</tr>
<tr>
<td>D.TTPD</td>
<td>598(95)</td>
<td>571(97)</td>
<td>552(87)</td>
</tr>
<tr>
<td>D.TTPD</td>
<td>585(93)</td>
<td>566(105)</td>
<td>539(85)</td>
</tr>
<tr>
<td>A.TDPT</td>
<td>587(85)</td>
<td>558(90)</td>
<td>561(91)</td>
</tr>
<tr>
<td>S.TDPT</td>
<td>593(85)</td>
<td>579(101)</td>
<td>550(83)</td>
</tr>
<tr>
<td>A.TDPT</td>
<td>613(93)</td>
<td>583(99)</td>
<td>563(94)</td>
</tr>
<tr>
<td>S.TDPT</td>
<td>579(92)</td>
<td>564(109)</td>
<td>534(82)</td>
</tr>
<tr>
<td>T.TDPT</td>
<td>581(95)</td>
<td>563(101)</td>
<td>554(88)</td>
</tr>
<tr>
<td>T.TDPT</td>
<td>584(92)</td>
<td>557(91)</td>
<td>542(96)</td>
</tr>
<tr>
<td>T.TDPT</td>
<td>595(82)</td>
<td>570(97)</td>
<td>566(88)</td>
</tr>
<tr>
<td>T.TTPT</td>
<td>597(92)</td>
<td>569(100)</td>
<td>545(93)</td>
</tr>
</tbody>
</table>

The same repeated measures ANOVA was conducted for cognitive biases at post-treatment, which revealed a significant main effect for word type \((F(3,35)=8.69, p<.0001)\). Post-hoc analyses again confirmed significant differences between the attentional biases towards sensory pain words and threat.
(F(1,37)=13.03, p < .001), disability (F(1,37)=21.43, p < .0001) and affective pain words (F(1,37)=10.57, p < .002). However, when indices of cognitive bias at follow-up were analysed, differences between word type were no longer significant (F(3,32)=.63, p=.60). Figure 6.1 summarises the pattern of biases for word types across time. These results suggest that while chronic pain patients preferentially processed sensory information at pre- and post-treatment, this pattern was not evident at follow-up.

In addition, a repeated measures ANOVA with time as the within subjects variable was conducted to compare sensory attentional biases at pre-, post-treatment, and follow-up. The main effect for time was significant (F(1,34)=4.11, p<.05). Further inspections revealed that although there was no significant change from pre- to post-treatment (F(1,34)=.29, p<.59), attentional...
bias to sensory words decreased significantly from time 1 to time 3 (F(1,34)=4.11, p<.05). There were no other significant differences in attentional biases for threat, disability of affective pain words.

Change scores were calculated for every outcome measure that significantly changed from pre- to post-treatment by subtracting scores at time 1 from time 2. Change scores were also calculated for cognitive bias to sensory pain words for changes from post-treatment to follow-up to allow possible predictions of the change in cognitive bias to be determined. Correlations between clinical change scores and cognitive change scores revealed that fear of pain/movement (TSK) (r=.37,p<.05) and catastrophising (PRSS) (r=.34,p<.05) were significantly correlated with change in sensory pain-related stimuli. No other change scores were significantly correlated with changes in the attentional bias towards sensory pain words. However, there was a correlation between gender and change in attentional bias (see Table 6.3).

Table 6.3 Inter-correlation of change scores for clinical measures, demographic features, and sensory index

<table>
<thead>
<tr>
<th></th>
<th>SEN</th>
<th>TSK</th>
<th>MPI</th>
<th>PSEQ</th>
<th>CATAS</th>
<th>DEP</th>
<th>STR</th>
<th>FPQ</th>
<th>ASI</th>
<th>SEX</th>
<th>AGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSK</td>
<td>.37*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MPI</td>
<td>.03</td>
<td>-.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSEQ</td>
<td>-.28</td>
<td>-.45**</td>
<td>.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CATAS</td>
<td>.34*</td>
<td>.32</td>
<td>.06</td>
<td>-13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DEP</td>
<td>.22</td>
<td>.30</td>
<td>-.06</td>
<td>-.22</td>
<td>.38*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STR</td>
<td>.11</td>
<td>.36*</td>
<td>.07</td>
<td>-.23</td>
<td>.50**</td>
<td>.68**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FPQ</td>
<td>-.01</td>
<td>-.28</td>
<td>.05</td>
<td>-.47**</td>
<td>-.07</td>
<td>-.15</td>
<td>-.15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td>-.18</td>
<td>.23</td>
<td>.002</td>
<td>-.23</td>
<td>-.14</td>
<td>.02</td>
<td>.08</td>
<td>-.26</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SEX</td>
<td>-.17</td>
<td>.29</td>
<td>.14</td>
<td>-.15</td>
<td>.08</td>
<td>.18</td>
<td>.14</td>
<td>-.36*</td>
<td>.35*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGE</td>
<td>-.17</td>
<td>-.03</td>
<td>.01</td>
<td>.10</td>
<td>.22</td>
<td>.09</td>
<td>.06</td>
<td>-.10</td>
<td>-.13</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>Dur</td>
<td>.02</td>
<td>-.10</td>
<td>-.14</td>
<td>.17</td>
<td>.19</td>
<td>-.01</td>
<td>.08</td>
<td>-.007</td>
<td>-.22</td>
<td>.17</td>
<td>.16</td>
</tr>
</tbody>
</table>

SEN: sensory index; TSK: Tampa Scale of Kinesiophobia; PSEQ: Pain self-efficacy questionnaire; CATAS: DASS depression; DEP: DASS stress; FPQ: fear of pain questionnaire; ASI: anxiety sensitivity index; Dur: duration of pain
A multiple regression analysis using simultaneous entry of variables was conducted to identify which variables independently predicted changes in sensory bias after treatment. Change score of sensory attentional bias index was entered as the criterion variable. Treatment-related changes in catastrophising, fear of movement (TSK), fear of pain (FPQ) and anxiety sensitivity (ASI) were entered as predictors. The regression equation model significantly predicted 29% of the variance in subsequent changes in sensory index \( [F(3,27) = 5.24, p=0.006] \). Only the change score for fear of movement significantly predicted independent variance in sensory index change \( (t=3.86, p=0.001) \). Post-hoc analyses indicated that TSK alone accounted for 24% of variance of the change in sensory bias index. Table 6.4 summarises the regression model.

Table 6.3 presents the regression model used to predict change score in attentional biases to sensory words from time 2-time 3. Predictor variables are change scores during the treatment period.

<table>
<thead>
<tr>
<th>Model</th>
<th>Adjusted R²</th>
<th>df</th>
<th>F Change</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory bias</td>
<td>.294</td>
<td>2, 26</td>
<td>4.118</td>
<td>0.01</td>
</tr>
<tr>
<td>Predictors</td>
<td>Standardised Coefficient Beta</td>
<td>T score</td>
<td>Significance</td>
<td></td>
</tr>
<tr>
<td>Catastrophising</td>
<td>.158</td>
<td>.916</td>
<td>.368</td>
<td></td>
</tr>
<tr>
<td>Fear of movement</td>
<td>.565</td>
<td>3.184</td>
<td>.004</td>
<td></td>
</tr>
<tr>
<td>Fear of pain</td>
<td>.111</td>
<td>.665</td>
<td>.512</td>
<td></td>
</tr>
<tr>
<td>Anxiety sensitivity</td>
<td>-.198</td>
<td>-1.147</td>
<td>.262</td>
<td></td>
</tr>
</tbody>
</table>
6.4 Discussion

We hypothesized that participation in a CBT pain management program would result in a reduction of selective attention towards sensory pain stimuli. The results confirmed the presence of cognitive biases towards sensory pain words in chronic pain patients prior to treatment. As hypothesized, the results also indicated that these biases are modified following CBT. Importantly, changes in fear of movement during treatment predicted subsequent changes in attentional processing. These results have important theoretical and clinical implications.

Most chronic pain models suggest that fear is important in the development and maintenance of chronic pain through promoting hypervigilance towards painful stimuli (e.g. Lethem et al., 1983, Vlaeyen et al., 1995a). However, the nature of fear differs between models. For example, Lethem et al. (1983) describe fear of pain as the central vulnerability, whereas Vlaeyen et al. (1995a) emphasize fear of (re)injury and movement while Asmundsen et al (1999) favour anxiety sensitivity. The present study included self-report measures that related to each of these three concepts allowing the subtle differences between predictions from these theories to be tested.

In addition to the theoretical variables, described above, we included additional outcome variables that we thought might be associated with attentional bias. The only three variables that were associated with subsequent changes in attentional...
Bias were gender and treatment-related changes in catastrophizing and fear of movement. However, only changes in fear of movement independently predicted changes in selective attention during follow-up, accounting for 24% of the variance. Changes in attentional biases where evident at follow up time, which may suggest that patients need to practice the strategies they have learned during the pain management program. Since CBT program is an extensive course which demands a lot of time and effort, perhaps by the termination of the course, patients had more time to put the strategies in practice in touch with the real life situations. This practice may contribute to desensitisation to pain-related information which did happen by follow up time.

The failure of other variables to predict change deserves comment. For example, Taylor (1996) has suggested that anxiety sensitivity is a trait, and hence may not be amenable to therapeutic change. Therefore, the failure of changes in anxiety sensitivity to predict changes in attentional bias may reflect the trait-like nature of anxiety sensitivity, rather than its lack of theoretical importance. Such an argument would be supported by the lack of statistically significant change in anxiety sensitivity over treatment. Nonetheless, the lack of relationship between anxiety sensitivity and attentional biases is consistent with other research. Anxiety sensitivity was not associated with selective attention towards pain-related words in a sample of College students (Keogh et al., 2001a) or a group of chronic pain patients (see chapter 4).

Keogh et al. (2001a) did find a linear relationship between fear of pain and attentional bias. In their sample of undergraduate students, those low in fear of
pain avoided pain-related stimuli, while those high in fear of pain attended to
pain-related words. It is possible that in a healthy population, relationships differ
from those in a chronic pain patient population. For example, in chronic pain
patients (chapter 4) it was found that the effect of fear of pain was the opposite to
that predicted. That is, highly fearful chronic pain patients responded more slowly
to sensory pain words, which the authors interpreted as evidence of inability to
disengage from pain-related stimuli. Therefore, reductions in fear of pain would
not necessarily result in less attention towards sensory pain words. Further, since
fear of pain failed to change significantly over treatment, any potential predictive
ability would have been restricted due to floor effects in change scores.

The finding that changes in fear of movement predicted subsequent changes in
selective attention towards sensory pain words is nonetheless consistent with the
fear of (re)injury model of chronic pain. As such, these results provide direct
support for the fear of (re)injury model of chronic pain (Vlaeyen et al., 1995a).
That is, fear of movement and (re)injury promotes hypervigilance towards pain-
related stimuli and reducing fear of movement reduces this hypervigilance. Fear
of movement scale (TSK) contains items referring to daily activities/feelings and
the perception of patients about these activities or feelings which determine to
what extent patient is likely to confront his/her pain. As such, the main target of
every pain management program is to modify the perception of patients and
desensitise them to pain. As a result, this desensitisation also appears to generalise
over time to the cognitive level and pain-related information becomes less salient
to patients, presumably because it is no longer so threatening to patients.
This finding has potentially important clinical implications. If selective attention predicts function in chronic pain patients, as has been shown (e.g., Pincus & Newman, 2001), then changing attentional biases should be a target of intervention. Our results suggest that targeting patients’ fear of movement in treatment should reduce hypervigilance to pain. The present results suggest that when patients are able to fear movement less, they become less sensitive to pain-related stimuli. Such desensitisation to movement is likely to contribute to recovery from the vicious cycle of fear and disability. An important implication of this study is that pain management programs should focus on reduction of fear of movement as a primary outcome. Indeed, the attentional biases evident at follow up, were more similar to those described in a healthy control group in chapter 5.

On a cautionary note, despite careful attention to methodological issues, this study does have some limitations. Firstly, while our results demonstrate that changing fear of movement is associated with changes in subsequent attentional bias, our results do not demonstrate that fear of movement has a causal role in the development of chronic pain. However, in a recent prospective study investigating predictors of back pain Picavet et al., et al. (2002) found that catastrophising and fear of movement predicted chronicity in back pain six months later. It was both these variables that were associated with selective attention in the present study, although only fear of movement contributed to the unique variance in multivariate analyses. Future research with those yet to develop chronic pain, including measures of attentional bias may confirm whether hypervigilance is one of the mechanisms through which fear of movement confers vulnerability towards chronic pain.
Secondly, the present study did not include a no-treatment control group and therefore the possibility that observed changes represent a practice effect cannot be excluded. However, if these results were simply the result of practice, all responses should be similarly affected regardless of the words presented. Our results indicate specific changes in reaction times to sensory words and not other pain-related words. In addition, practice effects should be evident between assessment 1 and 2, which they were not, particularly since these assessments were conducted over a shorter time interval. It is also unlikely that the relationships between changes in fear of movement and attentional bias would be observed if a practice effect accounted for changes in selective attention.

Another limitation of this study is the short duration of follow-up. It is unclear whether the observed changes in attentional bias would be maintained over time. The other problem associated with the short follow-up is that we could not determine whether changes in selective attention translate into functional changes. Theories predict that this should be the case, but future research is needed to determine whether these relationships can be confirmed.

Finally, the sample size in the present study is modest. The thirty-five patients completing all three assessments would be sufficient to detect large effect sizes (such as those observed in sensory index) but not moderate to small effect sizes. For example, there appears to be a change in attentional strategy towards affective pain words over treatment, where initially chronic pain patients tend to avoid...
affective pain words (i.e. attend away from) but subsequently are more balanced in their attention towards these words. However, the effect size (ES = 0.20) is not sufficiently large to be statistically significant in this sample size. It may be in larger samples that these differences translate into significant changes. Although the sample size used in this study would be small for treatment outcome research, in terms of the cognitive bias literature the sample size is relatively large. With the exception of the study on chapter 4 that included 168 patients with chronic pain, the range of samples in cognitive bias studies has been between 16 (Pearce and Morley, 1989) and 42 (Denton, Sharpe & Schreiber, in press).

Despite these limitations, this is the first study to investigate whether a pain management program focusing on changing maladaptive beliefs and behaviours can result in changes in attentional biases. The study benefits from a high recruitment rate of consecutive patients, suggesting participants in this study are representative of patients attending pain management programs. In addition, a range of outcome variables was administered with a view to testing theoretical relationships between different factors and selective attention (Lethem, et al., 1983; Vlaeyen et al., 1995a, 1999, 2000; Asmundson et al., 1999; Turk and Okifuji, 2002).

In conclusion, the present study confirms the presence of cognitive biases towards sensory pain-related words in chronic pain patients. More importantly, the results suggest that a pain management program can result in changes in cognitive biases after a fairly short period of time. Changes in cognitive processing following treatment were predicted by changes in fear of movement during treatment. These
results support the fear of (re)injury model of chronic pain (Vlaeyen et al., 1995a). Treatments that can change these fears can also result in the correction of biased information processing in chronic pain patients and should be a focus of intervention in pain management programs.
Chapter Seven

General Discussion

In this final chapter the aims and main findings of the preceding studies will be reviewed and discussed within the context of the existing literature. In addition, a number of other issues will be discussed, including methodological problems, strengths and limitations. Finally, the potential clinical implications of the present findings will be discussed.

7.1 General Scope

The present series of studies were conducted to examine and extend the previous research, which had focused on the role that anxiety-related constructs play in the maintenance of chronic pain as postulated by the contemporary models of chronic pain. Study one examined the core concepts of these models using Structural Equation Modelling to clarify the importance of these concepts and their interaction.

In the 2nd and 3rd studies the role of hypervigilance as measured by selective attention to pain-related stimuli in chronic pain patients was examined. Chronic pain patients were compared to a matched sample of healthy pain-free volunteers in order to investigate attentional biases to pain-related information in both groups. In study four, we investigated the effectiveness of a cognitive behavioural-based pain management program, in modifying patterns of selective attention in chronic pain patients. The main findings of the four studies will be reviewed in turn.
7.2 Limitations

Prior to a full discussion of the findings, there are a number of limitations that must be considered when interpreting the results.

7.2.1 Cross-sectional nature

Although we used Structural Equation Modelling to predict the relationships between variables, the directions of relationships imposed into the model are based on theoretical assumptions. That is, whether the direction of causality proposed by these models is correct or the relationships are purely correlational remains untested. To be able to determine causality, there is a need for longitudinal studies with adequate samples both from acute pain patients and chronic pain populations.

Similarly, the attentional biases evident in chronic pain patients were evident in a group of patients who already had developed chronic pain. Whether biases are a cause or consequence of chronic pain is unknown. The fact that healthy control subjects also exhibited similar biases, may suggest that biases may precede chronic pain. However, the fact that these biases change with intervention suggests that they may have a role at least in maintenance of chronic pain. Only, studies designed to assess the biases of patients with acute pain and follow them prospectively will conclusively determine the direction of causality.
7.2.2 Long term follow up

Although chapter six reported a non cross-sectional study, and found that change in specific fear of pain/movement predicted change in attentional biases at follow up, this study is limited for at least two reasons. Firstly, it was not possible to follow the patients for more than one month. The nature of the study required the presence of the patient at the hospital to complete the dot probe task. Since a majority of patients participating in the CBT program were funded by workers’ compensation or insurance companies, and their policy does not cover follow-ups later than one month after termination of treatment, patients were not able to attend for further follow up sessions. In addition, most of the patients lived in regional areas, and were not in the position to afford voluntary attendance at further assessment due to their physical, financial, and time constraints.

Even if we could conclusively determine that attentional biases changed over time and that these changes resulted in subsequent functional changes, we could not determine that these biases caused chronic pain. It could be than concluded that attentional biases were important in the maintenance of chronic pain. Further, if this proved to be the case we could assert that these changes produced functional improvements. However, studies following patients with acute pain would be required to determine whether these cognitive biases play etiological role in chronic pain, as theories would suggest.

7.2.3 Sample size

Although in chapter two and four a large sample was recruited, relatively few controls were available for chapter five. Largely as a result of lack of power, no significant differences between controls and patients were found. While the
patterns of attentional biases where similar in both experimental groups, analyses of effect sizes suggest that differences may exist but these are only moderate. Hence, in the present results, one cannot conclude that differences between the groups exist. Similarly, in chapter seven although the sample size is large in terms of experimental designs of this nature, the sample size remains modest. To investigate treatment effects, larger samples are needed. In fact, the heterogeneous nature of chronic pain population demands larger samples than similar studies with homogeneous populations due to the possible differences in chronic pain patients in terms of pain site or the type of onset of pain. In addition, clinical samples for the studies reported in chapters 4, 5, and 6 were actually part of the large sample reported in chapter 2. Since the samples were drawn from the same pool, it is possible that the consistency across the experimental studies is due to the over-lapping participants, rather than the robustness of the effect.

7.2.4 CBT intervention

In chapter six, the modifying effects of cognitive behavioural therapy on attentional biases were examined. However, the lack of a control group limits the conclusions that can be drawn. Specifically, a control chronic pain patient group without CBT intervention would allow us to determine whether practice effects could account for the findings.

Another difficulty with chapter six is the multi-component nature of cognitive behaviour therapy. Pain management programs are a combination of different disciplines, with different components from each. As a result, programs are a very complex combination of interacting components, which makes it difficult (if not impossible) to determine which factor(s) are responsible for any given change. Therefore, although in chapter six it was found that changes in fear of movement might predict the changes in attentional bias, how these changes are best
facilitated remains speculative. Further, whether these changes translate into functional benefits remains unclear. Whether modification of attentional biases following CBT may have improved physical performance in the patients was not examined in the present series of studies.

7.2.5 Validity of Dot Probe Task

Although the dot probe paradigm has been applied to research on anxiety disorders extensively, so far no there has been no study which has been published on its validity. It is not clear also whether results of dot probe task are really reflecting the proposed theoretical concept (for example, hypervigilance to mood congruent information) because of (at the best) moderate correlations with the results of other measures. Different modifications have been made to the original program. For example, interval time between presentation of words and probe, and also the allowed time for subjects to endorse the response (e.g. 1500ms to 3000ms) is different across studies. These modifications may result in different findings (Mogg et al., 2000). In addition, data (reaction times) has been treated in different ways. Dealing with outlier data is one of the most uncertain areas (Ratcliff, 1993). Selecting a cut-off point for outliers seems especially difficult when different experimental groups are involved. It is possible that applying the same cut-off point to both controls and patients due to their different reaction time distributions may increase type II error. In addition, dot probe results are poorly correlated with other self-report measures (Mogg et al., 2000; Keogh et al., 2001a, 2001b). Perhaps of more concern, evidence suggests that the results of dot-probe are not strongly related even with other measures of cognitive bias (e.g. Stroop test) in the same sample (Mogg et al., 2000). Such discrepancies have led to the
conclusion that these measures are tapping different constructs (Mogg et al., 2000).

However, it seems that this is not a problem associated only with measures of cognitive performance. For example, it is well known that psychophysiological measures (e.g. HR, SCL) are poorly correlated with self-report measures (Turpin, 1989). Similarly, even different measures of psychophysiological responding, such as HR and SCL are often discordant (e.g. Sharpe et al., 1995). Therefore, this problem is not specific to the dot-probe task. Nevertheless, these complexities complicate our understanding of the relationships between different theoretical constructs and compound concerns about reliability and validity of the results.

7.3 Strengths

Despite all the above-mentioned limitations, the reported studies in this thesis benefit from a number of strengths. In chapter two and four we recruited a large consecutive cohort of chronic pain patients referred to a tertiary pain management centre. The recruitment is excellent and in terms of patients’ characteristics, they are similar to previous reported samples by other studies at the same setting (Sharp and Nicholas, 2000; Asghari and Nicholas, 2001) and also other countries (e.g. Turner et al., 2000; McCracken, Gross, and Eccleston, 2002). Similar demographic characteristics between the chronic pain patients who participated in this study and those who are reported previously, add to the level of generaliseability of the results. In addition, speaking about experimental studies, including a large sample in chapter four as an experimental study, remains a main advantage of the study.
7.4 Psychological models of chronic pain

In chapter 2, two models of the chronic pain were tested. The results supported both models. According to Vlaeyen’s Model, the vicious cycle of the experience of pain and subsequent disability, with the central role of fear of pain/(re)injury, was examined. That is, in the presence of pain, it is postulated that the level of fear of pain and movement leads to subsequent disability. As described before, it is also critical in the model that individual vulnerabilities such as negative affectivity impact on the individual’s pain-fearfulness. Hence, predispositional factors might directly facilitate developing a fear of pain and, indirectly, contribute to disability. Therefore, in chapter two, this theorised vicious cycle with the proposed factors in that model were simultaneously tested. The model fit the data with few modifications. Results indicated that fear/avoidance of pain directly contributes to disability and accounts for 42% of the variance in disability. In turn, fear/avoidance itself was predicted significantly by negative affectivity, indicating the role of vulnerability factors. These results are consistent with the fear of pain/(re)injury of pain as described by Vlaeyen et al. (1995a).

In model two, we examined the diathesis-stress model of Turk (2002), which is very similar to Vlaeyen’s model except that Turk (2002) introduces the concept of self-efficacy and does not include negative affectivity. According to the diathesis-stress model, self-efficacy impacts the experience of pain despite possible susceptibility or fear of pain, and as a result impacts the perceived disability. That is, people who are confident that they can do things despite the pain are more likely to remain active even at the presence of vulnerability factors, and may show
less avoidance behaviour. The tested model approved self-efficacy as partially mediating between fear/avoidance factor and disability. According to the results, while similar to Vlaeyen model, catastrophising and fear of pain/(re)injury may increase the chance that a given patient will avoid activities, however, in addition self-efficacy may mediate the effects of fear/avoidance on disability. Similar to model 1, Turk’s model explained the same proportion of the variance of disability (42%), perhaps in a more parsimonious way.

Although both models received support and were found fit the data, Turk’s model was favoured in terms of statistical purity and parsimony. However, both models support the idea that fear of pain/movement contributes to the maintenance of pain and disability. While these models describe the relationships between variables in each model, the underlying mechanisms by which fear of pain increases pain and disability remain unclear.

Theoretically, when people fear something (i.e. pain), it has been argued that they tend to be sensitive to its signals in order to avoid the threat. This sensitivity is described as hypervigilance. According to anxiety research, fearfulness leads to hypervigilance to the source of threat, which in turn, triggers avoidance behaviour. Generalising the same paradigm to chronic pain, contemporary models also believe that fear of pain is followed by hypervigilance and consequently avoidance of movement occurs, which results in physical disability. This hypervigilance results in attention towards pain at the expense of other competitive information. Hence, it seems that hypervigilance might be the mechanism proposed by contemporary theories, which translate fear into avoidance and disability. It was the purpose of chapters four to six to investigate these phenomena in chronic pain patients.
7.5 Attentional biases in chronic pain patients

7.5.1 Study Two (chapter 4)

To date, the literature is conflicting with regard to the results of the studies on attentional biases. Two separate reviews (Pincus and Morley, 2001; Roelofs et al., 2002) have suggested that despite confounding results, there is evidence that chronic pain patients show attentional bias towards sensory and affective stimuli. In chapter four, we found that chronic pain patients are biased in attending to sensory pain-related words relative to other word types including affective, disability, and threat. These results were highly significant and robust, suggesting that selective attention to at least the sensory dimension of pain is present in chronic pain patients. However, this study did not address the issue of whether such biases are specific to chronic pain patients.

One area of concern in the literature is whether all chronic pain patients demonstrate equivalent attentional biases or whether only those high in anxiety, fear of pain, or anxiety sensitivity process pain-related information preferentially. The results of the study reported in chapter four, found a bias in processing in a heterogeneous group of chronic pain patients that did not appear to be a function of anxiety, depression, fear of pain, or anxiety sensitivity. Indeed, fear of pain appeared to have the reverse effect on information processing to that predicted. That is, chronic pain patients with higher levels of fear of pain were slower in responding to pain-related stimuli. It was argued that this may reflect a tendency
to have difficulties in disengaging from pain-related stimuli amongst chronic pain patients high in fear of pain. However, interpretation of this finding is complicated by the fact that chronic pain patients actually had lower fear of pain scores than those reported in healthy samples (e.g. Keogh et al., 2001a).

7.5.2 Study Three

Problems in the interpretation of the attentional bias results reported in chapter four, were compounded by the results reported in chapter five. The results of the latter study revealed that attentional biases to pain-related words, as tested by the dot probe task, are not specific to chronic pain patients. Although the results were in the predicted direction, the difference between chronic pain patients and controls was not significant, and was not sufficiently robust to be considered a trend when the responses of chronic pain patients were compared to healthy controls. The effect size for the attentional bias to sensory words was 0.26, as is consistent with the existing literature. Importantly, this is the first study to demonstrate the same effect size as derived from Stroop studies is also found using dot-probe in chronic pain patients. This strongly suggests that the biases reported in the literature are truly attentional biases and not due to response bias artefact. Interestingly, the pattern of attentional bias to different pain-words found in chronic pain patients was also evident amongst healthy control subjects.

Summarising the results of the studies reported in chapter 4 and 5, the following can be concluded:

1) Chronic pain patients do selectively attend to sensory pain-related material.
2) However, similar patterns of attentional biases are also present in healthy controls, although to a lesser degree.

As previous studies on attentional biases in healthy populations suggest (Keogh et al. 2001a; Keogh et al., 2003), our results confirm that at least a proportion of pain-free healthy population show similar attentional biases to pain-related information. This raises the question then of whether the differences between healthy controls and chronic pain patients are of critical significance, and therefore, worthy of empirical investigation. Pincus and Newman (2001) showed that such biases in chronic pain patients predict further health care utilisation and cost. If this is the case, then it is difficult to argue that attentional biases do not have importance since they predict objective measures of function. How then do we explain the lack of large and robust differences between chronic pain patients and healthy control subjects? There might be two possibilities. First, while attentional bias to pain-related information might have an adaptive function in healthy people as part of a survival response to pain, the same cognitive processing bias may be unhelpful for people in chronic pain. Although there is no study on attentional biases to pain-related information in healthy population to investigate whether those who show attentional bias may show a different health care seeking profile, Lethem et al., (1983) reported that healthy university students who reported higher levels of fear of pain had a different health care profile relative to those who did report lower levels of fear of pain. Second, it is not clear whether these attentional biases are also unhelpful for acute pain patients, considering the argument that these biases result in over attending to pain and facilitate sensitisation to pain. Conducting similar studies to Pincus and Newman (2001) on healthy people as well as pain populations with both chronic
and acute pain might be helpful in determining the importance of attentional biases in contributing to chronicity of pain.

Clearly, further research is required to determine which, if any, of these interpretations are correct. Nonetheless, the findings of attentional biases that predict function in chronic pain samples (Pincus and Newman, 2001), suggest that improving these biases should improve function. Hence, these attentional biases are an appropriate target of intervention.

7.5.3 Study four

The treatment of choice for chronic musculoskeletal pain is cognitive behaviour therapy (CBT) (Morley et al., 1999). One of the primary aims of cognitive behavioural pain management programs is to change the way that patients approach pain and activity. CBT for chronic pain is based on the assumption that patients’ thoughts about their pain alter their behaviour and subsequent physiological and emotional responses to pain. Accordingly, it seems logical to expect CBT intervention to alter cognitive biases as one of the primary functions of treatment.

In chapter six, we examined the effectiveness of a CBT-based pain management program in changing observed attentional biases in chronic pain patients. The results confirmed that selective attention does reduce following cognitive behavioural treatment to the levels of selective attention demonstrated in healthy control samples. Specifically, these programs are based on the models of chronic pain that were described and validated in chapter two. That is, patients who fear pain or (re)injury are more likely to avoid pain-provoking activities. As a result, they become increasingly disabled, experience physiological deconditioning and
become depressed and anxious about their deteriorating condition. This leads them to believe that they are unable to continue with daily activities (i.e. decreased self-efficacy), creating a vicious cycle. Hence CBT programs aim to help patients confront activity and challenging their fear of movement/(re)injury, gradually increase their confidence that they can re-engage in daily activities. If programs are successful in challenging the content of patients’ fears, and patients’ fears and beliefs cause patients to be hypervigilant to pain cues, then one would expect selective attention to change following successful intervention. Indeed, that is what the present results confirmed.

In fact, following a CBT intervention, it was found that changes in specific fear of movement during treatment, could predict the change in the pattern of attentional biases during follow up. These results are consistent with Vlaeyen et al.’s (1995a) model that suggest you can reduce fear of pain/(re)injury, then cognitive biases will shift. Therefore, one would want to focus on CBT strategies whose primary aim is to shift fear of (re)injury. Whether this is best achieved through cognitive challenging or simply approaching activity, however, is unclear. It may be that both are necessary to produce robust change in fears. Future research should investigate the effects of specific treatment strategies on attentional processes, and is needed to confirm that these interventions that change cognitive processing result longer term benefits with regard to function.

7.6 Theoretical implications

One potential inconsistency in the present program of research is that while in chapter two, self-efficacy showed considerable promise in its strong relationship with disability, it was unrelated to selective attention in chapter 4 and 6. While
fear-related constructs were found to be more strongly associated with selective attention, it seems unlikely that the mechanism through which self-efficacy confers vulnerability to disability is attentional bias. Vlaeyen et al.’s model also received good support in chapter two, and fear-related constructs were more consistently related to cognitive biases than self-efficacy. However, the relationships were not straightforward. For example, neither fear of pain nor anxiety sensitivity had the predicted relationships with cognitive processing biases in chapter four. Indeed, the results indicated that fear of pain increased response latencies. If this is evidence of disengagement then it is not inconsistent with Vlaeyen’s theory. However, results of chapter 5 do not support the argument that these findings represent disengagement as fear of pain may significantly associated with attentional bias. Only the results of chapter six provided clear and unequivocal support for the relationship in this instance between changes in fear of movement/(re)injury (TSK) and subsequent changes in attentional bias. However, in study two as reported in chapter 4, fear of movement/(re)injury (TSK) was unrelated to indices of attentional bias in cross-sectional analyses.

Despite the inconsistencies in relationships between specific fear-related concepts, it is clear that fear-related variables were more commonly associated with cognitive bias than other non-fear concepts, such as self-efficacy. Nonetheless, self-efficacy when included in the modelling described in chapter 2, provided a more parsimonious account of disability. Hence, further research should attempt to explore the mechanism by which self-efficacy is associated with increased disability. Understanding these relationships might help us to better understand where self-efficacy and negative affectivity might interact to better explain pain-
related disability, since modelling attempts to include the two in the one model failed.

Similarly, future research should consider the various fear-related constructs in different homogenous samples (e.g. chronic pain, acute pain, and healthy controls) to determine their relative contributions. It may be, for example, that negative affectivity is more strongly predictive of disability in acute samples (i.e. causes transition from acute to chronic pain), whereas self-efficacy is a stronger predictor in chronic samples (i.e. important in the maintenance of pain). This study has provided a preliminary investigation of cognitive biases in chronic pain patients testing contemporary psychological models of chronic pain. However, many questions have been raised which require research to evaluate.

**7.7 Directions for further research**

Findings of the reported studies support fear of pain as a critical concept in the maintenance of pain-related disability. Self-efficacy was suggested to play a partial mediating role between fear/avoidance beliefs and disability. We also found that chronic pain patients were biased in their attention to sensory pain-related words relative to other presented word types. However, these effects are not specific to a chronic pain population. In fact, healthy people without pain exhibited similar patterns. This finding is consistent with the evolutionary characteristic of pain. In addition, it was found that maladaptive cognitive biases in chronic pain patients did change following a cognitive behavioural pain management program. However, these results are in their infancy, and different questions remain unaddressed and further research needs to investigate them.
Further research needs to evaluate the relationship between self-efficacy and negative affectivity in the development of chronic pain. To investigate this relationship, perhaps one of the most appropriate ways is to conduct a longitudinal study with a large sample size of acute pain patients, in order to study the complex interactions of a variety of variables theoretically involved in the development of chronic pain. Including a measure of selective attention into the study will clarify the role of selective attention in transition from acute pain to chronic pain.

The results of the studies in chapters 4-6 raised the question of whether fear-anxiety-related constructs have a relationship with selective attention. One of the unexpected findings in this research was that there was no significant difference in the level of fear of pain between healthy controls and chronic pain patients. Considering the important role attributed to fear of pain in chronicity, this finding is very important. Future research needs to compare acute pain patients with chronic pain patients and healthy controls to investigate more accurately the phenomenon. If acute pain patients have different levels of fear of pain in comparison with chronic pain patients and controls, it may imply that chronic pain patients habituate over time to their pain. Accordingly, as pain becomes chronic, it might be possible for hypervigilance to be replaced with insensitivity to pain-related information. Conducting prospective long-term studies including acute pain patients will provide valuable information about changes in the pattern of sensitivity to pain cues and the level of fear of pain across the time. As Pincus and Morley (2002) have concluded, “the most gaping hole in the evidence is the lack of prospective studies” (p. 138). Such a longitudinal design will shed more light on the nature of possible vulnerability factors such as negative affectivity, and also provide a more reliable basis for causal inferences. Similarly, future
prospective studies are needed to investigate to what extent cognitive biases to processing information related to pain might be responsible for maintenance of pain. Again, since similar attentional biases were found in healthy controls, the only way to investigate the nature of these biases is to compare acute, chronic, and healthy controls in a single study.

While we provided a preliminary support for the effectiveness of cognitive behaviour therapy in modification of attentional biases, it was not investigated whether these changes are associated with changes in function including physical performance or, as Pincus and Newman (2001) suggested, health care costs. In addition, stability of the observed changes in attentional biases has to be followed up to prove their value as a treatment target. Finally, even if the association of changes in attentional biases is evident, it is unclear what aspects of cognitive behaviour therapy are most effective in reducing selective attention.

While all these possible new avenues of research will certainly add to our knowledge to provide a better explanation of the complex relationship between anxiety and fear-related constructs and selective attention to pain-related information, as we reviewed, methodological constraints might be an important issue accounting for high variability in attentional bias studies.

Further research needs to move towards a unification in methodology. Speaking specifically about measuring selective attention using the Dot Probe Task, various types of presentation times, time courses, response times, word types, and outlier cut-off points have been applied. It seems that if future research be conducted according to a consistent methodological protocol, results and findings across
different studies would be more comparable and productive. At the present time, we do not know to what extent the different results might be a product of methodological differences. To conduct the selective attention studies in this volume, we developed a computerised dot probe program, which might be one suggested basis for such a protocol. This program is designed in a way to suit further research with sufficient flexibility to comply with different research requirements, in terms of managing different time courses in one study, presentation time intervals, word categories, and has shown sensitivity to relevant stimuli and change.

7.8 Conclusion

The results of the reviewed studies show that in the complex problem of chronic pain, psychological factors play a critical role. Once pain is chronic, neurophysiological explanations of pain are less efficient. Psychological approaches to chronic pain have developed over the last two decades with growing emphasis on the role of fear and anxiety-related constructs as a central concept. These advances have resulted in establishment of non-biomedical explanation of pain and acceptance of pain as a biopsychosocial problem. It is within these recent theoretical models that the current empirical work was situated. The aim of the present thesis was to test the current theoretical models of chronic pain. Specifically, chapter 2 attempted to test two competing models of chronic pain. Chapter 4 attempted to investigate the presence and nature of cognitive biases in chronic pain patients. Chapter 5 aimed to determine whether these biases were specific to chronic pain patients or also occurred in a well-matched healthy control group. Finally, chapter 6 aimed to see whether cognitive
biases could be modified in chronic pain patients through cognitive behaviour therapy.

The following conclusions can be drawn from the studies reported in this dissertation:

1) Consistent with the literature, fear of pain-related constructs play a significant role in maintenance of disability. This role, however, was shown to be partially mediated by self-efficacy. Both Vlaeyen et al.’s (1995) fear of (re)injury model and Turk’s (2002) diathesis-stress model were largely supported by the data.

2) Chronic pain patients demonstrated specific attentional biases to sensory pain-related words relative to other word types including affective, disability, and threat.

3) This pattern of selective attention to sensory words is not specific to chronic pain patients, but evident also in healthy control subjects, although to a lesser degree.

4) We found cognitive behaviour therapy effective in modification of these attentional biases. Preliminary support was provided that changes in fear of movement/(re)injury were associated with modification of selective attention.

In summary, this thesis in its entirety has provided strong support for the prevailing theories of chronic pain, namely Vlaeyen’s fear of (re)injury model and Turk’s (2002) diathesis-stress model. The thesis has also concluded that selective attention is important in chronic pain, although the precise nature of the
relationships between chronic pain and selective attention and other variables remain to be elucidated.

8 References


completion of a cognitive-behavioral pain management program by initial


Blyth, F.M., March, M.M., Brnabic, A.J.M., Jorm, L.R., Williamson, M., Cousins, M.J.


threatening facial expressions in anxiety: manipulation of stimulus duration.
Cognition and Emotions; 12: 737-753.

Bradley BP., Mogg K., White J., Groom C., de Bono J. (1999). Attentional bias for
267-78

the Depression Anxiety Stress Scales (DASS) in clinical samples. Behav Res
Ther;35:79-89.

Anxiety Symptoms Scale(PASS): Prediction of physical capacity variables Pain
84(2-3) 247-252


Fordyce W.E., Behavioral Methods For Chronic Pain and Illness, Mosby, St. Louis, 1976.


International Association for the study of pain, Subcommittee on Taxonomy pain terms. A list with definitions and notes on usage.(1979). Pain. 6, 249-252


Congress on Pain (pp.877-884). Seattle. WA: International Association for the Study of pain.


McGill Pain Questionnaire. Pain, 39: 115-121

Pengel LH, Herbert RD, Maher CG, Refshauge KM. (2003). Acute low back pain:
 systemat ic review of its prognosis. BMJ. 9; 327 (7410): 323. Review.

Diagnostic Systems, Worthington, OH.


Picavet HS, Vlaeyen JW, Schouten JS. (2002). Pain catastrophizing and kinesiophobia:


Pincus T, Morley S. (2001). Cognitive-processing bias in chronic pain: a review and

Pincus T, Morley S. Cognitive appraisal. (2002). In S.J. Linton (Ed.). New avenues for the
prevention of chronic musculoskeletal pain and disability; Pain Research and
Clinical Management,. Vol. 12, 123-141. Elsevier Science B.V.


behavioural perspective. New York: Guilford Press.


Turk (Eds.). Psychological approaches to pain management: A practitioners’
handbook (pp. 3-32). New York: Guilford Press.

traumatic injury; Pain Res. Manage. Vol 7 No.1: 9-19

Press.

Turk DC., Okifuji A. (2002). Psychological factors in chronic pain: evolution and

independently predict functioning in patients with chronic pain? Pain 85, 115-
125


Van Damme S., Crombez G., and Eccleston C. (2002). Retarded disengagement from pain

musculoskeletal pain. In: S.J. Linton (Ed); New Avenues for the Prevention of
Chronic Musculoskeletal Pain and Disability. Pain research and clinical management Volume 12: 83-103; Elsevier Science B.V.


9 Appendices