CHRONIC BACK PAIN AND DEPRESSION: A COGNITIVE-BEHAVIOURAL APPROACH

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ABSTRACT

The aim of this thesis was to investigate the relationship between chronic pain and depression and examine the role of possible mediating variables within a cognitive-behavioural framework. The latter is the focus of this research, although it is acknowledged that it is a subsection of a broader biopsychosocial perspective. Given the number of complex variables involved, and the relative lack of literature examining their interactions, this work must be considered to be an exploratory project. The participants were people with chronic back pain who had not been involved in clinical treatment and results from both cross-sectional and longitudinal studies were reported.

In Study 1 (N=30), pain severity and depression were not significantly correlated, but were mediated by cognitive-behavioural variables, perceived interference and pain self-efficacy. In Study 2, (N=105: 41M; 64F), 34% of the participants reported clinical levels of depression. Those participants who were depressed reported significantly more pain severity, interference and state anger and significantly less control, pain self-efficacy and general self-efficacy, than those who were not. Pain duration was not significantly related to increased risk of depression, as had been predicted. The chronic pain participants scored more highly than 66 matched controls on depression, but they did not differ significantly on other relevant variables. In contrast to Study 1, pain severity was significantly and positively correlated to depression and also mediated by interference, pain self-efficacy and state anger, mostly via the latter. It was found that response to chronic pain differed between males and females. For the males, there was not a significant relationship between pain severity and depression and most of the influence on depression occurred via mediating variables, perceived interference and general self-efficacy. In contrast, for females, depression was more likely to be a direct result of pain severity.

Study 3, a 5-year longitudinal follow-up, involved 44 of the original 105 respondents (16M; 28F). Analyses showed that, in contrast to Time 1, depression at Time 2 was more related to how much control the responders perceived they had over their lives indirectly as a result of chronic pain, with little direct effect from pain severity. Gender analyses found that for both males and females, pain severity and depression were not significantly related but mediated by pain self-efficacy and life control, respectively. Study 3 failed to provide

support for the hypothesis that chronic pain precedes depression as analyses showed that depression predicted pain severity over time slightly better than the reverse.

Although results need to be viewed with caution, they support the theory that depression is a significant correlate of chronic pain and that the relationship may be complicated by mediating factors and by gender differences. Results failed to provide clear evidence that chronic pain consistently precedes depression. Findings were consistent with a cognitive-behavioural approach to the study of chronic pain, supporting the premise that chronic pain is fundamentally a sensory, cognitive, behavioural and affective experience.

STATEMENT

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

I give consent to this copy of my thesis, when deposited in the University Library, being available for loan and photocopying.

Signed _____ Date 15/12/09

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Dedicated to my Mum
for her unconditional love and support:
Muriel Edith Johns, 1912 - 1997

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CHAPTER ONE

INTRODUCTION

> "It is not a pleasant trait, but we sometimes feel suspicious of people who say they are in pain but who do not groan, or writhe, or pound on the floor. Pain patients know what it means to face daily suspicion. [....] pain might issue from a hundred different hiding places, like the smoke from a smouldering ruin. Even if the process of medical unmasking might finally discover the last pain hidden beneath overlapping [...] strata, doctors might be unable to [...] relieve the suffering. [...] Chronic pain [...] had made [...] her life a permanent daily torment". (Morris, 1992, p. 6-7).

1.1 Chronic pain: A uniquely debilitating medical condition

This statement is a harsh commentary on the debilitating nature of chronic pain. It reflects the anguish and agony experienced by many people with chronic pain, unable to find relief. Understanding chronic pain is one of the most challenging of all health problems, not only to the person with chronic pain but also to the health professions and to society (Melzack & Wall, 1988). Pain has been researched within many health disciplines. Originally, research was conducted primarily within the boundaries of the medical profession. However, in more recent years, it has also been incorporated into the discipline of psychology (Weisenberg, 1987).

Psychology has contributed to the study of pain through research into the emotional and cognitive reactions to pain. These are important psychological processes, fundamental to the experience of pain and are part of a broad range of psychological factors which act as determinants of pain perception and behaviour. Psychological factors were once only thought to be correlates of pain but increasingly it is thought that they may be involved in all

stages of the pain experience including causes, maintenance and consequences (Linton & Skevington, 1999). Continued research into such factors is important to increase our understanding of pain and how it may be prevented, treated and managed. This thesis reports research undertaken from the psychological perspective, therefore relevant material from other disciplines is only considered as it pertains to a psychological approach. The size and complexity of the available research on the general construct of 'pain' requires a guide as to what is directly relevant to the research contained here and what does not bear on it. As a further aid to clarity, the overall objectives of the research are also set out in this chapter. Specific research aims and hypotheses will be presented in later chapters.

"The regulation of pain and suffering has been a preoccupation since the beginning of time" (Turk & Holzman, 1986b, p. 257). Chronic pain has been and still is one of the most debilitating medical conditions known to humans, affecting social, economic and psychological well-being (Elliott, Smith, Penny, Smith, & Chambers, 1999; Loeser, 1980). It continues to be one of the most common reasons for seeking medical care and estimates of annual costs to health care systems, the world over, exceed billions (Verma & Gallagher, 2000). For example, nearly a half a million work days are lost in America every year as a result of chronic pain with more than US$150B spent in related health care (Arnstein, Caudill, Mandle, Norris, & Beasley, 1999).

Chronic back pain, in particular, is one of the most insidious types of chronic pain (Pearce & McDonald, 1998) and considered to be the most demanding of medical problems, in terms of health care service utilization (Engel, Von Korff, & Katon, 1996). It has been estimated that about 80% of people are affected by back pain at some point in their lives (Flor & Turk, 1984; Mayer, Gatchel, Kishino, Keeley, Mayer, Capra, & Mooney, 1986). In addition, back and spine impairments are the major cause of morbidity and disability in the 18-44 year age group, for both males and females (Waddell, 1982).

Chronic back pain is likened to a silent 'epidemic', with victims suffering a wide range of physical and mental problems (Loeser, 1980). They often contend with restricted movements and reduced activity while the psychological and emotional upheaval, in

particular, exacerbate the suffering (Chapman, 1995; Fishbain, Cutler, Rosomoff, & Rosomoff, 1997; Gottlieb, Strite, Koller, Madnsky, Hocksmith, Kleeman, & Wagner, 1977; Kerns, Rosenberg, & Jacob, 1994). Unremitting pain may eventually consume one's life, contributing to a loss of control and confidence and adversely affecting social and sexual relationships. Subsequently, many become unemployed, housebound and socially isolated. Chronic back pain has been referred to as *"an integrated, bio-psychosocial syndrome"* (Katz, 1993, p. 104), composed of interactive sensory, cognitive, behavioural and affective components (Novy, Nelson, Francis, & Turk, 1995).

1.2 The chronic pain literature: "the tower of Babel"

Despite being such a serious health issue, chronic pain has only been researched as a unique topic since about the 1930s (Gamsa, 1994a), even though it has been a puzzling phenomenon for centuries (France, Krishnan, & Houpt, 1988). Regardless of this relatively short history, chronic pain research has generated a prolific literature, which Bonica (1979) referred to as "the tower of Babel" (p. 247). Gamsa (1994b) described the research as *"a field of study defined by multiple interacting variables (we cannot isolate and control the variables: they can't be separated and they won't stand still)"* (p. 25). This complex and complicated literature is widely criticised for its considerable ambiguity, controversial nature and lack of methodological rigour (Flor & Turk, 1984; Gamsa, 1994b).

Confounding this complexity is the considerable controversy over theoretical approaches that have guided research processes (Adams, 1997). Traditionally, medical and psychiatric conceptualisations of pain have taken a uni-dimensional perspective viewing pain as either a sensory-physical phenomenon, a behavioural manifestation or a purely cognitive experience (Melzack & Wall, 1982). However, this view has been criticized as 'restrictive' and incapable of fully explaining incongruous pain experiences. These include cases where there is no discernable injury associated with pain, injury without pain, phantom limb pain, pain that is disproportionate to injury and persistence of pain despite surgical and/or pharmaceutical intervention (Novy et al., 1995). Such an approach is seen to be detrimental

to the person with chronic pain in terms of clear diagnosis and effective therapy (Sullivan, 2000). In contrast, more recent research, particularly psychological, has taken a 'comprehensive' perspective that focuses on the multi-dimensional nature of pain in an effort to determine the most effective treatments (Kerns, Turk, & Rudy, 1985; Turk & Rudy, 1988; Turk & Rudy, 1990). This view recognizes that chronic pain consists of, not just sensory aspects but also cognitive, behavioural and affective elements. In recent years, there has been considerable development of theories that explain chronic pain from a 'comprehensive' perspective, especially theories and models of a cognitive nature.

Moreover, conceptual differences have also produced diverse research methods, a lack of controlled studies and a literature that has become even more complicated. These methodological inadequacies have plagued the literature. For example, in an early review of the literature, Romano and Turner (1985) noted that there were many methodological problems with research, including lack of controlled studies. They concluded that the extent of the relationship between chronic pain and depression was a controversial issue. In a later review, Roy (1986) criticized the pain literature for its lack of consensus on measuring the severity of pain, reporting that research findings lacked consistency on practically all levels. There were similar criticisms when Sullivan, Reesor, Mikhail and Fisher (1992) described the literature as "conceptually fragmented [...] with little cross-study consistencies in theoretical framework, methodology, or findings" (p. 5).

In another comprehensive review, Fishbain et al. (1997) reported that most clinical studies investigated conceptually different areas including the extent of pain, number of pain sites, severity and frequency of pain. Studies also differed in terms of sample sizes, population under investigation (e.g. age, type of pain, i.e. headache, arthritis, cancer etc.), study design, measures used and types of statistical analyses undertaken. For example, sample sizes from the literature covered in this review, ranged from nine female chronic headache patients (Rains & Lohr, 1993), who completed headache activity and mood diaries, to over 5,000 Hispanic people in the United States who were surveyed for depression related to abdominal pain (Magni, Rossi, Rigatti-Luchini, & Merskey, 1992).

Many studies did not use standard questionnaires to measure relevant variables and were not consistent in definition of chronic pain while study designs were mainly cross-sectional with some limited longitudinal analyses. While providing valuable information, such disparity hinders comparisons and generalizations across research studies.

Despite considerable and revolutionary advances in the understanding of chronic pain, Turk and Okifuji (2002) continue to call for more rigorous research to overcome the methodological inadequacies of many research projects. They caution about making generalizations from studies that may rely too heavily on retrospective studies, unrepresentative or inadequate samples of subjects or failure to investigate key variables (Turk & Okifuji, 2002). In addition, they maintain the importance of continued research into the diverse, interrelated aspects of chronic pain to gain a greater understanding of this major social problem. Furthermore, despite the extensive research, the intricate nature of chronic pain and its effects on people with chronic pain continues to be a relative mystery (Melzack, 2001).

1.3 The perpetual puzzles of chronic pain

In particular, one of the more puzzling aspects of this condition and the key issue addressed by this thesis, is that while some people adjust to constant pain and live relatively normal lives, others do not adjust and become severely psychologically impaired (Doleys, Crocker, & Patton, 1982; Jensen, Turner, Romano, & Karoly, 1991; Spence, 1993). Adjusting to chronic pain refers to "adaptive mental functioning" ...[or the] "ability to carry out normal physical and psychosocial activities" (Jensen et al., 1991, p. 250). Moreover, this definition does not necessarily inform about the meaning of 'adjustment' to the person with chronic back pain who experiences pain as a life-long companion. For such a person adjustment generally means learning to live with the condition and getting on with life (Nicholas, 1994). This is considered to be a more realistic proposition than expecting pain to be reduced or eliminated (Flor, Fydrich, & Turk, 1992; Spinhoven & Linssen, 1991). The person with chronic pain who thinks that normal living cannot occur until the pain has gone is

likely to be more psychologically distressed than the person with chronic pain who accepts the pain and adapts to the changes it entails (Nicholas, 1994). Adjustment to chronic pain has been measured in many different ways, including psychological well-being, medication use, employment status, level of activity, utilization of health services and pain severity (Jensen et al., 1991).

In terms of psychological well-being, one of the main ways of assessing adjustment, has been to identify the presence of depression. Besides being a major indicator of poor adjustment or failure to cope with persistent pain, depression is regarded as the most common, maladaptive psychological condition associated with chronic pain (Jensen et al., 1991; Romano, Syrjala, Levy, Turner, Evans, & Keefe, 1988; Turner, J., 1982). In addition, the extent of the relationship between chronic pain and depression is still considered to be inconclusive and unclear (Linton & Skevington, 1999; Verma & Gallagher, 2000). There is considerable debate about most other aspects of this relationship, including definition, measurement, classification and prevalence. A key issue relates to the temporal nature of the relationship, i.e. do chronic pain and depression occur concurrently or does one precede the other, and if so, is the relationship direct or indirect? The search for answers to these puzzling issues continues, as "[u]ntangling the relationship [...] has been tedious and remains incomplete" (Linton & Skevington, 1999, p. 25).

Depression is a serious condition in its own right and health in general is compromised when one is depressed (Angst, Kupfer & Rosenbaum, 1996; Hays, Wells, Sherbourne, Rogers & Spritzer, 1995). The most serious consequence of depression is undoubtedly suicide, and it has long been recognized that depression is associated with suicide completion (Klerman, 1987). For example, in Australia alone about 70% of all suicides committed each year are by depressed individuals (Burrows, 1994). If depression in the person with chronic pain is ignored, recovery and/or adjustment is seriously impeded (Romano & Turner, 1985; Verma & Gallagher, 2000). Coupled with chronic pain, depression therefore represents a considerable life-threatening co-morbid condition (Fishbain et al.,

1997). However, there is still much about this condition that is unclear (Flor & Turk, 1984; Gamsa, 1994b; Melzack 2001).

Knowledge about chronic pain associated with depression has also been limited by a dependence on a restricted and unique group of people with chronic pain. Most people endure significant pain without extensive medical intervention and/or treatment (Linton, 1994; Linton & Skevington, 1999; Von Korff, Dworkin, LeResche, & Kruger, 1988), so research about chronic pain is based on the minority who are referred from general practice physicians to pain clinics or hospital based treatment programs (Jensen et al., 1991; Magni, Moreschi, Rigatti-Luchini, & Merskey, 1994). These people have either been assessed before, during or after medically based treatment. They are likely to be more psychologically disturbed than those people with chronic pain from the wider community who are not referred on for more extensive medical procedures (Keefe, Wilkins, Cook, Crisson, & Muhlbaier, 1986; Merskey, 1980). This small group of people with chronic pain, who use most of the health services, mainly comprise those suffering work-related injuries with attendant litigation issues (Carey, 1994; Carron, DeGood, & Tait, 1985; Linton, 1994). This restricted group is not considered *"representative of the general population"* (Elliott et al., 1999, p. 1248), as they are unlikely to represent the majority of people with chronic back pain in the wider community (Crombie & Davies, 1998; Doleys et al., 1982; Jensen et al., 1991; Smith, Smith, & Chambers, 1996).

Such people with chronic pain are often referred to anecdotally by health workers has 'the hard cases', who are 'beyond hope' and reluctant to 'own' their condition. The lack of efficacy of traditional medical treatment approaches can force such people with chronic pain on to a 'merry-go-round' of medical visits, building up extensive medical files, disability, psychological distress and over-utilization of health care system with inadequate relief (Flor & Turk, 1984). Research based on such clinic attendees implies that conclusions made about the psychological state of people with chronic back pain are based on people who are *not* representative of the larger general community chronic pain population (Jensen et al., 1991; Novy et al., 1995). This indicates that there is limited real knowledge about how *most*

people in the wider community are affected by chronic pain. Furthermore, the participants used in much of the clinical research tend to be males (Ruda, 1993). This is despite literature reports indicating that most people with chronic pain are females (Chrubasik, Junck, Zappe, & Stutzke, 1998; Jensen, Dalquist, Nygren, Royen, & Stenberg, 1997; Turk, Okifuji, & Scharff, 1994). Findings based on research with males does not provide adequate information about how chronic pain affects females in the wider community, especially in terms of adjustment.

According to Blyth, March, Brnabic, Jorm, Williamson and Cousins (2001), there is little known about the reported prevalence of chronic pain in the wider Australian community. They subsequently conducted a general health survey including limited questions about chronic pain among a randomly selected sample of Australian adults. For this survey, chronic pain was defined as "experienced every day for three months in the six months prior to interview" (p. 128). Neither major pain site nor probable cause of the chronic condition was ascertained in this survey. Chronic pain was reported by 17.1% of males and 20% of females. Those most likely to be affected by chronic pain were males in the 65 to 69 year age group and females aged 80 to 84 years. In this sample, chronic pain was significantly related to increased age, female gender, less education and lack of private health insurance.

A non-specific questionnaire was used to assess psychological distress but it is not clear if this was a standardized instrument and questions relating to anxiety and depression were concerned with the four weeks preceding the survey only. However, there appeared to be a strong relationship between psychological distress and experiencing chronic pain. There was no attempt to determine causal relationships in this study. In a more recent NSW survey with 2092 (923M; 1169F) respondents, Blyth, March and Cousins (2003) found that about 24% of females and 20% of males reported chronic pain. However, as informative as this type of research is, it does not necessarily provide information about particular sub-groups of people with chronic pain, such as those who suffer with chronic back pain.

It is also not clear what happens to people with chronic pain over time as most research into psychological dysfunction associated with chronic pain is cross sectional.

There have been some longitudinal studies with clinic patients but evidence for causality is poor or mixed (Brown, 1990; Carter, Feuerstein, & Love, 1986; Feuerstein, Carter, & Papciak, 1987; Hurwitz, Morgenstern & Yu, 2003; Kazis, Meenan, & Anderson, 1983; Kubinski, Rudy, Turk, & Zaki, 1991; Skevington, 1983; Von Korff & Simon, 1996).

Brown (1990) found that initial pain predicted depression while Carter et al. (1986) and Feuerstein et al. (1987) did not find a significant relationship between pain and mood. Hurwitz et al. (2003) reported that psychological distress and pain could both "be causes and consequences of each other" (p. 463), although the associations were not particularly large, and that longitudinal relationships were weaker than cross-sectional associations. Kubinski et al. (1991) found that pain and depression were significantly correlated at both pre- and post-treatment assessment. They further found that pre-treatment pain severity predicted post-treatment depression levels, but pre-treatment depression levels were not predicative of post-treatment pain severity. Kazis et al. (1983) investigated rheumatoid arthritis (RA) patients in an effort to determine the predictability of psychological functioning and pain but did not find a relationship over a period of 6 months. Skevington (1983) found that belief that pain would not be relieved was related to increased risk of depression at follow-up. Conversely, Von Korff and Simon (1996) found that patients with back pain were not more likely to be depressed at follow-up.

Research is also limited to chronic pain that has been experienced for less than about 10 years. This is despite the fact that there are reports of many people who have experienced chronic pain for substantially longer periods. For example, Swanson, Maruta and Wolff (1986) found that people who have had chronic pain for a very long time, i.e. for more than an average of 25 years, reported greater depression, misery, drug dependency and treatment resistance than those who had experienced chronic pain on average, for less than 10 years. There is limited information about such cases of significantly long-standing pain, indicating another neglected area of research.

There has been some limited longitudinal research with people in the wider community who experience chronic pain. For example, Magni and colleagues (Magni,

Marchetti, Moreschi, Merskey, & Rigatti-Luchini, 1993; Magni et al., 1994; Magni et al., 1992) are one of the few groups of researchers who have looked at chronic pain in the general population. In a longitudinal survey of a large general United States community, they found that pain predicted depression but that depression also predicted pain. Moreover, this research was based on people who suffered mixed chronic pain (i.e. in various places in the body). Continued research with people with chronic pain from the wider community offers the opportunity to learn more about this debilitating condition which, in the long-term, may help a greater number of people lead more active and productive lives (Keefe, Dunsmore, & Burnett, 1992).

Another limitation of the chronic pain research is that it often assumes that all people with chronic pain are homogenous and does not account for the effect that different factors, including pain site or "anatomic locus and distribution of pain" (Toomey, Gover, & Jones, 1984, p. 390), may have on adjustment (Jensen et al., 1991). For example, many research studies have used patients with 'mixed' chronic pain syndromes, that is, the primary pain site may vary among individuals. Some may have pain in several places or only in one place, e.g. upper limb, neck, back or legs. This may be especially true of those with systemic pain, such as is experienced with rheumatoid arthritis.

Previous research has shown that the extent of psychological distress experienced by a person with chronic pain may depend to some degree on the place in the body where chronic pain is primarily experienced. For example, those suffering chronic pain in the lower back, sacrum and coccyx have been found to suffer more psychological distress and perceived pain that those who suffered pain elsewhere, such as in the face, head and mouth (Rudy, 1987). This suggests that confining research to people who experience persistent pain in a similar area of the body should reduce some confounding of factors.

Chronic back pain is considered to be one of the most debilitating chronic pain conditions and more common than persistent pain in other parts of the body (Flor & Turk, 1984). Anecdotally, 'the bad back' is the most common condition of this type referred to in the media and in the general community. Also, among general chronic musculoskeletal

pain, people with chronic back pain are somewhat unique in their experiences. This is due to a social stigma that has long been linked to the condition, partly due to an association with litigation in worker's compensation injury claims (Carey, 1994). Even for those who suffer back pain that is unrelated to work injury, this social stigma tends to influence how they and others respond to their condition. This relates especially to the continual feeling that they must justify their pain as being a 'real' phenomenon as opposed to an indication that it is "psychogenic...[or indicative that they are]..'mad or bad'" (Osborn & Smith, 1998, p. 72).

Given these perpetual puzzles of pain, it is reasonable to be curious about the association between chronic pain and psychological dysfunction in the wider community. As a result of the limitations of the psychological research into chronic pain and depression, little is known about how the vast majority of people, especially women, adjust to the condition. Learning more about those who cope in the long term, without extensive medical involvement, may be especially informative in terms of education and treatment for those who have the potential to become severely dysfunctional. Educating the general public about the consequences of chronic pain would seem a more logical step than waiting for the most dysfunctional individuals to be referred to pain clinics.

1.4 A cognitive-behavioural perspective

It has become increasingly clear that in order to address these puzzling issues, research must be conducted within clear and distinct conceptual paradigms. Traditional paradigms have been largely of medical origin and many take a restrictive view of the relationship between chronic pain and depression as either body-centred or mind-centred. Out of the complicated and chaotic pain literature, there has emerged a promising theoretical framework that appears capable of integrating many of the psychological and physical factors associated with pain. This approach is inherently multi-dimensional and incorporates a number of overlapping theories/models, including, for example, cognitive-behavioural theory, a diathesis-stress approach, fear-avoidance models, a reformulated cognitive-behavioural theory and the biopsychosocial perspectives of studying chronic pain.

Many of these have been proposed in more recent years and will be discussed in detail in Chapter 2. Moreover, some post-date the present research and are introduced merely to indicate the dynamic nature of chronic pain research. The cognitive-behavioural perspective, which forms the basis of this thesis, is one of many approaches that tend to explain chronic pain as a dynamic phenomenon consisting of sensory, cognitive, behavioural and affective facets. This perspective is in keeping with more recent approaches to research that acknowledge that pain is best explained from a multi-dimensional perspective. While it is acknowledged that there are many factors that may affect the person in chronic pain, this thesis concentrates mainly on the psychological or cognitive aspects of the chronic pain experience. It is beyond the scope of the present research to do otherwise.

The basic proposition of this perspective is 'reciprocal determinism', defined as a process whereby "each facet [of the chronic pain experience] affects and is [in turn] affected by every other facet" (Novy et al., 1995, p. 244). These facets interact so that a person in pain must constantly appraise and reinterpret the personal experience of pain in a process that may or may not lead to healthy adjustment (Bandura, 1977; Lazarus & Folkman, 1984). This 'cognitive appraisal' process is comprised of the beliefs, feelings, behaviours and emotions that a person has while experiencing chronic pain (Turk, Meichenbaum, & Genest, 1983).

Research has demonstrated that maladaptive cognitive appraisal by people with chronic pain is linked to a failure to adjust, reflected in the development of depression (Lefebvre, 1981; Rudy, Kerns, & Turk, 1988). In line with a cognitive-behavioural approach to understanding chronic pain, there is a range of psychological factors including beliefs, feelings, behaviours and emotions that have been shown to contribute to poor adjustment in the person with chronic pain. They include perceptions of pain severity, perceived interference of pain in daily life, perceived control over pain and perceptions of social support which are available to the person with chronic pain (Kerns et al., 1985; Turk & Rudy, 1988). In addition, the presence of low self-confidence or self-efficacy in terms of coping with pain has also been found to be detrimental to good recovery (Nicholas, 1994).

Furthermore, if emotions such as anger and anxiety become excessive, they too can restrict healthy adjustment (Chapman, 1995). Knowing more about psychological factors associated with chronic pain and depression is important in order to be able to identify those who are more at risk of developing depression (Averill, Novy, Nelson, & Berry, 1996). In addition, depression is more likely to be responsive to intervention and treatment than pain-related dimensions such as intensity, frequency (Haley, Turner, & Romano, 1985; Malone & Strube, 1988) and severity.

Although cognitive-behavioural theory is well respected, there has been no published research, to date, that demonstrates its main proposition of 'reciprocal determinism' (Novy et al., 1995). This may be due to the implication of bi-directionality of the theory and the difficulties inherent in testing such a model, statistically (Novy et al., 1995). There has been some limited research testing of the relationships between chronic pain and depression and specific constructs in linear, uni-directional models (Maxwell, Gatchel, & Mayer, 1998; Rudy et al., 1988; Turk, Okifuji, & Scharff, 1995). The over-riding conclusion of these studies suggests that chronic pain is an antecedent of depression rather than the reverse. It is this orientation to the complexities of pain research that is pursued in detail in this work and will be set in the context of current pain research.

1.5 Overall objectives

The present project was designed to extend previous research that has investigated the psychological characteristics of people with chronic back pain particularly as they bear on adjustment. More specifically, the main objective of the present research is to carry out a longitudinal investigation into the evolution of chronic back pain in a sample of community participants who have not been involved with pain-clinic treatment or programs. The guiding key questions in general terms are, 1) are chronic pain and depression associated and if so, is this relationship direct or indirect, and 2) does chronic pain precede depression or vice versa?

The participants of this research will therefore be referred to as 'community' or 'non-clinical' participants. They will have experienced 'chronic back pain', which refers to persistent pain confined to the thoracic and/or lumbar/sacral regions of the spine. If they also complained of chronic cervical or neck pain or chronic pain in other parts of the body, they were not included in the research.

Investigating people with chronic pain *before* they become so psychologically dysfunctional that they are referred to a pain clinic is important in terms of costs to both the individual and society. Increased public and medical awareness about the condition may help in the identification of those who are most at risk of depression. This has implications in terms of the quality of life enjoyed by people in chronic pain as well as their possible over-utilization of health services. This information is pertinent from a long-term perspective, when adjustment rather than cure is often the only way to cope with intractable pain. Poor adjustment implies psychological dysfunction and poor quality of life for many people.

Specific aims will be outlined in more detail in Chapter 2. The overall objectives of the empirical work presented in this thesis were to:

- determine the general characteristics of non-clinical chronic back pain participants in terms of demographic and pain-related characteristics and cognitive-behavioural responses to pain;
- determine the relevance of 'profiling' to people with chronic back pain;
- compare chronic back pain participants to non-pain participants on several specific cognitive measures to determine the extent to which pain affects cognitive functioning;
- compare sub-groups within the sample of people with chronic back pain, i.e. specifically compare those with depression to those without depression on demographic and cognitive variables;
- consider the role of cognitive appraisal in the chronic pain-depression relationship by testing cognitive-behavioural mediation models, especially gender-specific models;
- investigate the longitudinal relationship between chronic pain and depression to determine whether a cognitive-behavioural mediation model is stable over time;

- discuss the implications of this research for cognitive-behavioural theory;
- examine the implications of this research in terms of community education including the possibility of raising community awareness about the dangers of long-term pain conditions to mental and physical health, social relationships and subsequent economic costs to society.

1.6 Structure of the thesis

This thesis presents an analysis of the psychological characteristics of people with chronic back pain in a community sample of South Australians, within a cognitive-behavioural framework. Chapter 2 reviews the literature relevant to chronic pain and depression, types of pain, aetiology, epidemiology and prevalence of chronic pain and the chronic pain-depression relationship. This includes a critical review of the diverse theories that have evolved to explain the chronic pain experience, including cognitive-behavioural theory. Chapter 3 extends the literature review by examining the history of research into the psychological factors associated with chronic pain and depression, including demographic information and the role of cognitive beliefs and behavioural functioning.

Chapter 4 considers the methodological and conceptual issues related to the design of the research, the questionnaires used, as well as the constraints encountered given that this research topic has been so fraught with controversy. The first or pilot study is also presented in this chapter and incorporates the testing of instruments and determination of hypotheses to be tested. Chapter 5 is concerned with the second study involving the investigation of the demographic and cognitive-behavioural variables associated with chronic back pain and depression. In addition, the possibility of replicating the chronic pain profile classification system developed by Kerns et al. (1985) is also addressed. Included in this chapter is a comparison between people with chronic back pain and non-pain control participants on a selected range of variables. Those who scored as 'depressed' are compared to those who did not, in terms of demographic characteristics and cognitive responses to chronic pain. Finally, this chapter addresses the testing of the role of cognitive

appraisal in a cognitive-behavioural model of chronic pain and depression with a view to inferring causality. In addition, gender specific cognitive-behavioural models of chronic pain and depression will be presented.

Chapter 6 considers the third study, which is a 5-year follow-up study of the original chronic pain participants from Study 2, conducted to inform about the evolution of chronic pain in a community sample. Chapter 7 presents an integration of the findings of the present research and considers implications for community education about the consequences of chronic back pain as well as limitations of the project and recommendations for future research.

As can be seen from the preceding sections, the number of variables and their potential interactions involved in the comprehension of chronic pain is multitudinous. Therefore, it is reiterated that this research is exploratory in nature and all findings must be viewed in a cautionary manner.

CHAPTER TWO

THE HISTORY OF PAIN RESEARCH AND THE CO-MORBID ASSOCIATION BETWEEN CHRONIC PAIN AND DEPRESSION

2.1 Introduction

As indicated in Chapter 1, the chronic pain literature is vast and complicated. This chapter provides an overview of research findings about chronic pain and its association with depression. Initially, the definitions, epidemiology, measurement and research findings related to both chronic pain and depression are reviewed. This is followed by a review of theories that have guided research, and more specifically, the rationale for using cognitive-behavioural theory as the basis for the present research. Methodological constraints, limitations and inadequacies associated with this type of research will be addressed throughout.

2.2 What is chronic pain?

In 1999, Loeser and Melzack endorsed the definition of pain promoted by the International Association for the Study of Pain (IASP), the official global organization developed to study pain. Pain is therefore defined as *"an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"* (Merskey & Bogduk, 1994, p. 1607). There are different types of *pain* referred to in the literature, but most research targets *acute* or *chronic* pain (Loeser & Melzack, 1999; Melzack & Wall, 1988). The distinction relates primarily to *time,* from the Greek word, *chronos. Acute pain* is described as tissue damage that disappears with healing, but if pain persists beyond 6 months, it is commonly considered to have evolved into *chronic* pain (King, 2000). However, researchers do not necessarily adhere to strict definitions of acute and chronic pain. In addition, dichotomising pain into *acute* and *chronic* as a function of *time*

does not take into account the very complex nature of the chronic pain experience that involves both sensory and psychological dimensions.

In the literature, chronic pain has traditionally been further classified into chronic *malignant* pain and chronic *benign* pain (Weisenberg, 1987). Chronic malignant pain is characterized by repeated, continued tissue damage. This type of pain often has long-term damaging or even fatal consequences. Specific virulent cancers and types of arthritis generally produce this type of unremitting pain, which is referred to in the literature as 'real', 'respondent', and 'organic'. Conversely, chronic benign pain refers to pain for which there may be no discernible injury or if it is, damage that has healed. Chronic benign pain is the focus of the present thesis.

This type of chronic pain continues to be one of society's most baffling (Melzack, 2001), incapacitating and expensive medical conditions (Elliott et al., 1999; Newman & Seres, 1986; Verma & Gallagher, 2000). A continual source of distress to those who experience chronic benign pain is that often a lack of physical evidence perpetuates the idea that their pain is not 'real' (Gamsa, 1994b) or that it is 'psychogenic', 'non-organic', 'imaginary' (Pearce & McDonald, 1998) or nonexistent (Turk, 2002). Many people have negative experiences with health systems that question the legitimate nature of pain complaints, especially when objective evidence is lacking (Banks & Kerns, 1996; Maxwell et al., 1998). For example, the practice of diagnosing pain as 'psychogenic' may only serve to stigmatise the person with chronic pain (Sullivan, 2000). Health professionals as well as lay people may conclude that complaints made by those with chronic pain are related to litigation processes or a desire for drugs (Carey, 1994). Although this can be a common reaction, research attempting to discriminate psychologically based pain from physically based pain, has not been successful (Joukamaa, 1991; Perry, Heller, & Levine, 1991).

The long medical tradition of referring to chronic pain as either malignant or benign, 'real' or 'imaginary', has recently been criticized as a redundant diagnosis by Turk (2002). As he notes, to those who suffer with prolonged pain, irrespective of its considered source, "no pain is benign" (p. 75). This is not to invalidate the very real pathology associated with

cancers and other pain producing medical conditions, but to acknowledge that the nociceptive and psychological aspects of pain deserve consideration, irrespective of the source. Also, there is increased evidence that persistent pain may be fatal, regardless of origin, due to suppression of the immune system (Liebeskind, 1991). In the present literature review, chronic pain refers to prolonged pain not specifically linked to pathology, however the term 'chronic benign pain' will also be used in some instances, in keeping with its use in the literature.

2.2.1 Measurement of chronic pain

It is difficult to capture the true essence of the difficulties associated with measuring pain. A term such as *chronic pain* refers to what is essentially an inner state which can only be measured indirectly and which consists of different aspects such as intensity and variable persistence (McDowell & Newell, 1987). Different terminology may be used to describe a similar notion i.e. disability, severity, intensity, persistence etc. Definitions and theories of pain have puzzled and eluded researchers and health care workers alike for centuries (France et al., 1988). As it is now generally accepted that the experience of chronic pain is always subjective (Merskey, 1991; Verma & Gallagher, 2000), *measuring* chronic pain is clearly one of the most difficult and challenging areas of health measurement (McDowell & Newell, 1987).

According to Roy (1986) in his critical review of the pain literature, most researchers have implied that chronicity was synonymous with severity without clear definitions of either. Many have failed to report frequency or intensity of pain and some failed to report duration of pain. Even though the traditional definition of chronic pain assumes that pain has been experienced for most of the time, for at least the previous six months, this definition is ignored in many research studies. Each of these aspects of pain alone is a poor indicator of the extent of suffering associated with chronic pain. A decade later, Jensen, Turner, Turner and Romano (1996) voiced much the same concerns about lack of consistency in

measurements. These criticisms are appropriate today as many research studies still fail to use reliable and valid measures to assess perceived pain severity.

Confounding measurement difficulties and consistent with the traditional medical perspective, it appears that most people believe that pain perception is directly proportional to tissue damage (Philips, 1989). Moreover, medical tests often fail to reveal or identify damage in many cases (Loeser, 1980), which can leave people with chronic pain frustrated about the questioning of their credibility. No technological instrument has yet been developed which can clearly objectify the severity of chronic pain. Unlike other chronic conditions such as malignancies and arthritic conditions, the severity of chronic pain "cannot be graded in terms of patho-physiologic signs or objective radiographic or laboratory results" (Von Korff, Ormel, Keefe, & Dworkin, 1992, p. 134).

Although "[p]ain is a personal experience and not readily reduced to descriptive language" (Craig, 1989, p. 221), the only way of gauging pain severity is to *ask* people to report their perceived level of pain. Such 'self-reports' have been criticized for being 'subjective' and subject to under and over reporting, thus lacking scientific credibility and validity (Jensen & Karoly, 1991; Turner & Jensen, 1993). However, Turk and Rudy (1986) argue that pain is "ultimately a private, subjective, experience or perception and thus by definition, a patient's report of pain is the end result of a cognitive process" (p. 764). Jensen, Turner, Romano and Karoly (1991) also argue that people in pain know themselves better than anyone and they alone are privy to their thoughts and covert coping techniques. White and Strong (1992) and Yang, Clark and Janal (1991) consider self report to be valid especially with respect to pain intensity and levels of activity. Only the person who suffers with chronic pain can know the duration and severity of his or her pain and self-reports are easy to obtain and relatively inexpensive, in resource terms. Despite the controversy, Jensen, Romano, Turner, Good and Wald (1999) have provided more recent evidence that self-report continues to be a valid measure of beliefs. Most researchers accept the self-report as an important measure of severity, given that there is no 'gold standard', or objective measure (Von Korff et al., 1992).

Turk and colleagues (Rudy, Turk, Zaki, & Curtin, 1989; Turk et al., 1995; Turk & Rudy, 1988; Turk & Rudy, 1990) and many other researchers have used the Multidimensional Pain Inventory (MPI) to measure pain severity. This is considered a standard, reliable and valid measure that is used in the present research and will be discussed fully in Chapter 4. Suffice to say that it has been shown that those who perceive their pain as high also tend to perceive it as a great interference in their lives, they report greater affective distress and less control. In contrast, those who appear to adjust to pain, report lower levels of pain severity. However, the relationship between pain severity and depression may be affected by mediating factors as Rudy et al. (1988) have demonstrated. This will become more apparent in the following sections.

2.2.2 Epidemiology of chronic pain

According to epidemiological research, chronic pain is widely prevalent and of considerable public health concern (Elliott et al., 1999; James, Large, Bushnell, & Wells, 1991; Von Korff et al., 1988). Seers (1992) indicated that "chronic pain is a major health problem ....[affecting up to]..... 30% of [industrialized] populations" (p. 452), while other estimates range from 7% (Bowsher, Rigge, & Sopp, 1991) to 40% (Brattberg, Thorslund, & Wikman, 1989). Many studies have investigated the prevalence of chronic pain in surveys, often as part of community general health surveys.

For example, Von Korff et al. (1988) found in a postal survey in Seattle, in the US, that 41% of patients reported back pain, 26% reported headache, 17% abdominal pain, 12% chest pain and 12% reported facial pain. For most of the respondents, pain was persistent and recurrent. The life-time probability of people in this survey experiencing back pain was estimated to be about 85%. It was reported that those with some type of pain were more likely to be anxious or depressed. The frequency of major depression was estimated to be 3 to 5 times higher in those with a pain condition compared to those with no pain.

Croft, Rigby, Boswell, Schollum and Silman (1993) found that chronic widespread pain was reported by 11.2% of 1319 adults who completed questionnaires in two general

practice areas in northern England. More females (15.6%) reported chronic pain than did males (9.4%). Of those in pain, 31% also reported that they were depressed. Andersson, Ejlertsson, Leden and Rosenberg (1993) investigated chronic pain in two general Swedish populations (N=1806). They reported that 55% of the population had been experiencing persistent pain for more than 3 months (54.9% M; 55.5% F). Males aged 55 to 59 years and females aged 50 to 55 years experienced the greatest occurrence of persistent pain, which then tended to slowly decline with age. For both genders, pain was most common in the neck-shoulder area, followed by the low back area.

In a more recent UK community study of 3,605 people, Elliott et al. (1999) concluded that 45.5% of the general public suffered chronic pain and about one third of these suffered with chronic back pain and/or arthritis. In a four-year follow-up to this study, Elliott, Smith, Hannaford, Smith, & Chambers, (2002) reported that chronic pain in this community had increased to 53.8%.

There is limited data on the prevalence of chronic pain in Australian communities (Helme & Gibson, 1997). As mentioned in Chapter 1, Blyth et al. (2001) (N=17,543), found, in an Australian community survey, that 17% of males and 20% of females reported chronic pain. Chronic pain was most reported by males aged 65 to 69 years and females aged between 80 and 84 years. While depression was not specifically assessed in this study, 'psychological distress' was found to be significantly greater in those respondents who reported chronic pain, especially pain perceived as an interference. Pain was more likely to interfere in daily life if one was younger, female and did not have private health insurance. More recently, in a NSW survey with 2092 (923M; 1169F) respondents, Blyth et al. (2003) found that about 24% of females and 20% of males reported chronic pain.

With respect to research with community samples, they are especially difficult to acquire, from both a financial and a logistic perspective (McWilliams, Cox, & Enns, 2003). Random sampling from electoral rolls, general practice registers or phone registers, while considered to be the ideal way of acquiring random samples, is not straightforward as it is necessary for proposed participants to already be experiencing chronic pain. Some

researchers have used national health surveys to acquire research participants (Blyth et al., 2001; Magni et al., 1994), but this is not always possible with research projects. Even if selected registers are used they may still be biased. For example, Croft et al. (1993) found that more females were not only registered in selected general practice areas, but also that women were more likely to complete questionnaires.

These studies emphasize that chronic pain needs to be studied at the community level, not just in pain clinic attendees, and that without appropriate management those with chronic pain are at risk of over-utilization of medication and health care services. According to the Australian Bureau of Statistics (2002), taking medication is one of the most health-related behaviours performed by Australians. In 1995, 59% of the population had recently used one or more medications and the most commonly used medication was for pain relief (24%). By gender, 64% of women had taken one or more medications recently and 27% of this medication was for pain relief. The corresponding figures for males were 54% and 20%, respectively. Also provided by the National Health Survey, 2001, 24% of the Australian public consulted a general practitioner or specialist in the previous two weeks. Females (27%) were more likely to consult most types of health professionals than males (21%) and were two to three times more likely to visit allied health professionals such as naturopaths.

With respect to age and gender of people with chronic pain, findings are mixed. For example, Bowsher et al. (1991) have reported that more chronic pain is found in older age groups while Brattberg et al. (1989) reported the opposite, but in a younger population sample making comparisons difficult. There are other controversial findings about prevalence and age depending upon pain site and type (James et al., 1991; Sternbach, 1986). Crook, Rideout and Browne (1984) have reported that women suffer more chronic pain than men while Brattberg et al. (1989) found no gender differences. Women are more likely to seek medical treatment (Bush, Harkins, Harrington, & Price, 1993; Nolan, 1994; Verbrugge, 1985), but they appear to be underrepresented in clinical research, which is based predominantly on males (Ruda, 1993).

Despite these findings with respect to the occurrence of chronic pain, Crombie (1999) noted that epidemiological research into chronic pain is still in its infancy and publications are widely spread among journals from different disciplines, which hinders comparisons. Published statistics often appear disparate, possibly due to different definitions used by researchers (Crombie, 1994). If pain is defined as 'minor', (Von Korff et al., 1988), prevalence is higher than if pain is described as 'severe' (Crombie, Davies, & Macrae, 1994). In a review of the epidemiological literature, Verhaak, Kerssens, Dekker, Sorbi and Bensing (1998) also noted that empirical studies varied in type of pain investigated, for example, musculoskeletal pain, abdominal or general pain. Definitions of chronic pain varied from one month (Magni et al., 1994) to six months (Brattberg et al., 1989) or no time specified at all (Andersson, 1994). Some studies gave incidence of chronic pain (Potter & Jones, 1992), while others gave prevalence estimates (Andersson, 1994; Sternbach, 1986).

It seems that establishing the prevalence of chronic benign pain is extremely difficult given lack of aetiologies, the complex nature of the experience (Verhaak et al., 1998) and diverse methodologies. Despite this, "forceful statements have been made about the prevalence and the costs and impact on social security systems of chronic pain" (Verhaak et al., 1998, p. 232). These include conclusions that chronic pain affects about 30% of the British (Seers, 1992) and US populations (Bonica, 1990) and costs the US $150B annually (United States Bureau of the Census, 1996). The previously mentioned studies appear to bear these figures out – that about 20 to 25% of adult populations in industrialized countries experience chronic pain. Moreover, describing the frequency and costs of chronic pain does not inform about the suffering or complexity associated with the syndrome (Crombie, 1994). Furthermore, knowing the prevalence of chronic pain does not inform about the prevalence of specific chronic pain conditions, such as chronic back pain.

2.2.3 *Chronic back pain: Prevalence, costs and aetiology*

Within the chronic pain population at large, there are sub-groups who deserve continued research and one that is most unique is a distinct subgroup, those who experience chronic back pain. As mentioned, chronic back pain is one of the most common and disabling types of chronic benign pain (Holzman & Turk, 1986; Loeser, 1980; Pearce & McDonald, 1998). This is described broadly as persistent and prolonged pain that occurs in the thoracic, lumbar and sacral-coccyx areas of the spine[1]. Despite the methodological difficulties of pain research, estimates of the prevalence of chronic back pain and its societal costs have been proposed. Western countries, including the United States and Europe, provide most data, with a limited amount from Australia (Walker, 1999).

Chronic back pain affects most of the population at some time, accounting for millions of medical visits each year (Mayer et al., 1986). Despite much research and medical and technological advances, the widespread disability associated with chronic low back pain continues to escalate (Waddell, 1998, cited in Turk & Okifuji, 2002). Back pain is documented as the third leading cause of physical limitation and disability with about 4% of the population permanently disabled by it. Verma and Gallagher (2000) report that annually, up to 15% of US adults experience work disability related to back pain alone. It has been estimated that once a back pain condition becomes chronic, only 50% of those experiencing chronic pain will be able to return to full-time employment. Most recently, Main and Williams (2002) described the prevalence of chronic pain, "including back pain [....] as an epidemic" (p. 534).

Carey (1994) reports that the "[United States] has the highest rates of surgery for back pain" (p. 113) in the world. This type of surgery is also a major consumer of health care resources, leading to early retirement and demands for compensation payments (cited in Linton, 1994). Back pain and pain disorders cost America more than $US50 to $US100

[1] Note here that there is also a vast research which has arisen from the study of chronic low back pain specifically or pain that occurs from the 12th thoracic vertebrae through to the coccyx. Due to the difficulty of acquiring participants for the present research, people with thoracic back pain were also included rather than just focusing on chronic low back pain.

billion every year in compensation, health-care and lost productivity (Bonica, 1980; Frymoyer, 1992; Osterweis, Kleinman, & Mechanic, 1987).

In the United Kingdom, back pain is estimated to cost £480 million per year, with a population prevalence of 16.5 million (Pearce & McDonald, 1998) and a 1-year prevalence of nearly 50% (Palmer, Walsh, Bendall, Cooper, & Coggon, 2000). Elliott et al. (1999) have concluded that about 15% of the general public suffer with chronic back pain and/or arthritis in the UK. Thomas et al. (1999) estimated that 7% of adults in the UK report to their doctors annually with acute low back pain which, in a small percentage of cases, persists to become a chronic condition. In a study with 180 participants who provided complete follow-up data, they found that 42% continued to have persistent low back pain at 12 months. Consistent with this, Macfarlane et al. (1999) comments that less that "10% of the approximately 23 million episodes of low back pain occurring annually in the UK lead to a general practitioner consultation" (p. 113). In Europe, it has been estimated that low back pain constitutes about 13% of all chronic conditions (Frølund & Frølund, 1986; Mäkélä & Heliövaara, 1986).

In Australia, Walker (1999) reported that the "true prevalence of low back pain in the Australian community [is].... unclear" (p. 50). Although more recently, Blyth et al. (2003) reported that the most common primary site for chronic pain, among people in an Australian survey, was the back. In their NSW survey, they found that 45% of people indicated that their back was the primary site of chronic pain. Furthermore, and consistent with overseas figures, work-related back injury in South Australia constitutes about 25% of all workers' compensation claims (WorkCover, 1998). The total lump sum payment for permanent disability from back injuries for the same period was in excess of $A16B. Furthermore, these figures only relate to work-related back injury. These statistics do not necessarily inform about the level of incapacity experienced at a community or individual level, or actual causes of chronic back pain.

Despite extensive research, little is known about the causes or aetiology of chronic back pain (Flor & Turk, 1984). In most cases, no ".....specific pathology of the nociceptive system" (Birbaumer et al., 1995, p. 332) is demonstrated that can easily explain why a pain

response is maintained. Unlike acute pain, which protects a person from further harm, chronic pain seems to serve no useful, biological purpose or survival mechanism (Morris, 1992). A range of diagnostic terms (e.g. lumbosacral strain, sciatica, lumbago, spondylosis, osteoarthritis, myofascial pain syndrome etc.) is used to describe back pain, but the terms are mostly a description of symptoms rather than an indication of aetiology.

Disc herniation and arthritis of the vertebral joints are the only causes of chronic back pain that have been clearly identified (Melzack & Wall, 1982). Also, as previously mentioned, there are many cases of reported disc degeneration that do not produce excessive or prolonged reports of pain (Turk, Rudy, & Sorkin, 1992b). Various undetected visceral diseases i.e. kidney disease, have been found to produce low back pain and scarred tissue from back surgery can become a pain site. Some health workers consider that misalignment of the spinal column causes most pain (Keller & Colloca, 2000; Kerr, 2000). Others report that spinal abnormality may not be related to back pain (Riihimaki, 1991). This lack of consensus and cohesion among specialist health workers is an impediment to treatment efficacy for the person with chronic back pain.

The prevailing medical model of health tends to generate a belief that knowing the cause of a medical condition is essential. This is counterproductive in terms of chronic back pain, given that most people cannot identify a precipitating factor. From a psychological or cognitive-behavioural perspective it is more useful to investigate maintaining factors, which are more likely to be responsive to behavioural and cognitive changes.

2.3 Chronic pain and depression

As mentioned, depression is one of the most common conditions to be associated with chronic pain and this connection has generated an extensive literature in the last two decades (Banks & Kerns, 1996; Fishbain et al., 1997; Gallagher & Verma, 1999; Magni, 1987; Romano & Turner, 1985; Roy, Thomas, & Matas, 1984; Turk & Rudy, 1987a). As depression is a serious health condition it its own right, the following section provides details about how it is defined and assessed and its reported prevalence.

2.3.1 *Depression: Definition, assessment and prevalence*

Depression, referred to as one of the world's leading public health problems (Beck, 1967), has been defined as "a recurrent, episodic condition with a heterogeneous course, associated with varying degrees of social impairment, recovery, and susceptibility to relapse" (Coyne & Downey, 1991, p. 406). The term, *depression,* is used by various health workers including psychiatrists, medical doctors, psychologists, social workers and other para-medical workers to describe a range of psychological experiences including clinical syndromes/conditions, disorders, moods or a set of symptoms (Fombonne, 1994). It may be perceived as a single or a multi-dimensional entity that can result in disparate definitions and complicated assessment. *Depression* in the present research refers to an inner mood state or collection of depressive symptoms measured by self-report assessment, rather than a clinical diagnosis, which is beyond the scope of the present research. Consequently, in this thesis, *depression* and *depressive symptomatology* will be used interchangeably.

The presence of depression may be indicated from a wide range of assessment strategies including structured, semi-structured and non-structured interviews, chart reviews, projective tests and self-report assessments (Romano & Turner, 1985). There are three main medical sources of depression criteria used by health workers to identify and characterize depression symptoms (Diener, Van Schayck, & Kastrup, 1995). These are the Research Diagnostic Criteria (RDC: Spitzer, Endicott, & Robins, 1978), the Diagnostic and Statistical Manual of Mental Disorders (APA, 1994) and the International Classification of Diseases (ICD-10: World Health Organization, 1991). These sources are used mainly in interview situations and each incorporates a variety of terms, some of which may refer to similar conditions. These include terms such as major depression, major depressive disorder, depressive episode, endogenous depression, non-endogenous depression, dysthymic disorder, intermittent depressive disorder, atypical depression and minor depressive disorder.

The sources describe particular symptoms within each diagnostic category, and they may vary on the number of symptoms necessary for a diagnosis. The symptoms generally

include depressed mood, loss of interest, sleep, appetite and sexual disturbances, motor disturbances, feelings of guilt and suicidal ideation. There are also differences between the medical systems in required duration of symptoms. For example, for a diagnosis of a major depressive disorder, the DSM-IV requires the presence of specific symptoms for at least two weeks, whereas the RDC specifies only 1 week for a diagnosis of depressive episode.

Among the most common self-report inventories or questionnaires used to assess depressive symptoms are the Beck Depression Inventory (BDI: Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the Center for Epidemiological Studies-Depression Scale (CESD: Radloff, 1977) and the Zung Self-Rating Depression Scale (Zung, 1965). These measures were designed to assess depression in psychiatric groups or for general public screening (Estlander, Takala, & Verkasalo, 1995). They consist of a number of questions or statements that address various sensory, cognitive, behavioural and affective symptoms, characteristic of depression. Each statement is assigned a value and a sum score is calculated which indicates level of depressive symptomatology. The higher the score, the more the individual is considered to be at risk of depression, or a cut-off score distinguishes the depressed individual from the non-depressed individual.

This type of assessment allows depression to be graded on a continuum scale of severity, i.e. from absent to mild to severe as opposed to the discrete categories provided by the DSM and the RDC. Much of the chronic pain literature refers to the use of these self-report questionnaires due to their convenience and psychometric advantages (Flett, Vredenburg, & Kramer, 1997).

According to Diener et al. (1995), in Western nations, the estimated likelihood of suffering from a *major depression* during one's life is about 20 to 30%. Women are reported to be four times as likely to suffer from major depression, as are males (Klerman, 1988). In Australia, depression reportedly affects over 800,000 people each year, with 25% of women and 17% of men being affected at some time during their lives (beyondblue, 2001). Depression can have severe consequences in the long term, such as a deleterious effect on general health (Angst et al., 1996; Hays et al., 1995), or at worst, suicide (Klerman, 1988).

Depressed people have been found to suffer as badly, in terms of physical functioning and well-being, as persons with other chronic illnesses such as hypertension, diabetes and congestive heart failure (Hays et al., 1995). Depressed persons have also been reported to spend more time in bed than do non-depressed people with severe medical conditions (Angst et al., 1996).

2.3.2 The co-morbid association between chronic pain and depression

Chronic pain and depression together constitute a significant medical problem in society today (Verma & Gallagher, 2000). People with chronic pain who are also depressed are often considered to have a co-morbid condition, defined as "any distinct entity that has existed or that may occur during a patient's clinical course [with] the index disease under study" (Feinstein, 1977, p. 455). An indication of the gravity of co-morbidity is that such people are even more likely to consider and complete suicide at higher rates than the general public (Fishbain et al., 1997) or people not in pain. For example, Breslau, Davis, and Andreski, (1991) found that suicide attempts among young persons suffering chronic migraine were more common than among patients who did not suffer chronic pain. Depression associated with chronic pain clearly represents a potentially dangerous combination.

According to a review by Sullivan et al. (1992), the reported prevalence of depression among people with chronic pain ranges from 8% to 100% (Brown, 1990; Fishbain, Goldberg, Meagher, Rosomoff, & Rosomoff, 1986; Haythornthwaite, Sieber, & Kerns, 1991; Romano & Turner, 1985; Roy et al., 1984; Rudy et al., 1988; Turk & Rudy, 1987a). As with estimating chronic pain prevalence alone, this disparity could be related to diverse samples (i.e. psychiatric versus pain clinic) and measures (i.e. standard versus in-house), pain conditions and ages (Bukberg, Penman, & Holland, 1984; Magni et al., 1993; Morris, Robinson, & Raphael, 1990; Schleifer, Slater, Macari-Hinson, Coyle, Kahn, Zucker, & Gorlin, 1991). However, in keeping with the growing trend for more rigorous research, Banks and Kerns (1996) have concluded that depression among those with chronic pain ranges from 30% to

54%. This is much higher than that found in the general population. For example, Magni et al. (1993) found that depression in the general population ranged from 6% in those without chronic pain to 16% among those with chronic pain.

These estimates, however, depend on consistency of measurement and Fishbain et al. (1997) has reported that much of the research has failed to use standardized instruments for measuring both pain and depression. Pincus and Williams (1999) have further criticized many of the standardized instruments used in chronic pain-depression research, arguing that their use is often based on the assumption that 'pain' and 'depression' are "independent and homogenous syndromes [.....and yet may be assessed by.....] inappropriate measurements" (p. 215). That such assumptions may mitigate findings is highlighted by other research that has demonstrated that the very concept of depression may differ between clinically depressed people and those with chronic pain (Pincus, Pearce, & McClelland, 1995). Pincus and Williams (1999) indicated that continued research into the development of new measurements of depression in chronic pain is warranted.

Consistent with these ideas, Morley, Williams and Black (2002) found that responses of chronic pain patients to the individual items of the Beck Depression Inventory (BDI), one of the most used depression measures, was "strikingly different" (p. 289) to that generally found with psychiatrically depressed patients, not in pain. In particular, the tendency to self-denigrate in chronic pain patients was significantly less than has been observed in the clinically depressed. This supports comments by Williams (2001) who questioned the usefulness of anxiety and depression measures, originally developed for psychiatric populations, in the assessment of affect among those with chronic pain. Such findings emphasize the dynamic and controversial nature of chronic pain research and that there are contentious issues that remain unresolved.

Some general conclusions about chronic pain and depression have been reached, although, given the previous comments, such conclusions remain controversial. For example, it is believed that the likelihood of developing depression may be greatest in the early years post-onset of the chronic pain condition (Love, 1987). Sullivan et al. (1992)

suggest that this implicates mediation factors in the association between chronic pain and depression but most research, being cross-sectional, fails to address this. Also, the weight of research findings suggests that depression is more common among people in pain than those who are not. There is also considerable support for the theory that chronic pain and depression are related (Anke, Stenehjem, & Stanghelle, 1995; Holroyd, France, Nash, & Hursey, 1993; Krittayaphong, Light, Golden, Finkel, & Sheps, 1996; Stenager, Stenager, & Jensen, 1994; Vimpari, Knuuttila, Sakki, & Kivela, 1995) and that they occur in that order (Breslau, Davis, Schultz, & Peterson, 1994; Brown, 1990; Holroyd et al., 1993; Magni et al., 1994; Rains & Lohr, 1993).

(i) Longitudinal research

Most pain research to date has been cross-sectional (Turk & Okifuji, 2002). There has been longitudinal research although fewer studies particularly target chronic back pain and findings are mixed. For example, Kazis et al. (1983) investigated RA patients in an effort to determine the predictability of psychological functioning and pain but did not find a relationship over a period of 6 months. Carter et al. (1986) and Feuerstein et al. (1987) examined the reciprocal influence of mood and pain in chronic back pain patients over a 2-week period but did not find a significant relationship between pain and mood.

Brown (1990) conducted a comprehensive longitudinal study with 243 RA patients that involved six phases of data collection over a 3 year time period. No causal relationship between depression and chronic pain could be determined in the first 12-month period. However, in the last 12 months of the study there was evidence that initial pain predicted depression when initial depression was controlled for. Brown (1990) concluded, that this study "failed to find a strong and consistent causal relationship between pain and depression" (p. 135). Kubinski et al. (1991) found in 80 chronic pain patients that pain and depression were significantly correlated at both pre- and post-treatment assessment. They further found that pre-treatment pain severity predicted post-treatment depression levels, but pre-treatment depression levels were not predicative of post-treatment pain severity.

Both these latter studies used the CES-D in their research.

Most recently, Hurwitz et al. (2003) studied 681 low back pain patients in a treatment program to determine whether psychological distress affected chronic pain and vice versa. Patients were assessed at 6 weeks and then 6, 12 and 18 months, respectively. 'Psychological distress' was a composite measure, including depression and anxiety. The study concluded that psychological distress and pain could both "be causes and consequences of each other" (p. 463), although the associations were not particularly large, and longitudinal relationships were weaker than cross-sectional associations.

There has been some longitudinal research with people with chronic pain from the wider community, for example, Magni et. al (1994). Using the CES-D to measure depression, they investigated chronic pain in a large United States general community (N=2341) over 8 years. They found that depression was more common amongst people with chronic pain (16.4%) than those without (5.7%). They also found that depression at Time 1 (T1) significantly predicted pain, at Time 2 (T2) while pain at T1 also significantly predicted depression at T2. However, while pain predicted depression slightly more than the reverse, both effects were minimal. These results are similar to the later results found by Hurwitz et al. (2003) with clinical patients. Magni et al. (1994) concluded that each scenario could fit equally well for different groups of people. However, the research was based on individuals who suffered musculoskeletal or abdominal chronic pain and one survey was restricted to American Hispanics. Also, pain was defined as that which had persisted for more than 1 month, which is not consistent with the orthodox medical definition requiring persistent pain to be present for at least 3 to 6 months before it is considered to be chronic (Philips & Grant, 1991b). These limitations make comparisons with other studies difficult.

(ii) Duration of chronic pain and depression

There are limited and mixed findings about how duration of the chronic pain condition affects psychological functioning. Longer duration of pain and illness has been found to be associated with increased risk of depression and disability (Averill et al., 1996) and pain report and disability (Groth-Marnat & Fletcher, 2000; Sullivan et al., 1992). It has also been

found that depressed chronic pain patients respond to treatment better if they have experienced pain of a shorter duration (Dworkin, Richlin, Handlin, & Brand, 1986). There is also a suggestion that older people in pain are less psychologically affected (Riley, Wade, Robinson, & Price, 2000), but it is unclear whether this is due to age and experience or because they have been in pain longer and have habituated to the condition. Older people with chronic pain have been reported to experience fewer total hours of pain per day (Herr, Mobily, & Smith, 1993).

Most chronic pain research focuses on people who have experienced chronic pain for about 10 years. Furthermore, there are subgroups of people who have suffered chronic pain for substantially longer periods. For example, Swanson et al. (1986) identified a sub-group of 45 people with chronic pain who had experienced chronic pain for an average of more than 25 years and this was described as *ancient pain.* This group was compared to a control pain group, members of which had experienced chronic pain for an average of 6.6 years (N=217). On most measures, such as basic demographics, compensation status, neurological-orthopedic findings and personality measures, there were no significant differences between the groups. In addition the ancient pain group reported greater depression, misery, drug dependency and treatment resistance (Swanson et al., 1986). There is limited information about such cases of significantly long-standing pain, indicating another neglected area of research. Given these various findings, it is reasonable to be curious about the association between chronic pain and psychological dysfunction in the wider community.

2.3.3 Chronic back pain and depression

With respect to chronic back pain in particular, there is also little consensus on the prevalence of depression amongst those who experience it. Information about chronic pain, in general, is not necessarily informative about such a sub-group. Chronic back pain is considered to be "the most frequent manifestation of pain and one of the leading causes of early retirement" (Diener et al., 1995, p. 351). Patients who have repeated unsuccessful

back surgeries are often referred to as suffering the "failed back" syndrome (Wilkinson, 1983). According to Krishnan, France, Pelton, McCann, Davidson and Urban (1985) and Long (1988) the prevalence of depression in people with chronic back pain, with no clear injury, is reported to be about 85%, compared to a prevalence of 18% among single lumbar-disc protrusion patients (Hasenbring & Ahrens, 1987). In their 1992 review, Sullivan et al. found that 62% of people with chronic pain were clinically depressed while 21% were diagnosed with major depression or major depressive disorder. They also concluded that major depression is 3-4 times more prevalent in those clinical patients with chronic pain with chronic low back pain than in the general public. Again this does not indicate the rate of depression in the broader category of people with chronic back pain in the wider community.

Determining the prevalence of depression in chronic pain is often confounded by symptom overlap (Banks & Kerns, 1996). Somatic symptoms such as motor disturbances, weight and appetite changes, sleep disturbances and fatigue could be attributed to either condition. Some depression inventories have been modified to reduce this predicament, but this further confounds the issue by impeding direct comparison of pain and depression rates across studies (Banks & Kerns, 1996). The BDI in particular, has been criticized for problems with test-retest reliability with chronic pain samples due to the dynamic nature of pain. That is, if people with chronic pain are tested when pain is severe and then when pain is less severe, the correlation between the two has been found to be poor (Holm, Penzien, Holroyd, & Brown, 1994). Other problems have been highlighted by factor analysis of the BDI, revealing that the cognitive/affective factor does not consistently correlate with pain intensity whereas the somatic factor does (Holm et al., 1994; Wesley, Gatchel, Polatin, Kinney, & Mayer, 1991). Some instruments such as the CES-D scale (Radloff, 1977), tend to allow for this more than others. As mentioned, another problem with evaluating much of the research is that depression is as difficult to define as is chronic pain.

2.4 Perspectives, theories and models of chronic pain

Adequately defining and understanding pain and associated psychological dysfunction has proven to be a daunting task given that it is still one of the least understood medical phenomena (Bonica, 1974; Melzack, 2001; Morris, 1992). Historically, pain has been explained within various paradigms depending upon the times and associated philosophies. An early view was that Western conceptualisations of pain have evolved from a religious explanation. The word *pain* appears to be derived from the Latin *poena,* meaning punishment handed down by God (France et al., 1988). An alternative view, derived from Aristotle, the 'Father of Western Thought', is that pain is the opposite of pleasure and therefore a negative feeling state. Aristotle believed that "the heart was the center of sensation" (cited in France et al., 1988, p. 3) and that the flow of blood to the heart was involved in the movement of pain through the body. These original ideas about pain have been usurped over the centuries by radical changes in thinking.

Within the last decade or so, there have been prolific writings about the evolution of chronic pain research, which show its dynamic nature. Moreover, reviewing the literature is complicated by the tendency for 'old wine' to be presented in 'new bottles' as researchers attempt to improve understanding of pain by categorization of theories and models. For example, Novy et al. (1995) proposed eight 'theoretical perspectives' of chronic pain while Adams and Taylor (1997) presented five 'models' of chronic pain. While these two reviews address the topic from different positions, there is considerable conceptual overlap, which is typical of much of the literature.

Given these complications, reviewing the vast literature on this topic is beyond the present scope, so only that considered relevant will be presented here. Subsequently, this review follows the format of the Novy et al. (1995) work since that takes a unique and innovative approach to the evolution of ideas about chronic pain and is much more detailed than many other researchers. Consequently, some of the terminology may be considered unorthodox. As the Novy work provides insight into perspectives of chronic pain only until the early 1990's, additional literature that provides the most recent ideas on chronic pain to

the early 2000's will also be presented here. This includes research by prominent writers in the field including Vlaeyen and colleagues, Jensen et al. (1999), Pincus and Williams (1999), Sharp (2001), Main and Williams (2002), and Turk and Okifuji (2002).

2.4.1 Uni-dimensional perspectives

In the 17th century, Descartes, a French philosopher, explained pain from a *mind-body dualistic* perspective. This perspective has a single or uni-dimensional focus. According to Novy et al. (1995), such a focus is an example of a 'restrictive' perspective and they identify several other such perspectives. Novy et al. (1995) refer to these as the *psychological*, the *radical operant-behavioural* and the *radical cognitive* perspectives.

A mind-body dualism perspective derives from Descartes' assumption that, contrary to Aristotle's ideas, the brain, not the heart was the centre of sensation (1664, cited in Bonica, 1977). This was the basis for a view of pain that still dominates many areas of modern medical research and treatment. This view led to the *specificity* and *pattern* theories of modern medicine (see Melzack & Wall, 1982), which commonly define pain such that degree of symptomatology is directly proportional to degree of tissue damage. Consistent with this, medical laboratory work has historically investigated pain within a *stimulus-response* framework. Thus, pain is perceived as 'body-centred' and therefore comes under the jurisdiction of clinical medicine. As such, the principle methods of treatment generally involve pharmacological or surgical procedures, which attempt to block the sensory aspect of pain. The efficacy of such treatments, however, is not consistent (Carey, 1994; Linton, 1994) and this is compounded by the fact that physicians are often unable to establish a specific aetiology for chronic pain (Turk & Holzman, 1986b).

If there is a lack of physical evidence for the pain, it may be described as 'psychogenic', 'psychological' or 'psychiatric' pain. This encouraged the dichotomous way of perceiving pain as either 'body-centred' (somatogenic) or 'mind-centred' (psychogenic). This is embodied in the diagnostic category of the Somatoform Pain Disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; King & Strain, 1996). Pain that

cannot be attributed to physical causes but is subsequently defined as psychogenic is also thought by some medical workers to reflect 'abnormal illness behaviour' (Pilowsky, 1969; Waddell, Main, Morris, DiPaola, & Gray, 1984). As indicated earlier, the assumption that pain must originate from either one or the other continues to predominate in some health care circles.

The *psychological* perspective, according to Novy et al. (1995), takes a 'mind-centred' approach, focusing on the relationship between psychological factors, particularly depression, and chronic pain (Merskey, 1980). It is acknowledged here that the term 'psychological perspective' might be considered a misnomer by some writers. For example, Adams and Taylor (1997) referred to five 'psychological' models of chronic pain, "proposed to explain why pain may progress to become chronic" (p. 69), which were psychodynamic/personality, behavioural, cognitive, cognitive-behavioural and psychophysiological models. This is an example of the less than consistent use of terminology that has plagued the chronic pain literature.

Novy et al. (1995), in this instance, refer to an approach that is embodied in a prolific 'pain-prone personality' literature and/or similar (see Beutler, Engle, Oro'-Beutler, Daldrup, & Meredith, 1986; Blumer & Heilbronn, 1982; Engel, 1959; Pilowsky, 1982, for more detail). Researchers who favour such a psychiatric or psychodynamic perspective tend to support an antecedent hypothesis that depression precedes pain. According to this view, chronic pain is elicited by depression, which increases pain sensitivity and reduces pain tolerance thresholds (Brown, 1990). Consistent with the previous perspective, the underlying assumption of these ideas is that chronic pain, "without sufficient, identifiable organic pathology" (Turk & Rudy, 1987a, p. 239), is a variant of depressive disease with a genetic and learned basis. This implies that many people are more prone to developing pain as a result of underlying personality dysfunction (Adams & Taylor, 1997; Novy et al., 1995).

A number of studies have sought to demonstrate that depression precedes the development of chronic pain. People with chronic pain investigated include those with chronic mixed site pain, herpes zoster patients, rheumatoid arthritis, migraine and

temporomandibular disorder patients using widely disparate measures of depression and pain. Most of these studies failed to confirm the hypothesis (Fishbain et al., 1997). For example, Kazis et al. (1983) found in 729 RA patients that pain, not depression, was the best predictor of later pain. Gatchel, Polatin and Mayer (1995) found with 421 acute back patients that depression was not an antecedent of chronic pain. Spierings, Sorbi, Haimowitz and Tellegen (1996) studied 20 females who experienced migraine and could not demonstrate that depression was a predictor of subsequent pain. Kubinski et al. (1991) investigated temperomandibular disorder with 80 chronic pain patients and were also not able to demonstrate that pre-treatment depression was a predictor of post-treatment pain.

This view has generated a vast literature about the 'typical' person with chronic pain and the search for a particular personality profile but this "uniformity myth" (Gamsa, 1994b, p.22) has been seriously discredited in the last two decades. There has been an extensive, published debate about this approach, but, to date, criticism outweighs support. This is mainly because the concept lacks validity and methodology often lacks rigor, producing limited empirical evidence (see Von Korff, Resche, & Dworkin, 1993, for more detailed discussion). It would seem more helpful to people with chronic pain to view this theory in terms of the emotional contributions to suffering that it recognizes, rather than its assumption that psychological dysfunction is the cause (Adams & Taylor, 1997).

The *radical operant-behavioural* perspective is thought to have evolved from work by Callie (1913, cited in Turk & Rudy, 1986) and more recently, Rachlin (1985) who contended that pain is detectable mainly from observed "overt manifestations, [or] "pain behaviours" that is, [overt] nonverbal and verbal communications" (Novy et al., 1995, p. 240) of the person. 'Pain behaviours' include bodily posturing such as grimacing, limping and moaning as well as observed medication use-age, inactivity and interaction patterns with others. According to this view, pain behaviours are overt operants (i.e. susceptible to contingencies of reinforcement and shaping) and are thus 'shaped' by the environment of the person with pain. This approach proposes that eventually the pain behaviours become so entrenched that pain or actual sensory physiological experiences become irrelevant (Rachlin, 1985).

Pain behaviours are thought to 'take on a life' of their own, irrespective of the original pain from which they may have evolved.

According to Novy et al. (1995), much of the empirical evidence supporting this approach comes indirectly from outcome results of pain management programs in pain clinics that use behaviour modification techniques. There is a subset of people with chronic pain who respond to this type of treatment. In addition, it is also suggested that some people with chronic pain will modify their behaviour to successfully negotiate a program, because "they have simply learned a new operant response – that of stoicism" (Gamsa, 1994b, p. 25). These ideas need to be treated with caution as one could argue that a successful treatment does not directly provide evidence that a theory is accurate. It has been argued that this approach neglects a person's needs and that outcome measures fail to assess suffering which is considered to always be subjective (Merskey, 1991). Fundamentally, this view has been widely criticized for failure to account for the interrelationships among the sensory-physical, cognitive, behavioural and affective elements of chronic pain (Turk et al., 1983).

The *radical cognitive* perspective, encompassed in work by Ciccone and Grzesiak (1984), derived from Stoic Greek philosophy (Turk et al., 1983). According to this view, pain is a consequence of dysfunctional thinking by people with chronic pain, implying that cognition accounts for the major proportion of the pain experience to the exclusion of other factors. From this perspective, chronic pain results from "a series of mental events that occur between peripheral nociception and behavioral response" (Jerome, 1993, p. 160). According to some writers, some support for this theory has come indirectly from treatment outcomes involving biofeedback (Holroyd, Penzien, Hursey, Tobin, Rogers, Holm, Marcille, Hall, & Chila, 1984) and cognitive therapy (Blanchard & Andrasik, 1985). Rybstein-Blinchik (1979) cites support for this perspective when reporting that rational restructuring, used in a cognitive treatment, was partially instrumental in reducing pain self-reports and pain behaviours. Moreover, this explanation has been criticized as too simplistic, as it is unlikely that cognition alone accounts for successful treatment outcomes (Novy et al., 1995). It is

likely that there are several mechanisms, both psychological and biological, that link chronic pain and psychological dysfunction (Keefe et al., 1992; Rudy et al., 1988).

2.4.2 Criticism of uni-dimensional perspectives

These examples of a uni-dimensional perspective of chronic pain have been criticized for their 'restrictive' nature, as they all have a single focus, either that pain is somatic/body-centred or psychological/mind-centred (Novy et al., 1995). As explanations, they do not account for many chronicled inconsistencies. For example, simply trying to link pain directly with tissue-damage has proven to be inadequate. In many cases, degree of pain is not reported to be consistent with degree of injury (Melzack & Wall, 1982). There have been reported accounts of soldiers, critically injured in battle, who have remained oblivious to their pain and also patients undergoing major surgery who have reported no pain, post-operatively, when it would be expected (Beecher, 1959). In addition, there are countless anecdotal reports of athletes who continue to participate in sporting activities while bearing broken bones or torn ligaments, which do not manifest as pain until the 'action' is over. According to the sensory perspective, these people should complain of severe pain. It has been speculated that the reason they may not is because pain is 'contextual', i.e. the situation supersedes tissue damage and injury (Beecher, 1959).

There is further evidence that a uni-dimensional perspective is inadequate. For example, pain can be disproportionate to severity of injury. Passing a kidney stone involves an area of the body that has relatively few nerves yet the pain experienced is reported, and observed, to be excruciating. Another example is pain that is experienced when a part of the body no longer exists, such as with phantom limb pain. When limbs have been amputated or paralysed, an individual often experiences a sense that the limb is still there in its entirety and can experience intense crippling pain (Loeser & Melzack, 1999; Melzack & Wall, 1982).

A further incongruity is chronic pain without discernable tissue damage. As mentioned, for example, most people with chronic back pain have been reported to

demonstrate no apparent sign of injury, despite agony that may persist for years (Linton, 1994; Morris, 1992). In contrast is the phenomenon of 'congenital insensitivity' to pain where individuals suffer injury but feel no pain. There have been many documented cases of people who are born with this condition and they are often so afflicted they are likely to suffer permanent disfigurement, such as loss of fingers and toes, due to failure to withdraw from a painful stimulus, e.g. an open flame (Melzack & Wall, 1982). Research into families with this abnormality has shown widely diverse results - some cases at biopsy showed abnormality of nerve roots, while others did not. There is another affliction known as Lesch-Niehan disease, a rare and congenital disorder, in which victims bite away parts of their own bodies in a compelling self-destructive urge whereby the usual feelings of pain do not stop the behaviour (Melzack & Wall, 1982). A uni-dimensional view of pain is also unable to explain why some people have objective evidence of physical damage, i.e. x-rays of degenerative spinal changes, and yet do not report pain (Riihimaki, 1991; Turk et al., 1992b).

According to Turk and Rudy (1987a), a uni-dimensional approach implies several erroneous assumptions about the diagnosis and measurement of chronic pain. These include the idea that medical technology is currently capable of identifying the aetiology of chronic pain, that perceived pain can be reliably measured, that normative data exists with which it can be compared and finally, that the extent of 'excessive' pain can be reliably determined. Such a view implies that chronic pain and psychological disturbance cannot occur in the same person or that chronic pain cannot lead to the development of psychological dysfunction (Turk & Rudy, 1986). Gamsa (1994b) has further contended that there is a lack of empirical evidence to support such an approach, and an absence of physical evidence is not just cause to assume that pain is therefore psychologically based. Furthermore, as Sullivan (2000) more recently noted, diagnosing people with chronic pain according to a psychiatry manual such as the DSM is flawed because it pits medical and psychological causes against one another. This is clearly detrimental to the person, both in terms of clear diagnosis or effective therapy. It also perpetuates the mind-body dualistic perspective.

The inconsistencies reported in the literature show clearly that explanations of pain need to take into account more than just the sensory or the psychological aspects of the experience. To that end there has been a revolution in theories and definitions, aimed at addressing the limitation of explanations that focus on a single, often different aspect of chronic pain. Thus, to again draw from the work of Novy et al. (1995), more recent ideas take a 'comprehensive' perspective, which emphasizes the multiple facets of the chronic pain experience, especially psychological. Several of these are addressed in the following section.

2.4.3 Multi-dimensional perspectives

More recent multi-dimensional perspectives of chronic pain have had a major influence on the study, assessment and treatment of chronic pain. According to Novy et al. (1995), they are best described as the approach provided by the *International Association for the Study of Pain (IASP),* the *non-radical operant-behavioural* perspective, *gate control theory* and the *cognitive-behavioural perspective.*

The *International Association for the Study of Pain* is the official global organization developed to study pain and its approach has been used as a working (dynamic) approach rather than as a theory (Novy et al., 1995). This is evident from the changing definitions of pain that the organization has endorsed. Early definitions included the requirement of actual tissue damage whereas more recently, pain is seen as *"....always subjective. Each individual learns the application of the word through experiences related to injury in early life"* (Merskey, 1991, p. 153). In 1999, Loeser and Melzack stated that the IASP endorsed the best definition of pain, describing it as "*an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*" (Merskey & Bogduk, 1994, p. 1607). This clearly incorporates both the sensory and psychological aspects of chronic pain. This approach has had a prevailing influence on contemporary medical treatment and research, although it is not explicit about how the sensory and psychological aspects of pain interrelate. Turk and Rudy (1987a) have

provided some evidence by demonstrating interrelationships between objective medical tests, life interference and psychological distress. Further research is needed to provide more explicit detail of interrelations in order to provide some basis for an empirically testable model of the relationship network.

The *non-radical operant-behavioural* perspective is a more recent and comprehensive version of the radical operant-behavioural perspective previously discussed, because it acknowledges the suffering that occurs as part of the total pain experience (Fordyce, 1978). Pain behaviours are seen as a consequence of suffering and are implicated in the persistent disability of people with chronic pain. This view implies that ignoring 'pain behaviours' and rewarding 'well behaviours' (e.g. increased activity, reduced medication use) should result in a reduction in overt displays of pain (Turk et al., 1992b). 'Well' behaviours do not appear to be compatible with expressions of pain (Gamsa, 1994a).

Empirical support provided by Fordyce (1982) suggests that pain behaviours may be influenced by the environment; i.e. people with chronic pain may be able to avoid onerous tasks because others in the environment respond to the overt signs of pain to which they are exposed. This means that such behaviours may be selectively reinforced and maintained even when nociceptive stimulation has ceased (Turk et al., 1992b). For example, Kerns and colleagues (Kerns, Southwick, Giller, Haythornthwaite, Jacob, & Rosenberg, 1991; Kerns & Turk, 1984) found level of pain report to be directly related to perceived positive attention from significant others. Flor, Kerns and Turk (1987) and Flor, Turk and Rudy (1989) reported that when overt pain behaviours were ignored there was an associated decrease in reported pain severity. However, other research does not support this approach. For example, Summers, Rapoff, Varghese, Porter and Palmer (1991) found that when spouses ignored people with chronic pain, there was an increase in reported pain severity.

Other critics have implied that this approach ignores the needs of people in chronic pain and that outcome measures do not account for actual suffering (Merskey, 1985). Even if pain behaviours are reduced in a clinic or laboratory setting, it does not automatically follow that this will continue to occur in the long-term when people with chronic pain have

returned to the 'uncontrolled' environments of their normal lives (Gamsa, 1994a). The home environment is probably the most important in terms of reinforcement contingencies that help maintain the pain problem (Pearce & McDonald, 1998).

Outcome results from pain treatment programs may be misleading because the percentage of people with chronic pain, especially back pain, who are referred to pain clinic programs is quite small (Elliott et al., 1999; Linton, 1994). In addition, evidence supporting operant treatment has been flawed by methodological inadequacies such as lack of controls and inadequate measures (Turk et al., 1992b).

It is not clear what the mechanisms of change are in operant programmes. Turk (1996) suggested that an operant approach is problematic partly because the validity of the pain behaviour construct is questionable. More recently, Sharp (2001) suggests that "there is a logical flaw in the assumption that the operant model is supported simply because behavioural treatment has an effect on patients' behaviour" (p. 789). Specifically, Sharp cautions that many studies which support an operant approach by claiming that pain behaviours are reduced as a result of environmental modification fail to acknowledge to what extent patients' interpretations of this environmental modification influence the reduction of the pain behaviours.

It has been argued that this type of treatment is not considered suitable for some people, e.g. those experiencing high levels of identifiable, persistent pain (Fordyce, Roberts, & Sternbach, 1985). How efficacious it is for those with unidentifiable, persistent pain has yet to be fully determined.

Gate control theory (Melzack & Casey, 1968; Melzack & Wall, 1965) is one of the most popular explanations for the chronic pain experience (Loeser & Melzack, 1999; Novy et al., 1995). This is despite the fact that it assumes that pain is preceded by nociceptive input, which may not be present in some cases of chronic pain (Linton, 1994). The theory primarily defines pain in terms of its sensory or nociceptive aspects but acknowledges the role of psychological processes on pain perception and response. The basic premise is that there is a neural 'gate' in the spine that is linked to mechanisms in the brain. The 'gate' effects

both sensory input and response on a physiological as well as psychological level. Any neural mechanism within the spinal cord that functions as a 'gate', does so by increasing or decreasing the flow of nerve impulses from the peripheral fibres to the central nervous system.

The 'gate' influences the degree of sensory transmission determined by activity in specific nerve fibres as well as descending influences which come from the brain. Information passing through the 'gate' needs to exceed a critical level before there can be activation of those neural areas, which interpret the experience of pain and response. This suggests that modulation of input occurs via some neural mechanism before pain is experienced. It is considered that both sensory inputs and psychological factors can facilitate or inhibit the signals of injury (see Melzack & Wall, 1965, for more detail about the mechanisms of the gate).

According to this theory, pain is a dynamic process that can be exacerbated or inhibited depending upon the activity of multiple interrelated facets. Despite some criticism, the theory has evolved into a robust explanation for the puzzling phenomenon of pain. It has stimulated research into the basic science of pain mechanisms and inspired various clinical applications to control and manage pain. These include neuro-physiologically based treatments, pharmacological advances, behavioural treatments and interventions which focus on modifying attention and perception that are integral to the pain experience (Novy et al., 1995). It also contributed to the cognitive-behavioural perspective, yet to be discussed.

Loeser and Melzack (1999; Melzack, 1999) suggested that despite its considerable contribution to the study of pain, the gate control theory still cannot fully explain some types of chronic pain, such as phantom limb pain. They have suggested the theory be expanded to include a greater understanding of the brain, which must hold the answers to the enigma of chronic pain. This was based on the proposal of a neural network theory in which a 'neuromatrix', a pattern-generating mechanism existing in the brain, is genetically based and subject to modification by sensory input (Melzack, 1990). According to this notion, the neuromatrix generates characteristic neurosignature patterns of nerve impulses producing a

multi-dimensional experience that is interpreted as pain. These ideas are introduced merely to acknowledge that the study of pain is a dynamic work in progress. Further discussion of this version of the gate control model is beyond the scope of the present thesis, but it is an innovative approach to the debate and augurs well for the future of pain research.

(i) A cognitive-behavioural perspective

The *cognitive-behavioural*[2] perspective derives from a melding of cognitive and social learning theories, behaviour modification research (Bandura, 1977; Beck, 1976; Ellis, 1962; Mahoney, 1974; Meichenbaum, 1977; Neisser, 1976) and Melzack and Wall's (1965) gate control theory. There has been a vast amount written about this perspective and currently there are many versions of cognitive-behavioural theory in the literature (Sharp, 2001). As with the other comprehensive perspectives, this approach also endorses the idea that sensory-physical, cognitive, behavioural, and affective facets are all important in understanding the pain experience.

According to Novy et al. (1995), the cognitive-behavioural perspective can be interpreted as an interactive, transactional or synergistic model, the basic premise of which is 'reciprocally determinism', that is, the person's perspective "interacts reciprocally with emotional factors, sensory phenomena, and behavioral responses" (Turk & Rudy, 1986, p. 762). From this perspective, pain is not just "nociception....[or] processing of stimuli that are defined as related to the stimulation of nociceptors and capable of being experienced as pain" (Turk et al., 1992b, p. 384). Instead, pain is defined as a perceptual phenomenon, which consists of "the integration and modulation of [...] afferent and efferent processes" (Turk et al., 1992b, p. 384). That is, all these factors interrelate and pain is considered to be more than just peripheral stimulation. Furthermore, unlike the gate control theory, the cognitive-behavioural perspective allows for cases where there is no detectable tissue damage or sensory stimulation.

[2] In the literature, the term 'cognitive-behavioural' is used to variously describe perspectives, theories and models and these terms are often used interchangeably (Turk & Rudy, 1992; Jensen et al., 1999). Some researchers also refer to variations of cognitive-behavioural theory. A similar approach will be taken here, in keeping with the literature.

Thus, people with chronic pain "are viewed as active processors of information" (Turk & Rudy, 1992, p. 103). This implies that the individual constantly appraises and reinterprets the personal pain experience in order to define and redefine it, thus the theory reflects the dynamic nature of chronic pain (Bandura, 1977; Lazarus & Folkman, 1984). This cognitive appraisal or 'cognitive experience' is comprised of the beliefs, feelings, behaviours and emotions that a person has while experiencing chronic pain (Turk et al., 1983). This theory also allows for the powerful influence that the 'meaning' or context of the situation has, in determining the nature of the pain experience (Beecher, 1959). The cognitive facet is not emphasized, to the exclusion of other facets, as one element is not necessarily more important than any other. The general understanding about this perspective is that these different aspects can pre-dominate at different times and under different conditions (Novy et al., 1995).

At a more fundamental level, it is the *interpretation* of the pain experience that affects both behaviour and emotions, rather than simply the sensory characteristics of the experience. For instance, when pain is interpreted as a sign of continued physical trauma or tissue damage, rather than just the result of minor trauma which is healing, psychological suffering is likely to be far more extensive (Turk & Rudy, 1992). Furthermore, this psychological suffering and dysfunction may be exacerbated in those pain cases where there is no clear sign of injury (Pearce & McDonald, 1998). Thus, the cognitive-behavioural approach argues that the development and maintenance of depression in chronic pain depends on how the person thinks about or *cognitively* appraises all aspects of the chronic pain experience including severity, persistence, perceived control, behaviours, responses from other people and the general interference in daily living. More specifically, it is maladaptive cognitive activity that makes a key contribution to the exacerbation of all aspects of the pain experience and so effects adjustment in the long-term (Turk & Rudy, 1992).

There are various ways that negative cognitive appraisal may contribute to the development of depression in those who have chronic pain. For example, people with

chronic pain may be prone to *cognitive distortion* (Beck, 1967, 1976) or a tendency to think negatively about oneself and the world. When faced with adversity, negative cognitive schema that tend to influence thought processing, are activated (Banks & Kerns, 1996). Individuals may distort perceptions of their situation and engage in 'logical errors', which help to perpetuate the negative thought patterns. Such people tend to over-generalize, take things personally, see the world 'in black and white', and/or usually imagine the 'worse-case' scenario with regard to their condition (Banks & Kerns, 1996). This negative pattern of thinking may result in dysphoric mood and subsequently, depression (Beck, 1967, 1976). Lefebvre (1981) first reported an association between cognitive distortion and depression in people with chronic back pain. Early research suggested that depressed people do demonstrate greater cognitive distortion than non-depressed people (Hammen, 1978; Hammen & Krantz, 1976; Krantz & Hammen, 1979; Lefebvre, 1981). More recent findings have also found evidence for the implication of cognitive distortion in those who experience chronic back pain and rheumatoid arthritis (Keefe, Brown, Wallston, & Caldwell, 1989; Maxwell et al., 1998; Smith, Christensen, Peck, & Ward, 1994), as well as people with mixed types of chronic pain (Geisser, Robinson, Keefe, & Weiner, 1994).

It has also been thought that depression might develop as a function of *learned helplessness* (Abramson, Seligman, & Teasdale, 1978; Seligman, 1975). The severity of painful illness is significantly related to stress (Taylor & Curran, 1985) and some report that prolonged pain is the most stressful, uncontrollable and unpredictable part of their lives (Banks & Kerns, 1996; Turk & Rudy, 1992; Turner, Clancy, & Vitaliano, 1987). During stressful times a person is likely to appraise the situation "as taxing or exceeding his or her resources and endangering his or her well-being" (Lazarus & Folkman, 1984, p. 19). Such a person may therefore use different beliefs than when not stressed, beliefs that incorporate helplessness (Lazarus & Folkman, 1984). For example, the belief "that effective solutions are not available to eliminate or reduce the source of stress" (Turk & Rudy, 1992, p. 108) may predominate. This is likely because consequences of chronic pain conditions, such as family and marital problems, unemployment and social isolation are significantly stressful

(Jensen et al., 1991), and may exacerbate the pain condition. People with chronic back pain often have to cope with unremitting pain despite using a diverse range of, often unsuccessful, palliatives and 'cures' (Turk & Holzman, 1986a; Turk et al., 1992b). Over time, their situation can appear hopeless and as lack of control increases, so too does helplessness and subsequently, depressed mood.

This way of believing is thought to generalize to future events and form a basis for the development of depression. Furthermore, all individuals have a particular 'attributional' style or way of inference in daily life, which may be negative, positive or somewhere in between. Such 'causal attributions' may mediate between perceived lack of control and depression (Banks & Kerns, 1996). For example, a person more prone to develop depression may make *internal* (It's my fault that I have a back injury), *stable* (I've always been physically weak) and *global* (My life is never going to improve) attributions. This negative attributional style may come to eventually dominate the usual way of thinking for such a person. Subsequently, the feelings of helplessness are likely to lead to dysphoric mood, lack of motivation and other general life deficits associated with depression and chronic pain (Banks & Kerns, 1996).

Depression, while in chronic pain, may also result from a decrease in positive *behavioural reinforcement*, which is contingent upon particular responses (Fordyce, 1976; Lewinsohn, 1974), because maladaptive appraisals of reinforcement contingencies contribute to ongoing distress (Turk et al., 1992b). That is, when someone else takes over a task, provides sympathy or prescribes stronger drugs, the person may interpret this as confirmation that they are helpless and have little control. Inactivity increases and then subsequent attempts to become more mobile are followed by more pain. This confirms the fear that activity causes pain and previously rewarding activities become associated with pain and therefore punishment (Banks & Kerns, 1996). Activities may then be limited and this diminishes opportunities for reward or positive reinforcement. This applies to the work, sports and social activities that the person might normally do (Banks & Kerns, 1996). Such a cognitive interpretation then fuels fear and so drives the person deeper into the

hopelessness and helplessness phase. The fear and avoidance behaviour, which is commonly seen in people with chronic pain, attests to the interactive, integrative cognitive-behavioural nature of the condition (Turk et al., 1992b).

However, according to Banks and Kerns (1996) these three models, cognitive distortion, learned helplessness and the behavioural, do not adequately explain depression among chronic pain patients, which can be high, compared to other medical populations. These models imply that a predisposition to develop depression in the presence of illness should be equal across chronically ill people, not just those with chronic pain. These models may overemphasize vulnerability and not take into account the magnitude of the stressor.

In the case of chronic pain, Banks and Kerns (1996) proposed that a diathesis-stress framework was more applicable. Diathesis-stress theories have been mentioned in the literature since the 1960s but Banks and Kerns' model was the first of such models to be used to explain chronic pain. The basis of the model is that some people may have a characteristic or diathesis (psychological or biological), which predisposes them to illness. Any stressor, either environmental or life event, which threatens one's mental or physical health beyond a level which could be coped with, combined with a diathesis may precipitate the development of a disorder.

Pincus and Williams (1999) also discussed a diathesis model as one of a range of cognitive models. They further described a complex multistage model of cognition, disability and affect which focuses more on disability as a stressor, as opposed to pain. Further discussion of these ideas is beyond the present scope, but they are introduced here to demonstrate the directions that theory conceptualisation has taken, and continues to take, in the study of chronic pain.

The criticisms of Banks and Kerns (1996) have been borne out by research in the last decade or so, about the efficacy of a cognitive-behavioural perspective as a way of explaining the chronic pain experience. Adams and Taylor (1997) referred to the cognitive-behavioural model as "a unifying theory, in which the behavioural model is expanded to incorporate cognition and affect within behaviour therapy" (p. 86). In 1999, Jensen et al.

maintained the importance of beliefs and coping behaviours in adjustment to chronic pain and that there continued to be considerable empirical support for a cognitive-behavioural model of chronic pain. This confirms the idea that beliefs held by those with chronic pain should be able to predict adjustment or lack of it. Jensen et al. (1999) were also able to provide evidence that self-report continues to be a valid measure of beliefs and that this provided evidence for a cognitive-behavioural model. They suggested that further research into chronic pain, especially among those not actively seeking treatment at pain clinics, continues to be warranted.

An indication that pain research is a dynamic field is further exemplified by work on 'fear-avoidance' models of chronic pain in recent years. It is not possible to address this literature in great detail as it is beyond the present scope, however a brief overview is presented here. As mentioned earlier, some ideas about pain are not new, including a theorized link between fear and pain, which was implied even in the ancient works of Aristotle (refer to Eysenck, 1997 and France et al., 1988 for more detail). 'Fear-avoidance' refers to a tendency to avoid physical activity due to fear and is cited as a key mechanism in the chronic pain experience. Medical research has long demonstrated the link between tissue damage, physiological responses, fear and anxiety, although only in relatively recent times have the relationships between fear, pain and avoidance behaviour been investigated. Lethem, Slade, Troup and Bentley (1983) are acknowledged as pioneers of the first 'fear-avoidance' model of pain.

There has been considerable empirical support for such models with respect to pain. For example, using structural equation modeling, Asmundson and Taylor (1996) demonstrated that there was a direct relationship between fear of pain and escape and avoidance behaviours in chronic pain patients. Consistent with this, Crombez, Vlaeyen, Heuts and Lysens (1999) and Waddell, Newton, Henderson, Somerville and Main (1993) have also shown that fear of pain is more likely to be related to functional disability than is pain severity. Vlaeyen, Kole-Snijders, Rotteveel, Ruesink, and Heuts (1995c) and more recently, Crombez et al. (1999) demonstrated that fear of movement/reinjury or

'kinesiophobia' was greater among those whose pain began with a sudden traumatic incident compared to those in whom chronic pain developed more slowly. Despite much research in this area, most of it has lacked integration, a fact Vlaeyen and Linton attempted to redress in a 2000 review. This research partly evolved from the difficulty of teasing out "the mechanisms by which acute problems become chronic" (Vlaeyen & Linton, 2000, p. 317).

Both operant and cognitive frameworks have been applied to fear-avoidance models by notable writers such as Fordyce (1976) and Philips (1987a). Vlaeyen and Linton (2000) describe a most recent 'activity' avoidance model based on classical conditioning and operant principles. This model explains an 'activity' avoidance response through conditioning by direct experience, vicarious learning or observation. According to this model, a threatening and pain eliciting situation produces a conditioned response including physiological reaction and fear which leads to avoidance. This avoidance behaviour is subsequently reinforced when the painful stimulus is reduced.

Vlaeyen and Linton (2000) also discuss a second model, the 'fear' avoidance model, which builds on the activity avoidance model by introducing a more cognitive component. According to this model, disability can result from pain-related fear by several paths. For example, a tendency to 'catastrophize' may act as an antecedent of pain-related fear. The fear itself can take the form of avoidance and escape behaviour that encourages a decrease in functional ability. Anticipatory avoidance of pain can perpetuate because opportunities to counteract incorrect expectancies and beliefs are reduced. Subsequently, avoiding activity can lead to muscular and systemic atrophy that, in turn, is likely to perpetuate the pain condition. Avoidance also means that potentially positive reinforcement opportunities are lost leading to increased cognitive dysfunction, including feelings of depression (Philips, 1987).

According to this model, those in chronic pain may also be hyper-vigilant to sensory perceptions, thus rendering them less cognitively aware of able to perform other necessary tasks, such as coping. Pain-related fear may encourage the body to react more intensely

and more often to perceived threats on a physiological level, thus increasing stress both physically and mentally. This area of research is more complicated than stated here as this discussion was intended only to indicate the directions that pain research is taking. A more detailed discussion is beyond the present scope (refer to Crombez, Vervaet, Lysens, Eelen, & Baeyerns, 1998; Crombez et al., 1999; Vlaeyen, Haazen, Schuerman, Kole-Snijders, & van Eck, 1995a; Vlaeyen, Kole-Snijders, Boeren, & van Eck, 1995b, for more detail).

A further indication of the dynamic nature of pain research is exemplified by work by Sharp (2001), who has offered a reformulated cognitive-behavioural theory. Sharp notes that much of the cognitive-behavioural research has been largely based on operant principles, and while not denying the importance of this, stresses the need for more attention to be paid to the cognitive aspects of the pain experience. This reformulated model borrows from anxiety research (for example, work by Salkovskis and colleagues, Salkovskis, 1991; Salkovskis, Clark, & Gelder, 1996), the basic premise being that chronic pain problems arise from cognitive reactions to pain. Those disabled by chronic pain differ from those who do not largely as a consequence of differences in appraisals and interpretations of pain. The reformulated model is a comprehensive one that proposes that pain can be maintained even in the absence of original nociceptive input by the interplay between multiple factors. These include factors such as beliefs, culture, learning history, avoidance and safety behaviours, iatrogenic factors and environmental contingencies. Sharp (2001) cites support for this model with research that has found that fear of pain "is a better predictor of avoidance behaviour than is pain severity " (p. 795).

More recently, writers such as Turk and Okifuji (2002) and Campbell, Clauw and Keefe (2003) have promoted a biopsychosocial perspective of chronic pain and depression. Turk and Okifuji (2002) refer to a model, which "views illness as a dynamic and reciprocal interaction between biological, psychological and sociocultural variables that shapes the person's response to pain" (p. 679). While strictly speaking, this perspective appears more extensive than a cognitive-behavioural perspective, it is included here to indicate the direction that research is taking. Only an overview is given here as more detailed discussion

is beyond the scope of the present research as it post-dates the research plan, data collection and analysis.

The biopsychosocial model assumes that there has been some type of nociceptive action that has preceded the condition, in the form of physical pathology or 'physical changes' in bodily structures. The reaction of the person with chronic pain to the condition is based not only on a personal appraisal of pain, especially the meaning or context of the pain, but also in the usual belief system of the person involved. Reactions taken may then range from ignoring the pain and maintaining usual functioning through to adoption of the sick role with complete withdrawal from all activity and social interaction. The responses from significant others are then likely to shape the reaction to pain in a positive or negative sense – the former a healthy response and the latter, a sick response. Main and Williams (2002) have also stressed the importance of considering a biopsychosocial model of pain to improve the understanding of the chronic condition. Again, this perspective assumes noniceptive origin, which is not always true for chronic pain cases (Linton, 1994).

According to Turk and Okifuji (2002), some of the best predictors of development of chronic pain from acute injury have been shown to be psychological factors such as maladaptive attitudes and beliefs, lack of social support, increased emotional reactivity, job dissatisfaction, substance abuse, compensation status, prevalence of pain behaviours and psychiatric diagnosis (refer to Gatchel & Epker, 1999; Turk, 1997, cited in Turk & Okifuji, 2002). However, despite continued research about these issues, Turk and Okifuji (2002) have warned that methodological limitations still continue to plague pain research so that findings need to be treated with caution. There is still a tendency to rely on cross-sectional research and chronic pain without previous identified injury continues to be very puzzling.

It is clear that many, new and interesting ideas regarding chronic pain have emerged over the last decade or so. It is impossible to fully do justice to the thousands of words written on the subject in this thesis. In 1995, Novy et al. indicated that the multiple interrelationships of comprehensive theories and models of pain had not been completely tested and understood. Even today, despite the plethora of advancement in theory

development, there appears to have been a dearth of testable hypotheses offered for testing. Many of the more recent models and theories are largely untested and, as mentioned, post-date the current research, in terms of planning and design. There is considerable evidence that a cognitive-behavioural perspective is very useful as an explanation for the chronic pain experience. Although one cannot imply that a successful treatment provides evidence of an accurate theory, much of the research with cognitive-behavioural treatments and chronic pain has indicated that "multiple and diverse facets are interrelated" (Novy, Nelson, Francis, & Turk, 1995, p. 243). This mainly derives from cognitive-behavioural treatment (CBT) outcome results in multidisciplinary pain units.

There are many and varied types of cognitive-behavioural approaches to pain management but they generally involve providing people with strategies to help them gain a sense of mastery and competency over pain by modifying the sensory, cognitive, behavioural and affective aspects of the pain experience (Novy et al., 1995). For example, various techniques such as relaxation, biofeedback and hypnosis have all been found to be successful in terms of reducing feelings of helplessness and negative thoughts (Fernandez & Turk, 1989; Turner & Chapman, 1982). Newton and Barbaree (1987) assessed cognitive activity in a group of people with chronic headache and found that after treatment, there was an increase in positive appraisal and problem-solving coping techniques.

Successful treatment programs have also helped to change beliefs about pain, pain severity and coping competency, as well as changing behaviour related to pain (Dolce, Crocker, & Doleys, 1986a; Turk & Rudy, 1986; Turner & Clancy, 1988). For example, Nicholas et al. (1992) reported more improvement in terms of self-efficacy, functional abilities and coping strategies with a combined treatment, including CBT. Tota-Faucette, Gil, Williams, Keefe and Veeraindar (1993) and Jensen, Nygren, Gamberale, Goldie and Westerholm (1994) have reported that chronic pain patients reported increased control and improved functioning while reporting less severe pain and reduced catastrophizing, after treatment.

Williams, Nicholas, Richardson, Pither, Justins, Chamberlain et al. (1993) conducted one of the few longitudinal evaluations of a CBT program with 212 people with chronic pain and found improvements in pain intensity, depression and quality of life were maintained at a 6-month follow-up. It was concluded that this treatment can improve daily functioning even if more traditional treatment has been unsuccessful. Richardson, Richardson, Williams, Featherstone and Harding (1994) reported that CBT was effective in improving work performance and ability to return to work in people with chronic pain. In 1996, the National Institute of Health (NIH, 1996) of the United States, reported that CBT was among therapies which were effective in reducing distress and pain levels in people with chronic pain.

In 1999, Morley, Eccleston and Williams (1999) conducted a systematic review and meta-analysis of randomised controlled trials using CBT for people with chronic pain. They reviewed 25 studies, including nine that targeted low back pain, comparing CBT with waiting list controls and alternative treatments. It was concluded that those treatments based on CBT were more effective in producing changes in several dimensions including pain, mood, cognitive coping and appraisal.

More recently, Fishbain (2000) reviewed meta-analyses of non-surgical pain treatment studies to ascertain treatment efficacy. Several approaches, including CBT, were indicated as effective when combined with other techniques as part of a multi-disciplinary approach. Although there is not complete consensus about which treatments are the most effective for alleviating chronic pain, non-surgical treatments, including CBT, tend to be among the best in terms of positive, long-term results and supportive evidence (Fishbain, 2000). Loeser (1991, cited in Morley et al., 1999) has also argued that there is more evidence to support CBT as an effective treatment for chronic pain than there is for medical or physical treatments. These results support the theory that chronic pain is a multi-faceted experience involving cognitive appraisal processes.

There is a body of work that shows support for the cognitive-behavioural approach by specifically focusing on the 'profiles' of people with chronic pain, assessed by the MPI (Kerns et al., 1985). As mentioned, this questionnaire was founded on cognitive-behavioural

theory. The chronic pain profile refers to the pattern of psychological responses that people may make to chronic pain, indicating the extent of the impact that chronic pain has on daily functioning. Identifying such profiles can be useful in determining those most at risk and appropriate interventions (Turk & Rudy, 1988). Turk and Rudy (1986; Turk & Rudy, 1987b) proposed the profile classification system to allow for comparison of observations and results. The basic premise of this was to define, clarify and synthesize diverse variables which have been hypothesized to measure the experience of chronic pain i.e. the impact on "physical, psychological, social and behavioural functioning" (Turk & Rudy, 1988, p. 233). One advantage of profiling is that it can reliably discriminate between people who suffer pain in the same part of the body, e.g. the back, but who respond to pain quite differently (Rudy, 1987).

Turk and Rudy, (1988) used cluster analytical techniques to identify three distinct profiles, which represent the extent to which adjustment to pain occurs along cognitive-behavioural dimensions; 1) *dysfunctional*, 2) *interpersonally distressed* and 3) *adaptive copers*. Those classed as dysfunctional perceive their pain as high and greatly interfering with their lives. They also report higher psychological dysfunction, affective distress and lower perceived control over their lives, than the other two groups. Finally, they report lower general activity. Those classified as interpersonally distressed do not perceive that they have much support from their social networks. Adaptive copers report lower levels of pain severity, perceived interference and affective distress and higher levels of life control and general activity. They cope better than the other two groups and are most likely to adjust positively to the ongoing stress of chronic pain. They may also minimize the degree of impact that pain has on their lives.

Several studies by Turk and colleagues (Rudy et al., 1989; Turk et al., 1995; Turk & Rudy, 1990) and others (Jamison, Rudy, Penzien, & Mosley, 1994) have replicated the findings. These studies have consistently found that about 40% of clinical patients can be classified as dysfunctional, while about 30% will be classified as either interpersonally distressed or as adaptive copers. Altogether, most studies have reported that about 92% to

95% appear to be classifiable under the MPI system (Turk & Rudy, 1988, 1990), although more recently, McCracken, Spertus, Janeck, Sinclair and Wetzel (1999) were only able to report that 67.4% of 190 patients could be classified into the 3 main sub-types.

These profiles identified by Turk and colleagues are considered to be generally consistent for groups of patients suffering diverse pain problems (Zaza, Reyno & Moulin, 2000), although, the system may not work well for other pain groups. For example, Zaza et al. (2000) found that only 60% of 107 people with cancer pain could be classified into the three major profiles. A two-profile classification system was proposed as more appropriate for this group reflecting different social support requirements from people with other types of pain. Such findings imply that this classification system requires further examination with respect to other groups of people with pain, including those from the general community.

Rudy (1987) also identified three non-prototypic profiles. These are the 'hybrid', the 'anomalous' and the 'unanalysable' profiles. For a designation of 'hybrid', the MPI scores may "represent aspects of more than one of the prototypic profiles" (Rudy, 1987, p. 35), an occurrence found in about 4% of clinical patients tested with the MPI. 'Anomalous' may refer to nonsensical or extremely unusual scores with respect to any established theory. This might occur for several reasons, e.g., random responding, difficulty reading or under or over-exaggeration of symptoms. Finally, 'unanalysable' could result from too much missing data so the program is unable to provide enough statistics. Classification has been reported as difficult if there is too much data missing (Bernstein, Jaremko, & Hinkley, 1995; Okifuji, Turk, & Eveleigh, 1999b; Riley, Zawacki, Robinson, & Geisser, 1999). Okifuji et al. (1999b) found that greater clarification of the definition for 'significant other' improved the rate of classification because it allowed greater completion of the questionnaire.

Although cognitive-behavioural theory is well respected, there has been little published research that demonstrates its main proposition of 'reciprocal determinism' (Novy et al., 1995). In more recent years, there has been less mention of 'reciprocal determinism' in the literature, although it is often implied. Specific testing of models consistent with the theory has proven to be a daunting task. The fundamental nature of the theory implies that

a model must be inherently non-linear (Turk & Rudy, 1986) because "each facet affects and is affected by every other one" (Novy et al., 1995, p. 244). So while it is described in multi-directional terms, there has been no testing, at least published, of it in this fashion. More specifically, at the time of writing, a search of medical and psychological literature databases, failed to reveal specific testing of a multi-directional, reciprocal cognitive-behavioural model that clearly addresses the association between chronic pain and depression.

(ii) The cognitive-behavioural mediation model

Despite a plethora of recent theory development, there has also been limited testing of a linear, uni-directional model which is consistent with a cognitive-behavioural approach to the study of chronic pain and depression. The unique study by Rudy et al. (1988) claimed to be the first empirical demonstration of cognitive-behavioural theory. Rudy et al. (1988) called this model the 'cognitive-behavioural mediation' model. Although more than a decade has passed since this first demonstration, it has still been actively cited in recent literature as a worthy model with which to explain the chronic pain experience (Catley, 2000; Hurwitz et al., 2003; Maxwell et al., 1998; Verma & Gallagher, 2000).

In Rudy's study, it was hypothesized that "the direct link between pain and depression should be small and minimally useful in accounting for the relationship between the two syndromes" (p. 130). Results confirmed the theory that depression was a consequence of, or secondary reaction to, chronic pain in a uni-directional model with 100 mixed chronic pain patients. Therefore, according to this model, chronic pain severity is not enough to account for the development of depression. The statistical technique of causal modelling was used to show that an initial moderate correlation between chronic pain and depression was reduced to almost zero once cognitive variables were accounted for. It was further reported that pain together with lack of self-control and perceived interference accounted for more than 68% of the variance in depressive symptomatology. In addition, 50% of the participants in this study recorded clinical levels of depressive symptomatology.

Thus, chronic pain and depression were mediated by cognitive variables, in this case described as 'perceived interference' and 'perceived life control'. It was concluded that "depression is a function of a sustained reduction in instrumental activities and a concomitant decline in important social rewards ...[and] a decline in perceptions of control over reinforcement contingencies and personal mastery" (p. 130). Rudy et al. (1988) claimed that such a model is in keeping with the cognitive-behavioural theory. A cognitive-behavioural mediation model consistent with that model is presented in graphic form in Fig. 2.1.

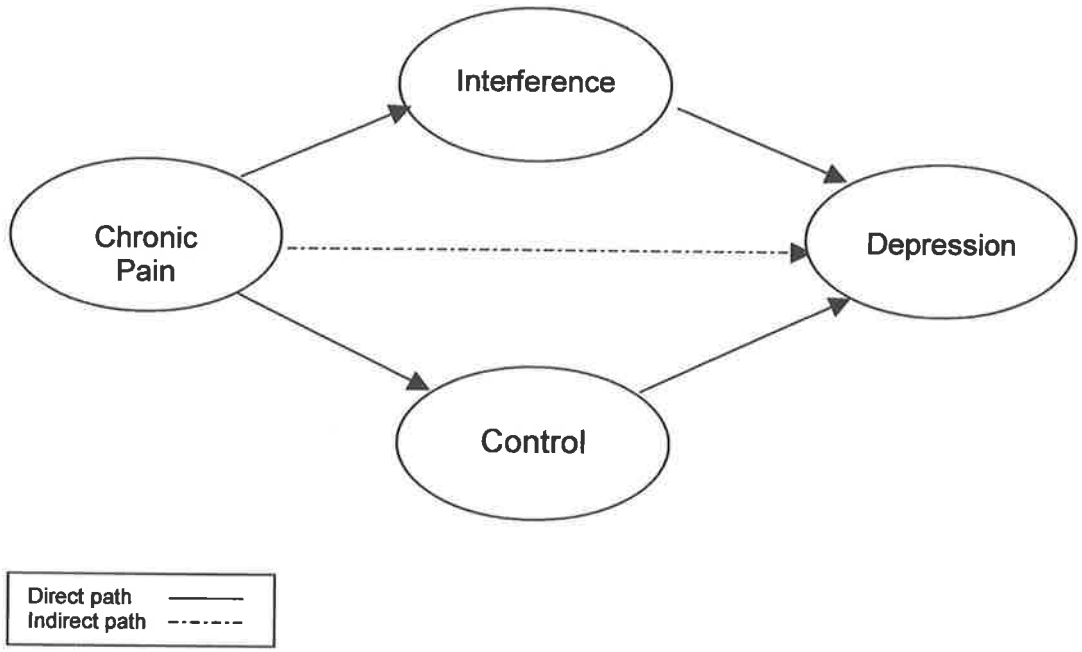

Figure 2.1. Cognitive-behavioural mediation model of chronic pain and depression (after Rudy et al., 1988).

This model indicates that while chronic pain and depression are related, most of the influence of pain on depression occurs via the mediating cognitive variables, in this case, perceived interference and perceived life control.

While this study was an important step in demonstrating the potential of cognitive-behavioural theory, the statistical technique used to demonstrate statistical significance may be contentious. Rudy et al. (1988) used structural equation modelling (SEM) to test their theory. At the time, SEM, made popular by Jöreskog, (1978), was a relatively new technique and was considered a powerful method of statistical analysis. It was considered especially useful to test psychological models such as those implied by theories like cognitive-behavioural theory. Critics have since suggested that while SEM was proposed as a means of addressing such difficulties, there have been many misapplications of it in research due "to the allure of [its] ... powerful analytic techniques" (Pedhazur & Schmelkin, 1991, p. 698). It is not necessarily suitable for all types or sets of data. In the case of the Rudy et al. (1988) study, there is a suggestion that the sample size may not have been adequate to provide the statistical reliability that was claimed (Mueller, 1996). Other subsequent research, e.g. Turk et al. (1995) and Maxwell et al. (1998), while demonstrating support for the theory, used different statistical methodology that mitigates comparisons. The study by Brown (1990) used SEM and showed a relationship between pain and depression, but did not address the idea of mediating variables.

Several studies have supported a model of mediation and, in so doing, support the hypothesis that depression is a consequence rather than an antecedent of chronic pain (Fishbain et al., 1997). For example, Haythornthwaite, Sieber and Kerns (1991) investigated 69 heterogeneous chronic pain patients to determine differences between depressed and non-depressed patients. The WHYMPI was used in this study and it was determined that the depressed patients reported significantly more pain, interference and less activity than the other group. This suggested that depression only developed in those who found the experience of pain to be a great interference in routine activities.

Mobily, Herr, Rizzo and Large (1993) confirmed the Rudy et al. (1988) findings with 128 chronic pain patients. Structural equation modelling confirmed that perceived life interference and control mediated the relationship between chronic pain and depression. The Turk et al. (1995) study also demonstrated, with 100 chronic pain patients, that cognitive

appraisal variables mediated chronic pain and depression in young participants (defined as those aged less than 69 years) but not in older persons. The Maxwell et al. (1998) study demonstrated, in a sample of 74 people with chronic back pain, that cognitive distortion along with control and perceived interference mediated the link between pain and depression. Most recently, Hurwitz et al. (2003) suggested that their results were "....consistent with the [.....] cognitive-behavioural mediation hypothes[is]" (p. 468).

In October, 2003, a search of commonly used internet search engines, such as Medline, Psyclit, Expanded Academic Index and Web of Science revealed that research into the aptness of cognitive mediation models continues, although mostly in spheres other than the chronic pain area. For example, Chang, Sanna and Yang (2003) found that affect mediates outcome expectancies and psychological adjustment in culturally diverse groups. In addition, Papworth and James (2003) found that creativity and affect were mediated by appraisal among 104 graduate students.

The afore-mentioned findings about the use of cognitive-behavioural theory and chronic pain provide a basis for further testing of such a model with a range of cognitive variables and indicate that further research into the association between chronic pain and depression within a cognitive-behavioural framework is warranted. Cognitive-behavioural theory has evolved into a robust approach to study the association between chronic pain and depression, particularly when the origin of the pain is unknown or ambiguous. This approach, originating mainly from the psychiatric and psychology disciplines, has allowed the operationalisation of subjective and intangible experiences such as 'pain' and 'depression' for specific use in treatment programs.

These are psychological factors that are, by their very nature, inaccessible and we rely on measures that have been devised through considerable research to establish reliability and validity. Survey or questionnaire research is the most commonly used strategy to obtain measures of psychological factors. There is also considerable overlap between psychological concepts. For example, terms such as 'locus of control' and 'self-efficacy' may be used in similar ways to describe ways of coping (Jensen et al., 1991).

While psychological variables are subjective, considerable research has ensured that they are measured and quantified as much as possible. The cognitive-behavioural approach generally measures and quantifies these key constructs to allow for comparisons and assessments. Cognitive-behavioural theory allows for these measurements of phenomena experienced by the person in chronic pain. While such measurements use self-report, these are the closest measures of the pain experience available for investigation. This theory is consistent with a psychological approach to investigating and explaining phenomena that use pen and paper measures.

Although there are methodological issues that need clarification, this theory is deemed suitable as a framework for the present research. It appears to be the best way to attempt to explain why some people in pain also develop depression when others do not and the best way to test relationships between pain and depression, with the available resources. It provides a sound foundation to explore the psychological and affective components of chronic pain in a community sample as well as a framework to test models of chronic pain and depression.

2.4 Summary of the history of pain research

Chronic pain is considered to be one of the most common and debilitating health conditions. Depression is often a typical consequence of chronic pain and therefore when these conditions occur co-morbidly there is increased risk of psychological dysfunction. Depression in the person with chronic back pain indicates 'failure to adjust'. The evidence so far indicates that depression is more common in people with chronic pain, especially chronic back pain, than it is in those who do not suffer chronic pain. However, assessing chronic pain and depression can be confounded by overlap of symptoms between the two conditions. In addition, most research has centred on clinical patients and the nature and extent of depression in non-clinical populations is far from clear.

In terms of treatment and research, the study of chronic pain has primarily been, until recent decades, the province of the medical professions. As a consequence, assessment

and treatment have been generally conducted as if chronic pain related either to bodily tissue damage or a dysfunctional mind. In cases where there is no detectable aetiology, this has been offered as an indication that the condition is mainly of the mind and perpetuated the belief that the psyche and the body are separate entities. In the last few decades this approach has been found too restrictive, as it does not effectively explain such phenomena as 'phantom limb pain' or chronic back pain. Supportive empirical evidence for such a viewpoint is strictly limited.

In response to this limitation, many more comprehensive theories and models have been proposed. Cognitive-behavioural theory, which is a broad term for a range of related theories and models, has developed a strong foundation of supportive evidence although there are methodological concerns about the supporting data. The main premise of this theory is that chronic pain consists of several facets, which affect, and are in turn affected by, each other. These facets, identified as fundamental to the chronic pain experience, include sensory, cognitive, behavioural and affective aspects. This perspective has encouraged research into the role of cognitive appraisal in the development of depression. However, such a theory is difficult to test given its inherent nature of reciprocal determinism. In more recent years, theories have tended to concentrate more on testing interrelatedness of variables, as opposed to testing 'reciprocal determinism'.

In keeping with this, a uni-directional variation, the cognitive-behavioural mediation model, has been proposed as a useful mechanism to explain the relationship between chronic pain severity and depression. In particular, this model suggests that negative cognitive appraisal resulting from chronic pain predisposes the person with chronic pain to developing depression. A considerable amount of research has supported this idea.

In Chapter 3 the literature, related to cognitive appraisal and its role in the chronic pain experience, will be reviewed. In addition, certain demographic characteristics have been shown to influence response to persistent pain. The literature findings relating to this issue will also be reviewed.

CHAPTER THREE

REVIEW OF THE LITERATURE: DEMOGRAPHIC CHARACTERISTICS AND PSYCHOLOGICAL FACTORS ASSOCIATED WITH CHRONIC PAIN AND DEPRESSION

3.1 Introduction

There is a vast literature about the contribution that cognitive appraisal makes to the level of adjustment and associated dysfunction in those who suffer with chronic back pain (Jensen et al., 1991; Turk & Rudy, 1986). Much of this has not been examined consistently within theoretical frameworks (Gamsa, 1994a, 1994b), particularly from a cognitive-behavioural perspective. There is a range of factors associated with chronic pain and depression which are fundamental to cognitive appraisal and which have been shown to contribute to poor adjustment in the person with chronic pain. There are demographic characteristics that have been found to differentiate response to chronic pain and development of depression, including gender, age, marital status, education and employment status. The psychological factors that have been most identified as relating to both chronic pain and depression include beliefs people have about their pain experience. They are consistent with the cognitive-behavioural, and therefore psychological, approach to understanding chronic pain. This chapter reviews the literature about how demographic characteristics may influence response to pain and the role played by beliefs in the relationship with depression.

3.2 Demographic characteristics, chronic pain and depression

The following section reviews research findings about how demographic characteristics may influence response to chronic pain, most particularly how this might be related to the development of depression. Traditionally, girls report higher pain levels and

more fear and anxiety associated with pain than do boys, who display more anger when in pain (McGrath, 1990). Girls also report more headaches and abdominal pain with greater frequency and intensity (Lamberg, 1998) although these findings may not clearly reflect gender differences but rather a social bias. Generally, in most cultures, boys are conditioned to "be brave and not show their pain while girls are allowed to be emotional" (Lamberg, 1998, p. 1035). In addition, Loeser (1989) has pointed out that the suffering associated with pain, of which depression is a part, is a subjective experience that is influenced by prior experience and culture. This suggests that women, due to cultural influences, are more likely to suffer when in pain.

With respect to adults, chronic pain and demographic factors, most evidence is derived from a few studies and is mixed. Previous research by Novy, Nelson, Averill and Berry (1996) specifically addressed gender differences in an investigation of 245 people with heterogenous chronic pain complaints, because of a dearth in such research. Their investigation found some gender differences in responses to specific items of the BDI but overall, there were no differences in total depression scores. Similarly, Haythornthwaite et al. (1991) found no gender differences (or educational or marital status differences) between depressed and non-depressed patients in a sample of 69 heterogenous chronic pain patients. These patients were obtained from "an interdisciplinary pain rehabilitation program" (p. 178) and were diagnosed with depression via a structured psychiatric interview and psychiatrically determined criteria.

Alternative research findings indicate that females in pain may be more at risk. For example, Marbach and Lund (1981) found that chronic pain and depression were significantly correlated in a sample of mainly females with facial pain. In later work with more males with back and facial pain, no such relationship was demonstrated (Marbach, Richlin, & Lipton, 1983). Timmermans and Sternbach (1976) were also unable to find a significant relationship between chronic pain and depression in a sample of mainly males. White and Harth (1999) report the middle aged, single female with chronic pain with limited education is most at risk of developing depression. Consistent with this, Turk and Okifuji

(1999) found, with 428 chronic (back and leg) pain patients, that female patients were significantly more likely to be depressed than males. Diagnosis of depression was determined from scores on the CES-D and a clinical interview. However, there were no gender differences in reported pain severity. In addition, adaptation to pain, assessed by response to the MPI, did not reveal that either gender adapted better than the other to chronic pain.

Haley et al. (1985) also found that females were more likely than males to be depressed as a direct result of pain. In an heterogenous sample of 63 (36 females; 27 males) chronic pain patients, males were more likely to be depressed because pain interfered with their daily activities, rather than as a direct result of the pain. In line with an operant approach to explaining pain, it was concluded that this occurred because pain prevented males from conducting their daily activities, which previously provided positive reinforcement. Conversely, increased pain severity for women was likely to be linked to greater suffering and therefore increased likelihood of depression, also an indication of suffering (Haley et al., 1985).

Jensen et al. (1994) describe a study to determine gender differences in response to pain, among 121 clinical patients in Sweden (71 females; 50 males) with neck, shoulder and back pain. Although Sweden has one of the highest levels of women working outside the home in the world, women are still the primary home carers. This high work-load is thought to increase the psychological risks to females with chronic pain. In addition, 75% of Swedes seeking health care for chronic spinal pain are women, and there are more women than men affected in the long term by chronic pain with respect to sickness and disability (Lagerlöf, 1993). Findings showed that women used less effective coping skills when dealing with work related chronic spinal pain than did men. More specifically, women with lower education levels showed less effective coping abilities but no such relationship was found for men. The research concluded that using similar rehabilitation techniques for both males and females does not allow for gender differences in terms of coping strategies. This study did not investigate depression specifically however, although the findings imply that gender

differences in adjustment to, and management of, chronic pain need to be considered in non-clinical or community groups.

With respect to age differences in depressed people with chronic pain, the results are mixed. Kramlinger, Swanson and Maruta (1983) reported no age differences, while Haythornthwaite et al. (1991) found depressed patients were significantly younger (M=44.3 years, SD=11.4) than non-depressed patients (M=55 years, SD=13.8). This latter study concluded that people who develop chronic pain early in life (i.e. in their 30s) are more likely to suffer depression than those who suffer chronic pain once they are past 40 years of age. There were no significant differences in marital status or years of education between the depressed and non-depressed patients. In addition, 80% of the sample was unemployed and 51% of them were receiving workers' compensation payments. Averill et al. (1996) found in a sample of 254 pain clinic attendees that younger females (aged 20 to 40 years) were significantly more depressed than were younger males. Moreover, older males (aged over 40 years) were significantly more depressed than similarly aged women. This study revealed that employment status was the main predictor of depression, followed by education and marital status. However, the relationship between employment and depression is considered to be complex. Kramlinger et al. (1983) found that there was no significant relationship between depression and employment status. There has been limited research into how these factors interrelate and conclusions are unclear.

There has been limited research into the extent to which non-clinical depressed and non-depressed people in pain may be differentiated in terms of demographic characteristics. In the Magni et al. (1994) study mentioned earlier, of the original 3,023 persons surveyed, only 274 were found to report pain at both base-line and follow-up surveys. The measure used to assess depression was the CES-D and two cut-off points were applied, the standard cut-off of 16 and a higher cut-off of 20, to flag *high risk* major depression. Magni et al. (1994) reported that the main predictors of depression with the low cut-off score were chronic pain, female gender, poor education, and unemployment. At the higher cut-off point

(20), the predictive power of these variables was weaker and at follow-up, only unemployment predicted depression.

In sum, the response to chronic pain appears to depend to some degree on demographic characteristics. Although conclusions are far from clear, some general points can be made. To date, the evidence suggests that women are more likely than males to suffer depression as a result of chronic pain. In those people with chronic pain who do develop depression, men are more likely to become depressed because pain prevents them from conducting their usual daily activities. In contrast, women with chronic pain are likely to become depressed as a function of suffering associated with increased pain severity rather than because pain interferes with daily functioning. Depression is more likely to develop in single, middle-aged people with chronic pain who are poorly educated. However, most of these findings relate to clinical patients and response to chronic pain among non-clinical people in terms of demographic characteristics warrants further investigation.

3.3 Beliefs, chronic pain and depression

For the purpose of the present research, *beliefs* refer to several terms such as 1) *interpretations* about the overall pain condition; 2) *thoughts,* or *'self-statements'* while actually experiencing pain; 3) *cognitive reactions* or *appraisals* about the impact that pain has on general living (Turk & Rudy, 1986). Therefore, consistent with the psychological constructs found to be correlates of chronic pain and depression, literature related to the following beliefs will be reviewed:

1) beliefs related to perceptions of interference in daily living and activity levels;
2) beliefs about degree of control over pain;
3) beliefs about perceptions of social support;
4) beliefs about confidence or self-efficacy related to pain;
5) strong intensity emotions related to pain such as anger and anxiety.

Due to the complicated and overlapping nature of this literature, evidence for different constructs may be provided by the same studies.

3.3.1 Beliefs related to perceptions of interference in daily living and activity levels

One measure of 'adjustment' to chronic pain is the extent to which the condition is perceived to interfere in one's daily activities. Rudy et al. (1988) define interference as the extent to which people with chronic pain "feel pain has affected their ability to participate in social....recreational ... vocational ... family and domestic activities (and) the amount of satisfaction that they derive from these activities" (p. 131). It is similarly defined by Maxwell et al. (1998) as "interference in instrumental activities" (p. 132). It is also treated by some researchers as the *flip-side* of activity levels (Burns, Johnson, Devine, Mahoney, & Pawl, 1998). That is, high interference implies low activity. Throughout this thesis 'interference' may be used interchangeably with 'perceived interference', consistent with its use in the literature, and is used to refer to a general disruption to daily living. Many people with chronic pain exist in a 'holding pattern', not prepared to live a full and satisfactory life until their pain has gone. In terms of chronic pain, this is often an unreasonable expectation, given the long-term nature of the condition. As Turk and Okifjui (2002) have stated, chronic pain should probably be viewed "as a lifelong disease" (p. 687). Moreover, those who accept that their pain is unlikely to be temporary, and adjust their lives accordingly, are much less likely to suffer long-term circular psychological distress (Nicholas, 1994).

While some researchers have measured how much pain interferes with functioning, others have assessed activity levels to infer how much pain interferes with daily functioning or how activity has been changed by the chronic condition (Haley et al., 1985). This could include the extent to which pain has prevented routine functioning in normal household, outdoor and social activities. If individuals have become much less active in all these general areas, it is assumed that the chronic pain condition is having a deleterious effect on their lives and hindering adjustment. In some research, the terms are used interchangeably. Various methods have been used to measure interference and activity levels, including

questionnaire surveys (Burns et al., 1998; Findley, Kerns, Weinberg, & Rosenberg, 1998; Maxwell et al., 1998; Turk et al., 1995) and pain diaries to record hours spent standing, sitting or lying down (Haley et al., 1985). Findings associated with beliefs about how much chronic pain interferes with and disrupts daily life are consistent. Beliefs about interference and activity have been investigated as they relate to chronic pain and in relation to control over pain, confidence in dealing with pain and anger, general profiles of people with chronic pain and gender differences. They have also been investigated as mediators between chronic pain and depression.

Those people who perceive that chronic pain is significantly disruptive tend to report more depression and feel they have less control over daily life. Previously mentioned research by Rudy et al. (1988), Turk et al. (1995) and Maxwell et al. (1998) all arrived at similar conclusions about the relationship between interference and control over pain, despite using different measures and methodologies. That is, these variables mediated the effects of chronic pain on depression such that those patients who reported high perceived interference, also felt they had less control over their lives and reported more depression. In the related research by Turk and colleagues (Rudy et al., 1989; Turk et al., 1995; Turk & Rudy, 1990), on 'profiles' of people with chronic pain, similar conclusions were reached. 'Dysfunctional' people with chronic pain reported that pain interfered with daily life much more than other people with chronic pain. In work by Williamson and Schulz (1995), similar conclusions were reached in terms of interference.

Lin (1998) suggested that greater perceived pain intensity and interference in the long-term are related to low self-efficacy and more likely to be linked with less effective coping strategies. The implication of this is that adjustment, which is a long-term process, is likely to be impeded. Anderson, Dowds, Pelletz, Edwards, and Peeters-Asourian's (1995) work investigating self-efficacy beliefs in chronic pain patients, reached similar conclusions. Those who reported low self-efficacy reported greater interference, negative mood, depression, hopelessness and pain severity. These findings suggest that the degree to which chronic pain interferes in daily life is reflected in an erosion of confidence or self-

efficacy in ability to deal with the condition. Believing that an activity will lead to pain is likely to undermine the confidence one has to actually perform the behaviour (Council, Ahern, Follick, & Kline, 1988). The consequence of this is that the person is less likely to engage in the behaviour. This affects both self-efficacy and activity levels in a cyclical pattern. If self-efficacy is low to begin with and activities are avoided, pain is seen or believed to be less severe as a result of decreased mobility. This confirms to the person that he/she made the right decision, general activity level falls and muscles atrophy. Thus when an activity is undertaken and pain results, this confirms that the activity should have been avoided in the first place and so on.

There has been some limited research demonstrating gender differences and perceived interference. For example, the work by Haley et al. (1985) and Timmermans and Sternbach (1976) showed that males were more affected than females, by the interference of pain in terms of developing depression. Gender differences in perceived interference have also been implicated in studies of chronic pain and anger. Kerns et al. (1994), in a study with 142 chronic pain patients, reported that those who *suppressed* their anger were more likely to report higher pain severity and less activity than those who *expressed* their anger. More specifically, Burns et al. (1996), in a study with 127 heterogenous chronic pain patients, reported that males who suppressed angry feelings reported more interference, more severe pain and more depression. Females who expressed their angry feelings reported less activity and more severe pain as well. However, in a second study with 101 heterogenous patients, those findings for women were not replicated. Overall, Burns et al. (1998) concluded that those who failed to manage their anger effectively were poorly adjusted in terms of pain severity and activity levels.

If males do find pain to be more disruptive in terms of the development of depression, this may be explained by differences in role identity. For example, traditionally, in most societies women tend to be the *kin-keepers* (Turner, B., 1982), while most men are considered the *breadwinners* and tend to see themselves as responsible for supporting their families (Girard, 1993). Males are also more likely to be judged on 'work' performance,

which could result in job loss, changes in income etc. Mothers are not "fired [.....] for their child rearing performance" (Girard, 1993, p. 556). The implication of this is that male identity tends to be intrinsically tied to the chronology of work and therefore involves expected milestones (Settersten Jr. & Hägestad, 1996). A middle-aged, chronically ill male is less likely to be able to conduct the daily chores as expected and is likely to feel that his identity has been threatened more than a female who is stricken but who can still be at home for her family, albeit with restrictions. This is likely to make males more vulnerable to psychological ill-health than females (Girard, 1993), and thus make them perceive chronic pain as a greater interference in life.

In sum, these results have consistently demonstrated that increased levels of perceived interference and disrupted activity levels are linked to psychological dysfunction in people with chronic pain. These findings serve as a basis for investigating the extent to which chronic pain interferes with activity levels in the community chronic back pain sample chosen for this research. In particular, the findings on gender differences are informative for the present research. People with chronic pain who have not psychologically adjusted to chronic pain are more likely to report high interference, decreased activity and increased depression. This is likely to be particular true of males with chronic pain who suppress angry feelings and females who express anger when in pain.

3.3.2 Beliefs about control over pain

The perception of how much control one has over one's health, irrespective of the reality of that perception, is considered to be one of the key determinants not only of "health-related behaviour, but, ultimately, of whether [one stays] healthy or [becomes] ill" (Wallston, 1992, p. 184). 'Control' over pain, per se, does not have a well-accepted definition, despite being the subject of research for many years (Litt, 1988b). Turk and Rudy (1990) argue that a 'perceived' lack of control implies that adjustment and therefore, coping is poor. Litt (1988b) advises that such a viewpoint is general enough to include various types of control,

as well as implying that control need only be perceived, and not necessarily exist, to be effective in changing the meaning of the circumstances.

Perceptions of control, or control appraisals, are reported to play a key role in adapting or adjusting to serious persistent medical conditions, such as chronic pain (Affleck, Tennen, Pfeiffer, & Fifield, 1987). Attempting to balance both the necessity to surrender treatment to powerful others when appropriate, and the necessity to maintain some sense of mastery over one's life, can create unusual psychological conflict for the chronically ill (Reid, 1984). For example, if pain is very severe and a person feels they have little control, surrendering control to some powerful other (or to a deity) may be adaptive. Furthermore, maintaining some personal control over some aspects of the disease, even when it is not possible to control the entire course of the disease, can also be adaptive (see Burish, Carey, Wallston, Stein, Jamison, & Lyles, 1984; Miller, 1980; Reid, 1984; Rothbaum, Weisz, & Snyder, 1982, for more detail). Researchers have identified several different pain-related appraisals or beliefs about 'control' that are related to adjustment to chronic pain (Jensen et al., 1991). In the present research two of these, 1) perceived control over pain, and 2) health locus of control, will be addressed.

(i) Perceived control

Perceived control has been defined as "belief that one has at one's disposal a response that can influence the aversiveness of an event" (Thompson, 1981, p. 89). In the present thesis, 'perceived control' refers to an appraisal process that considers the stressful situation i.e. the pain condition, in a general sense only. As previously stated, control is not clearly defined but implies that a person experiences some power over various aspects of the chronic pain experience from symptoms to functioning. This cognitive construct is considered to be a 'situation-specific' construct unlike health locus of control (Litt, 1988b). Perceived control is expected to be relevant in specific situations, especially aversive ones such as those that occur to people in chronic pain, rather than generally. This belief

encompasses more than just how health is valued but also the perception of the degree to which certain health-related behaviours are likely to lead to better health (Wallston, 1992).

Most of the research findings about perceived control have been elicited from comparison of pre- and post-treatment assessments in pain patients and tend to be consistent. People with chronic pain identified as having greater control, reported less depression and less interference in daily functioning (Keefe & Williams, 1990; Maxwell et al., 1998; Rudy et al., 1988; Strong, Ashton, Cramond, & Chant, 1990). Affleck et al. (1987) and Jensen and Karoly (1991) found that increased control was related to better global adjustment. People with chronic pain who have greater perceived control also take a more active involvement in their own care as well as reporting that their pain is less severe (Philips, 1987). Jensen, Karoly and Huger (1987) found that male patients with high personal control over pain were more likely to engage in stress reducing techniques and exercise. Consistent with this, Philips (1987) reported that those who believe that they have less control over their condition are more likely to engage in *avoidance behaviour.* This means they tend to avoid tasks or behaviours that they think will cause them more pain, and in so doing miss out on opportunities to test themselves physically. This is likely to maintain physical disabilities and atrophied muscles, which in turn helps to maintain the pain.

In addition, the mediating effect of control on aversive stressors has been investigated (Averill, 1973). In the Rudy et al. (1988) study, internal health locus of control and perceived control were treated as a unified construct, called 'perceived life control'. Patients who endorsed a higher internal locus of control perceived that their pain severity was significantly less than those with a lower internalization. 'Perceived life control' accounted for a large proportion of the relationship between pain and depressed mood. That is, those in greater pain reported reduced feelings of control and subsequently, greater depressed mood.

This supports Wallston's (1992) premise that locus of control and perceived control are aspects of the same construct. Furthermore, in the Rudy study, a partial correlation between pain and depression, controlling for control, was reported as 0.06 (i.e.

approximately 0) suggesting that control was a main mediating variable in the relationship. In similar studies, Turk et al. (1995) and Maxwell et al. (1998) also found that control acted as a key mediator between chronic pain and psychological dysfunction. These studies will be referred to again in relation to other variables that are considered to be mediators as well. A wide variety of measures have been used to assess control making it difficult to compare findings directly (Jensen et al., 1991).

(ii) Health locus of control

Health locus of control (HLC: Wallston & Wallston, 1978) is a global or dispositional construct derived from the locus of control construct (Rotter, 1954) and based on Social Learning Theory. The HLC construct measures three distinct dimensions of control known as 1) internality, 2) powerful others externality and 3) chance externality. These are defined as beliefs that 1) one has control over one's own health or 2) control rests with some powerful other person/s, or 3) outcomes rely on chance or luck. People with high *internal* control are more likely to believe that being healthy is related to their own efforts and to engage in self-help type behaviours (Wallston & Wallston, 1978). People with chronic pain with high *external* control tend to think that it does not matter what they do, because other people, or fate, are responsible for their well-being. These people are more likely to believe that going to the doctor regularly and/or taking prescribed medication is the best way to manage their health, or that there is nothing they themselves can do.

Previous research findings related to locus of control, pain and psychological health have a similar theme, albeit with some inconsistencies. For example, researchers may have used specific study instruments rather than validated measures. In pre-treatment clinical research, Skevington (1983), used a specifically designed locus of control scale in a small study with 25 back pain patients and found that those with high internal sense of control were less likely to be depressed. It was concluded that those who had high internal locus of control but who could also relinquish control to (powerful) others if necessary, protected themselves against personal failure and were more likely to adjust to chronic illness.

However, those with a high *external* locus of control, manifesting as belief in powerful others, also did not suffer significant pain or depression (Skevington, 1983). Crisson and Keefe (1988) reported that among 62 chronic pain patients, those who had a high internal locus of control as well as belief in powerful others, demonstrated little psychological distress. In contrast to Skevington (1983), they found that those believing in 'chance' were more likely to suffer greater psychological distress and helplessness.

With respect to treatment effects, Fisher and Johnston (1998) found in a small sample of 54 chronic pain patients that locus of control and control cognitions did not change from pre- to post-treatment, despite clearly significant improvements in disability, pain severity and emotional distress. While emotional distress was found to mediate changes in pain and disability, control cognitions did not. It is not clear why this occurred but these authors concluded that may have been due to the particular instrument chosen for their study. They used the Multi-dimensional Health Locus of Control scale (MHLC: Wallston & Wallston, 1978). Why this could be a problem is unclear as they reported good reliability and validity for the scale and reported that the version used was specifically modified for pain patients.

Other research is not consistent with some of these findings. For example, those with high external locus of control with regard to their health, suffered more depressive symptomatology, more helplessness, and less satisfaction with their lives (Laborde & Powers, 1985; Nicassio, Wallston, Callahan, Herbert, & Pincus, 1985; Skevington, 1983). Nicassio and colleagues used 219 RA patients as their pain sample, while Laborde and Powers (1985) studied 160 osteo-arthritis (OA) patients. Affleck et al. (1987) also investigated these relationships in a sample of 92 RA patients and reported that belief in the self or high internal locus of control was related to increased positive mood. In contrast to Skevington (1983), Affleck et al. (1987) found that those who believed that some powerful other controlled the pain were more likely to experience negative mood. Chronic pain from these arthritis conditions cannot strictly be compared to chronic back pain, because generally arthritis has a much clearer aetiology. Arthritis research has an extensive

literature of its own and a discussion of it is beyond the scope of this thesis. These findings are reported here because persistent pain is a common denominator of both conditions and so research may be illuminating to some degree.

In sum, people with chronic pain who perceive that they have good control over their pain are less likely to suffer psychological dysfunction. In addition, those who appear to cope better with their pain and adapt or recover faster, tend to have a higher internal locus of control than those who cope less well. They also tend to be more satisfied with life. In addition, most of the available evidence is derived from mixed pain groups seeking treatment or undergoing treatment. There is limited information about how the different dimensions of control interrelate in those who are not involved in regular pain clinic treatment programs. Based on clinical findings it would be expected that those with greater perceived control and high internal locus of control will be less depressed and report less severe pain.

3.3.3 Beliefs about perceived social support and chronic pain

Social support has been referred to as the emotional aid provided by the home, work and social environments in which a person exists (Schiaffino & Revenson, 1995). Social support is a key element of the social environment in which cognitive appraisals about chronic illness are made. Exploring the nature of social support is therefore useful in terms of understanding adjustment to chronic pain (Schiaffino & Revenson, 1995). In the following discussion about social support, the term 'social network' refers to those likely to be closest to the person with chronic pain. This may be the immediate family, spouse or partner or if people live alone, may refer to those with whom they share their lives in the closest way.

Given the dynamic nature of chronic pain it is probable that the social environment or network affects, and so in turn, is affected by the chronic pain condition. A person with chronic pain is likely to experience considerable upheaval and changes in roles and responsibilities in lifestyle, employment and friendship networks. This, in turn, may have drastic effects on the mood of the person in pain, thus affecting the family or social network.

Again how the family copes with these changes can have extensive effects on the course of the illness (Flor et al., 1989).

DeLoach and Greer (1981) argued that adjusting to disability is influenced by the social expectations of significant others. This is particularly important for the chronic pain condition, which is generally acquired, as opposed to chronic pain from congenital disabilities. An acquired condition is much harder to accept and adjust to (Li & Moore, 1998). If a significant other is alienated in some way from the person in pain, social support may be reduced or withdrawn thus contributing to failure to adjust in the individual. Previous research has indicated that supportive social networks make a considerable difference in terms of healthy recovery from illness (Wallston, Smith, King, Forsberg, Wallston, & Nagy, 1983). Likewise, social support can protect someone "from the potentially harmful effects of stress" (Trief, Camrike Jr., & Drudge, 1995, p. 227), such as chronic pain.

The role of social support in chronic pain has been researched from several perspectives including social support, the operant conditioning, and the cognitive-behavioural frameworks (see Flor et al., 1989; Turk, Kerns, & Rosenberg, 1992a). According to the first model, social support is beneficial because either people are provided with positive experiences consistently (i.e. positive social reinforcement) or because social support acts as a 'buffer' to the effects of stress (Cohen & Wills, 1985). The basic premise of the social support model is that people in chronic pain with supportive social networks report less pain and tend to cope and adjust better (Brown, Wallston, & Nicassio, 1989; Meichenbaum & Turk, 1987). For example, Brown et al. (1989) found that social support was related to less depression in chronic pain. Trief et al. (1995) also concluded, in a study with 70 people with chronic back pain that those who lived in helpful, supportive environments were much less likely to suffer depression than those who existed in angry, argumentative and conflict-ridden environments. Given that a great deal of adjustment is often required by the families of people with chronic pain, rigid and inflexible family units are less conducive to healthy adjustment in the individual. Schwartz and Kraft (1999) made

similar conclusions with people with multiple sclerosis: those living with less support and more conflict fared less well psychologically.

In contrast to the social support model, an operant conditioning approach promotes the view that the social environment plays a major role in the reinforcement and maintenance of the chronic condition (Flor et al., 1989). That is, the more attentive and solicitous the spouse, the more pain and disability are likely to be experienced by the person in pain (Turk et al., 1992a). Spouses or partners are in a powerfully intimate position to reinforce overt pain behaviours such as grimacing and moaning, which reflect suffering. While these behaviours serve a possible protective function in the acute pain phase, it is unclear what their purpose is once the condition becomes chronic. A spouse is more likely to provide attention and sympathy, which is contingent upon an individual's expressions of pain. Likewise, a spouse may fail to reinforce 'well' behaviours (i.e. those incompatible with expressions of pain) and more likely to relieve the person in pain of unwanted chores (Flor et al., 1989), which can also lead to dependency. The operant view is therefore one which proposes that attention is only positive if it reinforces 'well' behaviours but it is negative if it supports 'pain' behaviours and is likely to result in increased pain report by people in chronic pain (Turk et al., 1992a).

The operant model has been supported with previous research, however most evidence relates to support and its relationship to pain severity, rather than depression. Block, Kremer and Gaylor (1980) found that patients reported more pain when observed by solicitous spouses than when observed by others who were not part of their normal social network. Romano, Turner, Friedman, Bulcroft, Jensen and Hops (1991) found in their study of people in chronic pain, their spouses and a simulated household task experiment, that there was a relationship between solicitous responding by spouses and increased pain report from people in chronic pain. Gil, Keefe, Crisson and Van Dalfsen (1987) found a positive correlation between satisfaction with social support networks and a greater frequency of observed pain behaviours. Consistent with this, Faucett and Levine (1991)

found that when displays of suffering were ignored, there was an associated reduction in reported pain and disability.

Other research appears at odds with the operant view and supportive of the social support model. For example, Jamison and Virts (1990) found that high levels of support were related to less pain report. Manne and Zautra (1989) reported that pain people in chronic pain existing in supportive environments coped and adjusted well to their condition. Likewise, Summers et al. (1991) found that patients reported increased pain severity when their spouses ignored their pain. Kramarczuk (1990) also found a negative correlation between social support and pain severity report in a study involving Australian and Polish people with chronic pain.

While social support is vital to recovery, it appears that the *type* of support one receives is crucial. Revenson and colleagues (Revenson, 1993; Revenson, Schiaffino, Majerovitz & Gibofsky, 1991) refer to 'positive' support (encouraging, doing favours or listening) and 'negative' support (critical, harassing or giving unwanted advice). Positive social support is integral to successful adjustment to chronic pain and disability (Li & Moore, 1998; Thoits, 1986; Wortman & Conway, 1985). In contrast, negative social support is thought to have deleterious effects such as increasing pain report and helplessness (Revenson et al., 1991). Most of the mentioned studies however, did not necessarily define support in 'negative' or 'positive' terms.

Social support is fundamentally related to how people in chronic pain themselves respond to the pain. If initially, they cognitively appraise the condition as negative or harmful but receive encouragement, they are more likely to adjust. Furthermore, if their initial negative response is later confirmed by significant others, this is likely to reinforce their worst fears and they are less likely to adjust (Schiaffino & Revenson, 1995). For example, Revenson et al. (1991) found with RA patients, that most people in chronic pain complained about significant others who thought they were being helpful, but who did not necessarily listen to them. This is typical of the 'double-edged sword' of social support (Revenson et al., 1991).

The social support and operant conditioning models contradict one another (Turk et al., 1992a). While the former views attention to the person in pain as beneficial, the latter implies the opposite. That is, more attention may encourage increased pain and depression and is only beneficial if it encourages the person in pain to adopt behaviours which are not conducive to the 'pain' self, i.e. productive behaviour. Proponents of this latter model indicate that inappropriate displays of suffering should be ignored in order for them to decrease in frequency (Radojevic, Nicassio, & Weisman, 1992). Even if people with chronic pain say that they are happy with the way their social network supports them, they may be unwittingly reinforced to perpetuate 'pain' behaviours, including reporting greater pain.

According to a cognitive-behavioural view, these two models fail to account for the cognitive appraisal people with chronic pain make about their condition, their coping ability and their social network (Turk et al., 1992a). The social support model does not inform about the possibility of the social network being maladaptive. That is, an environment that encourages dependency or which is perceived to be unhelpful even if significant others think they are being helpful. The operant approach fails to take into account the role of cognitions and suffering. In addition, research carried out from these perspectives, has used mainly small and/or male patient samples or failed to examine gender effects (Turk et al., 1992a).

The cognitive-behavioural perspective augments these approaches by incorporating maladaptive cognitive appraisals, self-efficacy and social support, as well as reinforcement contingencies, into the chronic pain experience (Turk et al., 1992a). How a person in pain interprets support is crucial to considering whether it is positive or negative, helpful or harmful (Turk, 1979). Turk et al. (1992a) maintain that the cognitive-behavioural approach can augment an operant approach because the person in pain's perception of spousal response affects not only pain expression, but also affective distress. If support is viewed as helpful and constructive, reports of pain severity may be lessened. Conversely, if people with chronic pain perceive the social network as unhelpful or critical, or that pain is trivialized, emotional distress is likely to rise.

Research into the effects of support on pain severity and depression support the cognitive-behavioural theory. Turk et al. (1992a) examined responses from 148 married chronic (unspecified) pain patients and their spouses. Solicitous responses were significantly related to increased pain report but only if patients were satisfied with the relationship. Negative responses were more related to an increase in emotional distress. This suggests that spouses might reinforce pain, which tends to support the operant view. These findings confirm earlier work by Kerns et al. (1991) that reported positive support, in the form of solicitous responses, was related to increased pain reports, but only if the spousal relationship was perceived as satisfactory by the person in pain.

Research findings with arthritis patients are not fully consistent with a cognitive-behavioural perspective. Brown et al. (1989) found that those patients who were satisfied with their social networks suffered less depression even when reporting severe pain. However, Schiaffino and Revenson (1995) found that social support, positive or negative, did not significantly affect depression levels in 64 people with RA at either baseline or 18 months later. Perceiving that the condition was a challenge at onset was not related to later depression. Those who saw their illness as a greater challenge, however, and had more positive spousal support at onset were more depressed at follow-up than those who reported less spousal influence. In contrast, when appraisal was for low challenge and positive spousal support was high, depression was reduced. This could be due to the fact that if the patient is optimistic but unrealistic, spousal encouragement may be disappointing in the long term (Turk et al., 1992a). Criticism from the spouse may confirm the reality, which is also depressing. In the Schiaffino and Revenson (1995) research, most participants were female suggesting that there may have been considerable traditional role reversal, which could contribute to the depression experienced by those in pain. This could also cause considerable frustration on the part of the spouse (Schiaffino & Revenson, 1995).

In sum, research within the operant and social support paradigms has provided opposing explanations for the role of social support in the chronic pain experience. Evidence supporting both positions has been described but there are inconsistencies.

Social support has been found not to directly influence changes in depression from pre- to post-assessment in people with chronic pain (Schiaffino & Revenson, 1995). Furthermore, it has been proposed as a mediator between chronic pain and depression (Turk et al., 1992a). Increased pain report has been found to be related to both negative spousal support (Summers et al., 1991), and positive support (Turk et al., 1992a). This was also found to be dependent upon whether the person in pain was happy with their spousal relationship or not. The cognitive-behavioural approach augments the other theories by endorsing the view that the extent to which social support increases pain report is dependent upon whether the social support is perceived as a help or a hindrance by the person in pain (Revenson et al., 1991; Turk, 1979). Given the controversy and convoluted nature of these findings about social support and chronic pain, the present study will attempt to determine how social networks relate to the psychological functioning of the person with chronic back pain from the wider community.

3.3.4 Self-efficacy beliefs about chronic pain

Self-efficacy is defined as the confidence one has in one's ability "to execute given levels of performance and to exercise control over events" (Bandura, 1977, p. 563). A person's sense of confidence is influenced by past performance experiences as well as how much one ascribes success to chance or skill (Sherer & Adams, 1983). According to Bandura (1977) behaviour is influenced by two types of expectancies: *self-efficacy expectancies* or belief that one can successfully perform a behaviour needed to produce a specific outcome, and *outcome expectancies* or the belief that a certain behaviour will result in that outcome. Self-efficacy expectancies differ from person to person and determine the activities people will engage in, how much effort is made, "and the length of time persevered in the face of adversity" (Shelton, 1990, p. 987). Research suggests that self-efficacy is a stronger predictor of behaviour than outcome expectancies (Bandura, 1977).

There has been considerable research into the role of self-efficacy in various health-related behaviours such as smoking cessation (DiClemente, 1981), weight loss (Weinberg,

Hughes, Critelli, England, & Jackson, 1984) and exercise (Ewart, Barr, Reese, & DeBusk, 1983). Self-efficacy is considered an important cognitive variable in terms of adjustment to chronic medical syndromes, such as chronic pain (Arnstein et al., 1999; Bandura, O'Leary, Taylor, Gauthier, & Gossard, 1987; Nicholas, 1994). It is further thought to play a pivotal role in a cognitive-behavioural model of chronic pain (Lin, 1998; Rudy et al., 1988; Turk & Rudy, 1992). Although the self-efficacy construct is relatively young in historical terms, it has generated a considerable literature in the last two decades (Litt, 1988b). However, this literature is somewhat confusing in that different types of self-efficacy have been identified. In addition, there have been few valid and reliable instruments developed to measure the different types of self-efficacy. The following discussion outlines these issues.

According to Bandura (1977), past performance experiences, physiological arousal, vicarious experiences and verbal persuasion can influence one's self-efficacy beliefs. Of these, *performance experiences* and *physiological arousal* provide key sources of information about self-efficacy for people in pain (Turk & Rudy, 1992). Performance accomplishments, i.e. actually completing a task, are thought to have the most influence on feelings and perceptions of mastery because they provide the most information about true competence. This refers to any ordinary task that a person in pain must perform, such as grocery shopping or making a bed. Physiological arousal, including perception of bodily functioning, is also crucial to the inferences that people make about their physical abilities. For example, exercise may be recommended as part of a recovery program for chronic pain conditions. Furthermore, if fatigue and pain are experienced while exercising, this may be interpreted as personal inadequacy, which can erode feelings of efficacy. The person in pain who interprets these signs as normal physiological responses is more likely to adjust to the dynamic process of pain recovery than the one who does not (Turk & Rudy, 1992).

(i) Pain self-efficacy

Bandura et al. (1987) argued that self-efficacy mediates the unpleasantness of stressful experiences. This idea has evolved into the concept of *pain self-efficacy* or "a

measure of pain-tolerance beliefs" (Nicholas, 1994, p. 1) similar to Bandura's concept. People with high self-efficacy are more likely to persist longer and expend extra effort in utilizing cognitive techniques to adjust to persistent pain (Litt, 1988a). Higher levels of self-efficacy should result in less anxiety, which in turn should minimize the distress of the chronic pain experience. From a physiological perspective, limited research suggests that self-efficacy may directly affect chemicals produced by the body, i.e. catecholamines and endogenous opiate levels, that influence perceived pain (Bandura, Taylor, Williams, Mefford, & Barchas, 1984). This implies that in times of stress, physiological measures such as heart rate and blood pressure are less likely to be adversely affected if self-efficacy is high.

Bandura, Adams and Beyer (1977) original research methodology required that particular dimensions of self-efficacy be measured. These are described as level, strength and generality of self-efficacy judgments, allowing for the prediction of self-efficacy in various domains. Operationalising these judgments involved identifying relevant target behaviours and ordering them into a hierarchy of *least* to *most* difficult with associated confidence ratings made for each step (for more detail on this methodology, see Bandura et al., 1977). However, many researchers have adopted a broader approach to the study of self-efficacy rather than the precise method that Bandura et al. (1977) have advocated (see Berry & West, 1993, for an extensive review about self-efficacy).

In non-treatment and/or pre-treatment studies, pain self-efficacy beliefs have been assessed in relation to many factors pertinent to adjustment in people with chronic pain. For example, Arnstein et al. (1999) assessed 126 chronic (unspecified) pain patients before their first consultation in a pain clinic. They reported that self-efficacy mediated the relationship between pain intensity and depression, with some contribution from disability. In particular, analyses showed self-efficacy accounted for more "of the explained variance in depression above that which could be accounted for by pain intensity and disability alone" (p. 488). They used a pain self-efficacy scale developed by Anderson et al. (1995).

Council et al. (1988), in a study with 40 chronic low back pain patients, reported that believing that an activity would lead to pain is likely to undermine the confidence one has to

actually perform the behaviour and so the one is less likely to engage in that behaviour. Other studies have variously found that high self-efficacy beliefs are related to high self-esteem, life satisfaction and less depression (Blalock, McEvoy DeVellis, & DeVellis, 1989), less anxiety and fewer physical symptoms (Martin, Holroyd, & Rokicki, 1993).

Nicholas (1994) assessed pain self-efficacy in 103 chronic low back pain patients with the Pain Self-efficacy Questionnaire. This was conducted in order to determine the state of confidence people with chronic pain had in dealing with daily life, before treatment began. Such a strategy was considered useful in determining appropriate interventions. This research developed from the recognition that many patients failed to benefit from treatment because they often believed that pain was only temporary and that life would resume as 'normal' once the pain had been 'fixed'. It was found that high self-efficacy was significantly related to higher control over pain and reduced perceived disability and depression (measured by the BDI).

More recently, Asghari and Nicholas (2001), using the PSEQ with 145 chronic pain patients, found that high baseline confidence in ability to perform several tasks despite pain, was a significant predictor of reduced avoidance and total pain behaviour, at the initial assessment and also, 9 months later. This was true even when controlling for variables such as age, gender, depression and pain intensity. Although there is still debate over specific pain behaviours, they are generally considered to be those that are incompatible with what would be perceived as 'positive adjustment' to chronic pain. In this case, they included such behaviours as taking medication, complaining of pain, rubbing pain sites, avoiding usual tasks and the like.

Other sources of information about self-efficacy and chronic pain are pre- and post-treatment comparison studies of chronic pain patients. Most of these studies used pain severity, interference with work, observed medication usage, exercise tolerance, or physical and psychological functioning as indicators of adjustment or as outcome variables. For example, in a study with 107 (unspecified) chronic pain rehabilitation patients, Strong, Westbury, Smith, McKenzie and Ryan (2002) found that pain self-efficacy was the best

predictor of positive treatment outcomes, such as decreased pain and increased activity. In a study with 212 chronic pain treatment participants, Williams et al. (1993) found that all measures of physical and psychological functioning, including self-efficacy, showed significant improvement from pre- to post treatment and were related.

Lorig, Chastain, Ung, Shoor and Holman (1989) reported that higher self-efficacy (for physical functioning and pain management) at pre-treatment was related to less perceived pain intensity and depression at four month post-treatment. Dolce et al. (1986a; Dolce, Crocker, Moletteire, & Doleys, 1986b) found that self-efficacy expectancies significantly improved following treatment and these were related to decreased observed medication usage and increased exercise tolerance. Philips (1987) found that low self-efficacy was related to lower perceived control over pain, as well as greater tendencies to avoid any activity seen as potentially pain producing. All these results support Bandura's (1977) argument that successful experiences are likely to increase self-efficacy beliefs.

However, other findings based on pre- and post-treatment comparisons are not consistent. O'Leary, Shoor, Lorig and Holman (1988) found that higher self-efficacy before treatment was predictive of less disability post-treatment although changes in self-efficacy were not related to changes in depression. Likewise, Nicholas, Wilson and Goyen (1992), using the PSEQ, reported that changes in self-efficacy pre- to post-treatment did not relate to changes in depression. Several clinical studies have reported that self-efficacy acts as a moderator of stressful experiences. Kores, Murphy, Rosenthal, Elias and North (1990) found in an inpatient-management program that those pain patients with higher self-efficacy for general activities and improvement were able to tolerate sitting and standing better and also felt that they had improved more overall, than were those with lower self-efficacy.

It is difficult, however, to compare much of this research. Only two studies assessed patients with the same type of pain (i.e. chronic back pain). Several of the studies had small samples of fewer than 40 participants (Dolce et al., 1986b; Kores et al., 1990; Nicholas et al., 1992; O'Leary et al., 1988). Again, some of this research involved arthritis patients who may not be readily comparable to those with back pain. Only a few appear to have used

standardized self-efficacy scales, developed for people with chronic pain. Most research used self-efficacy ratings developed solely for the study reported, which raises questions about reliability and validity. Also, most of the studies have looked at self-efficacy in isolation, giving little information on interrelationships between other aspects of the chronic pain experience, particularly control beliefs.

(ii) General self-efficacy

Besides pain self-efficacy, there has been research into a 'general self-efficacy' construct. General self-efficacy is defined as "an individual's past experiences with success and failure in a variety of situations [which] should result in a general set of expectations that the individual carries into new situations" (Sherer, Maddux, Mercandante, Prentice-Dunn, Jacobs, & Rogers, 1982, p. 664). When a person feels generally competent at most tasks and on an interpersonal level, general self-efficacy is high. The Sherer et al. (1982) 'generalized' construct does not appear to differ conceptually from Bandura 's (1977) specific construct, except in place on a continuum (Sherer, 1990). That is, Bandura proposed that "self-efficacy expectancies vary in generality" (cited in Sherer, 1990, p. 1242). This implies that Bandura concentrated on one end of the continuum while those investigating general self-efficacy have concentrated on the other (Wang & Richarde, 1988). Moreover, "general self-efficacy [is said to influence] ....specific self-efficacy" (Shelton, 1990, p. 991). This implies that general self-efficacy is likely to affect the confidence that one has about dealing with pain.

However, there has been limited research on general self-efficacy and at the time of writing, no published research could be found that was strictly relevant to the current research, i.e. general (as opposed to specific, i.e. pain) self-efficacy, chronic pain and relevant variables. Most general self-efficacy research has involved students and/or alcoholics (Lennings, 1994; Sherer & Adams, 1983; Sherer et al., 1982; Tipton & Worthington, 1984; Woodruff & Cashman, 1993). High general self-efficacy was found to be related to high internal beliefs that one is responsible for one's own life and better

adjustment to life (Sherer et al., 1982). General self-efficacy is related to locus of control, mastery and self-esteem and specific self-efficacy (Woodruff & Cashman, 1993). More recently, Bosscher and Smit (1998) found that older adults with high general self-efficacy were very confident about maintaining autonomy and living independently.

In sum, pain self-efficacy and general self-efficacy appear to be very important in the chronic pain experience. However, there is little consistency in terms of measures used to assess the different types of self-efficacy, often with limited or no reference to reliability or validity. Many studies have used *in-house* scales or scales developed solely for the purpose of the research being conducted (Blalock et al., 1989; Dolce et al., 1986b; Lin, 1998). Evidence to date implies that the two types of self-efficacy are related to each other and to other beliefs involved in the chronic pain experience. Therefore, people who have adapted to chronic pain would be expected to report higher general *and* pain self-efficacy and control and lower levels of interference and depression.

3.3.5 Beliefs related to emotions and chronic pain

In the psychological literature, emotions are considered to be "a fundamental part of the pain experience" (Chapman, 1995, p. 283). This is in keeping with a cognitive-behavioural perspective, but it has not always been so. While historically, pain was considered by the ancient Greeks to be "the negative counterpart of pleasure" (Craig, 1995, p. 307), and by Aristotle to be an 'affect' itself, these ideas were over-ridden by the orthodox sensory model of pain. More recently, the focus on emotion has resurfaced with the rise of the multi-dimensional models of pain which include the sensory as well as the affective aspects of the experience. This has been recognized by the IASP and, as mentioned previously, their most recent definition of pain acknowledges the very real place that emotion plays in the suffering associated with chronic pain.

Despite this acknowledgement, limited research has been carried out to elucidate the role that emotions play in chronic pain. This is confounded by the fact that emotional distress is part of any pain experience, as well as a consequence (Chapman, 1995). This is

particularly true in chronic pain where the individual is likely to become more emotionally distressed at continued inability to find relief. Adding to this dilemma is the fact that there is little general agreement on how to define 'emotion' which is really a generic term including "many different subjective states" (Craig, 1995, p. 308). Rolls (1986) referred to emotions as "states elicited by reinforcing stimuli" (p. 126). Fonberg (1986) defined emotion as "the nervous process that determines what kind of stimuli coming from the inner and outer environments are desirable for the organism and what are not" (p. 302). These are just two of many disparate definitions, which reflect markedly different frames of thought (Chapman, 1995). Lazarus (1993) suggested that emotions are either positive (i.e. happiness, love) or negative (i.e. anger, anxiety) thought-based feelings. As controversy continues about their role in the chronic pain experience, for the purposes of this thesis, anger and anxiety are considered to be affective aspects of chronic pain that may, if excessive, impede adaptation or adjustment to the condition.

(i) Anger

During the last two decades or so, researchers have speculated on the role that anger may play in the development and maintenance of particular medical conditions, including coronary heart disease, diabetes and cancer (Burns, Friedman, & Katkin, 1992; Chesney & Rosenman, 1985; Cox & MacKay, 1982; DeShields, Jenkins, & Tait, 1989) and adjustment to chronic pain (Greenwood, Thurston, Rumble, Waters, & Keefe, 2003; Jensen, Turner, Romano, & Karoly, 1991). Anger has been examined in relation to depression and disability in people with chronic headache, adjustment to chronic pain and the prediction of treatment outcome in chronic pain patients (Burns et al., 1998; Burns et al., 1992; Burns et al., 1996; Duckro, Chibnall, Terry, & Tomazic, 1994; Kerns et al., 1994). Pain theories, including the gate control and the neuromatrix theories, endorse the view that pain can increase negative emotions like anger by affecting pain modulation systems (Greenwood et al., 2003). Research has concluded that anger is not only a critical concomitant of the chronic pain experience (Burns et al., 1998; Burns et al., 1996; Kerns et al., 1994; Romano

& Turner, 1985; Wade, Price, Hamer, Schwartz, & Hart, 1990), but also affects relationships between those experiencing pain and their social networks and health care providers (Greenwood et al., 2003). People with chronic pain appear to experience high levels of anger compared to other groups of people (Hatch, Schoenfeld, Boutros, Seleshi, Moore, & Cyr-Provost, 1991; Okifuji, Turk, & Curran, 1999a). Research has mainly focused on four constructs of anger, anger management style, aggression and hostility. This thesis will address only the former of these, anger and anger management style.

'Anger' usually refers to an unpleasant or aversive emotion, which may range from a mild reaction to intense rage (Smith, 1994). From a conceptual perspective, anger is an emotion that has been described in two forms: state and trait anger. *State* anger has been defined as " an emotional state marked by subjective feelings that vary in intensity from mild annoyance or irritation to intense fury and rage" (Spielberger, 1988, p. 1). State anger can manifest itself physiologically as tense muscles and an aroused autonomic nervous system. State anger intensity is situation specific and tends to vary over time according to how an individual perceives the world in terms of injustice and unfair treatment. Becoming angry can be an adaptive reaction, if expressed constructively, however, if one habitually reacts in such a fashion, it may become maladaptive (Greenwood et al., 2003).

Trait anger has been defined as " the disposition to perceive a wide range of situations as annoying or frustrating and the tendency to respond to such situations with more frequent elevations in state anger" (Spielberger, 1988, p. 1). This implies a more stable characteristic which is less prone to fluctuation and which is reflected in judgements such as 'she is an angry person'. Spielberger, Johnson, Russell, Crane, Jacobs and Worden (1985) described *anger management styles* of anger expression and suppression. The former refers to anger that is expressed, outwardly toward other individuals or objects in the environment, while the latter refers to angry feelings that are suppressed or held inside. According to Burns et al. (1998), both anger expression and suppression are "implicated in the development and/or maintenance of poor physical health [....] including chronic pain" (p. 1052).

There continues to be speculation on the extent to which anger may contribute to the pathogenesis of chronic pain and how it may manifest (Kerns et al., 1994). Traditionally, the predominant view was the psychodynamic perspective (Blumer & Heilbronn, 1982; Engel, 1959). The basic premise of this theory is that repressed anger manifests as somatic pain. Pilowsky and Spence (1976) cite support for this view in research that found people with chronic pain reported more repressed anger than did a control group. Major proponents of this view, Beutler et al. (1986), hypothesized a bio-physiological element by suggesting that the inability to express anger lowers the body's endogenous opoid levels (i.e. endorphins), thereby increasing sensitivity to pain. Difficulty in expressing anger is therefore thought to be related to higher perceived pain intensity and reported pain behaviours and a tendency to avoid conflict with others (Beutler et al., 1986). This suggests that anger may interfere with adaptive functioning in some way and that a cycle is initiated so that reduced functioning becomes increasingly aggravating, and anger escalates further, interfering with efficient functioning and so on. This can lead to reduced perceived self-control and self-efficacy which would, in turn, be likely to lead to increased levels of anger and frustration, thus perpetuating the cycle (Kerns et al., 1994).

Some have argued against the psychiatric perspective due to a lack of support. Furthermore, there has been an increase in biopsychosocial research that has augmented some of these ideas with stronger clinical evidence. For example, it has been shown that anger may encourage endogenous opioid dysfunction such that the body becomes less efficient at deal with chronic pain (Bruehl, Burns, Chung, Ward, & Johnson, 2002; Bruehl, McCubbin, & Harden, 1999). In addition, there is evidence to suggest that negative emotion, such as anger, may adversely affect the immune system making it more difficult for the body to manage pain (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002).

There have been empirical investigations of anger as it relates to chronic pain (Okifuji et al., 1999a), particularly from a cognitive-behavioural perspective. Maladaptive cognitive activity exacerbates the chronic pain condition (Turk & Rudy, 1992) and in some circumstances, anger is maladaptive if it contributes to the aversiveness of the chronic pain

experience (Okifuji et al., 1999a). Not only do people with chronic pain often deny their anger but some may be reluctant to express anger, as it is socially inappropriate in many cultures and can result in interpersonal conflict (Corbishely, Hendrickson, Beutler, & Engle, 1990).

Anger is thought to be commonly experienced among people with chronic pain (Blumer & Heilbronn, 1982; Okifuji et al., 1999a), but most research studies fail to specify the type of anger being investigated (Wade et al., 1990). There are few, if any, clear findings with regard to state anger in people with chronic pain, although one may suspect that, as it is not a stable characteristic, it would fluctuate depending upon a myriad of factors. It is thought that people with chronic pain are likely to be more prone to anger and therefore they should demonstrate higher trait anger (Beutler et al., 1986). For example, Achterberg-Lawlis (1982) found that anger was a dominant personality characteristic in people with RA, but support for this view among other groups of people with chronic pain is limited.

While there is some evidence that expressed anger is related to depression (Tschannen, Duckro, Margolis, & Tomazic, 1992), most studies report that suppressed anger is more of a predictor of pain severity and depression. Work by Hatch et al. (1991) supports this with the finding that suppressed anger was significantly related to pain in 47 people with headache but expressed anger did not differ significantly between these people and a control group. Kerns et al. (1994) also made similar conclusions regarding 142 mixed chronic pain patients. Similarly, Duckro et al. (1994) found that suppressed anger was involved in depression, leading to perceived disability but that expressed anger had no such effect. However, in contrast to these studies, Conant (1998) did not find that anger suppression was related to pain, although patients who felt they had more control over pain, tended to report less pain severity. In addition, Materazzo (1997) found, with a community sample of migraine headache patients that expressed, rather than suppressed anger, predicted pain severity.

There has also been some research into gender differences in the relationship between anger, pain severity and depression. While it appears that males and females

respond differently with respect to expressions of anger (Okifuji et al., 1999a), research findings are not consistent in either non-pain or pain populations. For example, while Hashida and Mosche (1988) reported that females generally tend to score higher on measures of anger expression than males, other researchers report the opposite (Fischer, Smith, Leonard, & Fuqua, 1993; Kinder, Curtiss, & Kalichman, 1993; Sternbach, Wolfe, Murphy, & Akeson, 1973). In addition, some research has found no difference between males and females in anger expression (Averill, 1983; Stoner & Spencer, 1987).

Amongst people with chronic pain, both Sternbach et al. (1973) and Kinder et al. (1993) report that males are more able to express their anger than are females. However, Curtiss, Kinder, Kalichman and Spana (1988) found that for some males and females, the opposite is true, although adjustment was not investigated. In the previously mentioned work by Burns et al. (Burns et al., 1998; 1996), gender differences were reported but were not consistent from one study to the other. Overall, males who suppressed anger were less well adjusted to chronic pain. However, while females who expressed anger appeared to also be less adjusted, this was not confirmed by a second study. Burns et al. (1998) concluded that anger management style affects outcome more than proneness to anger (trait anger). Most studies have not had sufficient female participants to determine clear gender differences (Kerns et al., 1994; Lane & Hobfoll, 1992) or have not reported relevant analyses (Hatch et al., 1991; Wade et al. 1990). In the present research, state and trait anger plus anger management styles will be assessed in the participants with particular emphasis on gender differences.

(ii) Anxiety

Overall, consensus on the role that anxiety plays in chronic pain remains obscure. Dersh et al. (2002) have noted that while research has shown that anxiety is associated with chronic pain, the chronic pain-depression relationship has tended to receive more attention in terms of research and theory development. Furthermore, research is complicated by, not only alternate views of the role that anxiety plays in the chronic pain experience, but also

various forms of anxiety mentioned in the literature. For example, McCracken, Zayfert and Gross (1992) refer to 'pain anxiety' while Hadjistavropoulous, Hadjistavropoulos and Quine (2000) refer to 'health anxiety'. Some of this literature is not strictly relevant to the present research, so this discussion will mainly refer to anxiety or trait and state anxiety, related to chronic pain. In addition, few studies in the chronic pain literature appear to distinguish between state and trait anxiety but refer only to the broader category 'anxiety'. In the present thesis and in keeping with this practise, state and trait anxiety will only be referred to as such if they have been specified in research.

Anxiety has been described as "a multifaceted response to threatening situations [.....] characterized by cognitive apprehension, neurophysiological arousal, and a subjective experience of tension or nervousness" (Leary, 1991, p. 161). This is just one of various definitions of anxiety, which refers to both an emotional situational state and a more stable personality trait (Adams & Taylor, 1997). The former is referred to as *state* anxiety while the latter term refers to *trait* anxiety. The distinction is similar to anger. Bouts of anxiety reflect state anxiety, while experiencing a generally high level of anxiety most of the time reflects a personality characteristic. An individual who is more prone to be anxious all the time is also more likely to suffer situational anxiety.

Some of the symptoms that an anxious person may exhibit include motor responses such as twitching and shaking, muscle tension, aches, soreness, restlessness and fatigue. There is also a range of extreme physiological responses that may be experienced including shortness of breath, tachycardia, sweating or clammy palms, dryness of mouth, dizziness, nausea or diarrhoea, and extremes of body temperature such as chills and hot flashes. Other symptoms include hypervigilance, concentration difficulties and feelings of dread and fear (Feldman, 1993).

Increased anxiety has been shown to be associated with increased pain report and disability in various chronic pain groups (Main & Waddell, 1991; McCracken, Zayfert & Gross, 1993; Vassend, Krogstad, & Dahl, 1995), especially trait anxiety (McCracken, Gross, Aikens, & Carnrike Jr., 1996). Anxiety has also been indicated as a significant predictor of

adjustment to chronic pain (Vlaeyen et al., 1995b). Those in chronic pain tend to be more fearful and anxious according to reviews by both Craig (1994) and Asmundson, Norton and Norton (1999). Also, it appears that anxiety is more prevalent among those in chronic pain than in the general population (Asmundson, Jacobson, Allerdings, & Norton, 1996).

Craig (1995) speculated that anxiety is more likely to be associated with acute pain when a person is fearful of future consequences, whereas sadness and depression are more likely to dominate when the problem becomes chronic. Conversely, Fordyce and Steger (1979) suggest that anxiety is related to both acute and chronic pain but in different ways. For example, while in acute pain, anxiety is more likely to be experienced until successful treatment has resulted in an end to the pain. For those in chronic pain, if treatments become increasingly unsuccessful, anxiety is likely to escalate which may increase pain severity.

Ackerman and Stevens (1989) reported that anxiety exacerbates both chronic pain perception and depression. According to a psychological perspective, anxiety is characterized by feelings of 'dread', 'fear' and 'tension', while depression is described more as feeling 'blue', 'sad' and 'gloomy' (Feldman, 1993). Moreover, there is much controversy over the extent to which anxiety and depression are distinct syndromes or whether they can occur co-morbidly (Craig, 1995). The symptoms which are most reported to overlap include, fatigue, concentration problems, insomnia and psychomotor agitation. Preskorn and Fast (1993) and Gulley and Nemeroff (1993) maintain that there is such a large overlap of symptomatology, it can be very difficult to meaningfully distinguish between the two.

Some research has suggested that it may be due to an inability of the commonly used self-report scales to discriminate between the two syndromes. For example, Feldman (1993) re-analyzed data from 4 studies investigating anxiety and depression in non-chronic pain samples and reached a similar conclusion. Confirmatory factor analyses were used to conclude that depression and anxiety self-report scales were probably measuring a 'general negative mood state' rather than distinct constructs. Lovibond and Lovibond (1995) cite work by researchers such as Gotlib and Cane (1989) and Clark and Watson (1990) who

found that most anxiety and depression scales correlate very highly and that clinicians' ratings duplicated these high correlations. They also report on work by Beck, Epstein, Brown and Steer (1988) and Costello and Comrey (1967), which was little more successful at developing discriminating scales.

Nelson and Novy (1997) suggested that better discrimination could be achieved by more rigorous analysis of the commonly used scales. They assessed 220 heterogenous pain patients on the BDI and the STAI and factor analysed the results, concluding that it was possible to discriminate between depression and anxiety factors, although this was minor. However, they did find that a 'negative affect' factor was probably more of a primary underlying construct than either depression or anxiety. This is consistent with Feldman's (1993) findings.

Lovibond and Lovibond (1995) developed self-report scales that were able to distinguish anxiety and depression in normal, non-clinical samples. Their 1995 study, the culmination of research carried out between 1979 and 1990, evaluated the Depression Anxiety Stress Scales (DASS) as well as compared them to the Beck Depression Inventory (BDI: Beck et al., 1961) and the Beck Anxiety Inventory (BAI: Beck et al., 1988). This research, with 717 non-clinical participants, found that while the DASS could discriminate between three 'negative emotional syndromes', depression, anxiety and stress, there was still a high degree of correlation between the three, especially between anxiety and stress.

It was suggested that these states are similar in both clinical and non-clinical groups, although varying in severity and chronicity. Lovibond (1998) more recently reported that the DASS had been used in a 3 to 8-year longitudinal research project and that the depression, anxiety and stress syndromes appeared stable over time. However, to date these scales have not been used specifically to assess anxiety and depression in those with chronic pain. In addition, Creamer, Foran and Bell (1995) point out that the process of development of measures may be problematic. For example, different aspects of the construct may be defined in the process, possibly leading to validity problems, and removing items may lead to the omission of key aspects of the disorder from the measuring scales. In addition, it has

been suggested by Gotlib and Cane (1989) that new measures should be treated with caution, if they use non-overlapping symptoms of the disorders because this might lead to problems with construct validity.

However, the issue may not just be about developing better discriminating scales as a clinical diagnosis of co-morbidity occurs more often than not (Watson, Weber, Assenheimer, Clark, Strauss, & McCormick, 1995). Rouillon (1999) noted that "whether anxiety plus depression represents pure co-morbidity or a mixed disorder is [still] a subject for debate" (p. S87). In 2001, Tyrer stated that the combination of mixed anxiety and depression occurs so commonly in the population that medical doctors make frequent diagnoses to that effect. The term "cothymia" (Tyrer, 1989) has been coined to describe the occurrence of two equally significant moods that occur together. Despite this, at the time of writing Tyrer (2001), noted that neither the DSM-IV nor the ICD-10 had a diagnosis for a mixed anxiety and depression co-morbid disorder, except as a 'sub-syndromal' disorder. Most recently, Greenberg and Burns (2003) noted that most of the empirical evidence related to anxiety in chronic pain has supported the idea that anxiety is part of "a general malaise also composed of depression and suppressed anger" (p. 224).

In recent years, there has been a great deal of research into fear-avoidance models of chronic pain of which anxiety is a key component. This has been briefly discussed in chapter two, but some of this information is also pertinent to a discourse about anxiety and chronic pain. Although several such models have been proposed they share a common premise: that being afraid of pain or injury or the anticipation of pain encourages those in pain to avoid or escape from those activities which are potentially likely to cause pain (Norton & Asmundson, 2003).

While these models focus mainly on fear, anxiety is also considered as a distinctly different construct, although highly enmeshed with fear as a response to pain. According to a general fear-avoidance model (Vlaeyen & Linton, 2000), injury resulting in pain may lead to either of two responses. Firstly, if pain is not seen as threatening, a positive adaption is made (i.e. rest followed by gradual return to normal activity). Alternatively, if pain is viewed

as threatening, the person may become trapped in a cycle of fear, avoidance of potentially pain producing activity, and hypervigilance for pain sensations, followed by muscular and systematic atrophy, depression and ultimate disability (Norton & Asmundson, 2003). Norton and Amundson (2003) have recently amended this model by giving greater credence to "the physiological effects of autonomic dysregulation and muscular tension" (p. 74), than has previously been considered. Greater discussion of this point, however, is beyond the present scope).

Furthermore, the anxiety that arises from perceiving pain as threatening could be either a type of phobic response to chronic pain or a manifestation of an underlying predisposition to anxiety (Greenberg & Burns, 2003). According to the former, anxiety is the driving force behind a particular phobic response to chronic pain, variously described as 'kinesiophobia' (Kori, Miller, & Todd, 1990), 'fear avoidance' (Waddell et al., 1993) and 'pain anxiety' (McCracken, Zayfert, & Gross, 1992). This idea partly derives from the work of Lethem et al. (1983). Those who believe that pain will occur through activity become anxious and learn to avoid potentially painful activities and in so doing miss out on opportunities to disconfirm fear and anxiety associated with movement (Philips, 1987). A body of work has supported this idea that 'pain anxiety' is some type of phobia, distinct from a general negative affect and trait anxiety (Greenberg & Burns, 2003). Some studies have found that pain anxiety predicts changes in adjustment beyond that accounted for by trait anxiety and negative affect (Crombez et al., 1999; McCracken et al., 1992).

There is also the idea that someone predisposed to anxiety, expects and is sensitive to anxiety associated with potential threats, i.e anxiety is "a manifestation of an underlying predisposition to fear symptoms of anxiety" (Greenberg & Burns, 2003, p. 225). This idea developed from work by Asmundson et al. (1999), Reiss and McNally (1985) and Reiss, Peterson, Gursky and McNally (1986). Subsequently, the person fears the anxiety symptoms associated with the pain more than the pain itself. Greenberg and Burns (2003) found, with 70 chronic musculoskeletal pain patients, that most of the pain anxiety associated with performing certain tasks was accounted for by sensitivity to anxiety, even

when depression and trait anxiety were controlled for. They concluded that pain anxiety was probably therefore more likely to represent anxiety sensitivity than a particular phobia.

A slightly different perspective of anxiety related to chronic pain is given by Sharp (2001) in the reformulated cognitive-behavioural model. While the Vlaeyen and Linton (2000) model focused on the key role that fear plays in the development of chronicity, Sharp emphasizes more the impact that anxiety has on beliefs and appraisals about pain. For example, anxious appraisals may help to maintain physiological arousal, confirming the belief that original trauma (real or perceived) still exists. Moreover, being anxious may increase the likelihood of negative appraisals, maintain avoidance behaviours and help to perpetuate a dysfunctional cycle.

These latter ideas about anxiety related to chronic pain are introduced here, given that they represent a burgeoning focus of research. In addition, it is also a relatively recent area of investigation, and the longitudinal nature of the present project precluded a more detailed discussion of literature that post-dated the original study.

Some specific research has investigated the relationship between anxiety, depression and pain. The research by Wade et al. (1990) found that while both anxiety and anger were important concomitants of depression in pain, anxiety was the most important predictor of the unpleasantness associated with pain. It was also suggested that in the long term anxiety and anger contribute to decreased pain tolerance and increased depression that would further interfere with daily life. In addition, the reliability and validity of the instrument used in this study were not reported. Kuch, Cox, Evans, Watson and Bubela (1993) reported that anxiety was significantly related to depression, in a sample of 61 fibromyalgia patients. It was concluded that, as anxiety appeared to be unrelated to pain intensity or frequency, mood was probably more related to the ability to cope with pain as opposed to actual perceived pain. They noted that the anxiety-depression-pain association is far from clear. As with other researchers, these have also argued that chronic pain patients tend to be quite heterogenous and therefore warn against making generalisations from one syndrome to another.

McCracken and Gross (1998) investigated 70 chronic pain patients before and after a 3-week treatment program. They were assessed on pain levels, affective distress, interference with activity, daily activity and pain-related anxiety with respect to 'functional restoration'. Reduced pain-related anxiety predicted improved functioning on each of the outcome measures, on all of which they showed significant improvement. Even when change in depression levels was controlled for, changes in pain-related anxiety was still a significant predictor.

Other research has not demonstrated a consistent relationship between anxiety, depression and chronic pain. Philips and Grant (1991a) assessed anxiety in a study of 117 people with acute pain, 40% of whom continued to suffer chronic pain after 6 months. They concluded that anxiety was not a significant problem in the acute phase, nor did it become a problem as the condition became more chronic. The greatest change occurred in what they call the 'sub-chronic' phase (at about 3 months) with a stabilizing during the following 3 months. However, these findings do not shed light on what happens to the emotional health of a person who continues to be in pain past the 6-month point.

There is also a dearth of research into possible gender differences with respect to anxiety and chronic pain (Edwards et al., 2000). In the wider community, women commonly report more anxiety (Kroenke & Spitzer, 1998) and there is speculation that females with chronic pain may experience higher anxiety than males, but empirical evidence for clinical populations is limited (Bolton, 1994; Dougher, Goldstein, & Leight, 1987; Kroenke & Spitzer, 1998; Unruh, 1996).

To date, Edwards et al. (2000) have completed one of the few studies showing gender differences among clinical chronic pain patients. In their research, with 215 males and females with chronic pain, they found that increased anxiety was related to poor adjustment to chronic pain, but only in males. This is despite the fact that males and females did not differ in the amount of anxiety reported. Highly anxious males reported greater pain severity, lower daily activity levels and perceived that pain was a greater interference to daily life. This suggests that, either males become more anxious because of

chronic pain relative to general female anxiety, or that anxiety is more diverse among females with chronic pain and that only males more prone to anxiety develop chronic pain (Edwards et al., 2000). Again, definitive conclusions are difficult to make because of the paucity of research findings on this issue.

In summary, findings about anger and anxiety in chronic pain are limited and/or inconsistent. Feelings of anger and anger management styles have been found to be associated with depression, daily interference and activity interference, but with some gender differences. The degree to which anger management style is related to pain severity may depend upon gender and perceived social support, although findings are ambiguous. Likewise, there is debate about the extent to which anxiety is actually related to pain severity, although it has been found to predict the unpleasantness associated with pain. It is also generally unclear which type of anxiety (state or trait) is most related to chronic pain and psychological dysfunction. While anxiety has been found to clearly relate to depression in some cases of chronic pain, in other research it cannot easily be distinguished from depression. It is also not clear how anger and anxiety interrelate with each other or the other variables pertinent to the present research. Given this lack of clarity, both state and trait forms of anger and anxiety will be examined in the chronic back pain sample of participants of the present research.

3.4 Summary of the literature review

Research has been conducted on various factors related to depression in clinical chronic pain patients. Most of the results are equivocal with regard to how demographic characteristics such as gender, age, educational, marital and employment status might affect response to pain. Research into cognitive-behavioural factors has shown some consistent results with clinical patients with chronic pain. For example, depressed persons with chronic pain report more pain, are less active, spend more time resting and do not cope well with their condition. Those who perceive that pain is a significant interference or disruption to their lives are also more depressed than those who accept pain as part of daily

life. Those people with chronic pain who cope better and adjust faster tend to believe that they are more in control of their condition and accept greater responsibility for their own health. Likewise, those who are confident about living a 'normal' life or have high self-efficacy, despite the pain, are more likely to persist longer and expend more effort in gaining control over their situations. A feeling of self-efficacy has also been found to act as a mediator between chronic pain and depression.

Lack of social support has been shown to be a risk factor for the development of psychological dysfunction in those suffering illness, including chronic pain. If those people closest to an individual provide negative (i.e. are critical or disparaging) rather than positive (i.e. emotionally helpful) support, suffering is likely to be exacerbated. The role of emotions in adjustment to chronic pain has been explored, albeit in a limited fashion. Anger has been found to be directly related to depression associated with the chronic pain experience, and to have an indirect influence on disability via depression acting as a mediator. Likewise, anxiety has been reported to exacerbate both depression and chronic pain perception and be inversely related to poor adjustment. However, there is still debate as to whether anxiety and depression represent different syndromes or a co-morbid state independent from chronic pain. Methodological limitations preclude reaching definitive conclusions with regard to people with chronic pain who are not clinical patients.

3.5 Aims of the research

Given the vast chronic pain and depression literature, the present thesis addresses the following aims that are fundamental to the key objectives outlined in chapter 1.

3.5.1 Aim 1: To determine the general characteristics of non-clinical chronic back pain participants in terms of chronic pain profiles

The first aim is to examine the chronic pain profiles of the participants to determine how similar or different these people are to clinical patients. Such a system developed by Kerns et al. (1985) has shown that in most clinical studies, participants can be readily

classified into three main groups of patients, dysfunctional, interpersonally distressed and adaptive copers. The latter group is that considered mostly likely to adapt to the condition and live positive lives, despite the ongoing stress of persistent pain. The extent to which the sample of participants involved in this research resemble or do not resemble typical clinical pain groups will be important in terms of the extent to which statistical results can be generalized to a non-clinical population.

3.5.2 *Aim 2: To compare chronic back pain participants to non-pain participants on several specific cognitive measures to determine the extent to which pain affects cognitive functioning*

This aim involves making a comprehensive examination of the demographic characteristics and cognitive-behavioural responses to chronic pain in a sample of people with chronic back pain who have not attended a pain clinic[3], in a longitudinal study. The people with chronic pain will be compared to a control group of people without chronic pain on selected cognitive variables to determine how pain may have influenced their psychological responses.

In the present research, variables that appear to inform a cognitive-behavioural approach to pain research were chosen. These variables have been studied mainly in the clinical context, thereby providing valuable information on which to base the present research. Consistent with a cognitive-behavioural approach to the study of pain, the variables to be investigated are categorized as follows:

1) *Demographic variables:* gender, age, educational status, marital status, and employment status;
2) *Sensory variables*: pain severity and pain duration;
3) *Cognitive variables*: perceived interference of pain, control over pain, social support and self-efficacy beliefs;
4) *Behavioural variable:* activity levels;

[3] It may be debateable whether the final participants can truly be said to be 'non-clinical' although every effort was made to recruit such subjects. This is discussed more fully later in the thesis as appropriate.

5) *Affective variables*: anger and anxiety.

In the present thesis, the chronic pain-depression relationship will be examined at two points in time, approximately 5 years apart. Chronic pain, depression and the afore-mentioned correlates will be measured at Time one (T1) and Time two (T2). These will be compared to determine how pain has affected people over a long period of time and also to determine the existence of significant differences in the prediction of depression at each time.

3.5.3 *Aim 3: To compare sub-groups within the sample of people with chronic back pain, i.e. specifically compare those with depression to those without depression on demographic and cognitive variables*

According to the literature, those people with chronic back pain who are depressed should differ on several demographic characteristics, compared to those who experience chronic back pain but who do not demonstrate depressive symptomatology. These characteristics include gender, marital status, age, education level, employment status, medication useage, amount of surgery and time in chronic pain. Furthermore, they should also differ in some cognitive responses to pain, specifically in terms of how much pain interferes in their daily functioning, anger levels, how responsible they feel for their own health and how confident they are, in general, and specifically about how they handle their pain.

3.5.4 *Aim 4: Testing general, and gender-specific, cognitive-behavioural models of chronic pain and depression*

The fourth aim of the thesis is to develop and test the role of cognitive appraisal in the relationship between chronic pain and depression in cognitive-behavioural models. While some research has suggested that chronic pain and depression are directly linked, other suggests that they are mediated by various cognitive-behavioural variables. Furthermore, testing of this premise has been limited to clinical populations and the nature of

the relationships is not clear. Only interference and life control and have been clearly identified as components of a cognitive-behavioural model. Furthermore, of all the variables that may be useful in such an endeavor, only interference, life control and self-efficacy have been tested as mediators in a chronic pain-depression relationship. In addition, none of these have been tested together in either a cross-sectional or a longitudinal study with a non-clinical group with chronic back pain. In the present research, models in the style of Rudy et al. (1988, see Fig. 2.1) will be tested with *path analysis*, a multiple regression technique, using difference scores in pain severity, depression and relevant variables.

The group will be tested in its entirety. However, as there is some suggestion that males and females differ in response to pain, part of the fourth aim of the thesis will be to investigate gender differences in the role of cognitive appraisal in the chronic pain experience. This will be determined by testing gender-specific cognitive-behavioural models of chronic pain and depression.

Research findings about gender differences in response to chronic pain and depression are equivocal. Although a 'typical' person with chronic pain at risk of depression has been identified as a middle-aged, single, female with poor education (White & Harth, 1999), there is little consensus on whether such a typical person with chronic pain exists, especially in the wider community. There is a dearth of studies that address gender differences in people with chronic pain with respect to psychological consequences such as depression (Novy et al., 1996). The few published studies, which have mentioned gender differences, are not consistent. Some limited evidence suggests that females are more likely to develop depression as a direct result of pain whereas for males, depression is likely to be more related to impairment of daily activities by pain than by pain itself (Haley et al., 1985).

The first study of this research will be presented in Chapter 4. Hypotheses will not be tested as it is essentially a pilot study conducted to test instruments and determine useful hypotheses for testing with a larger sample. It was also hoped to reduce the number of

variables in order to create a coherent model suitable for testing in accord with a cognitive-behavioural approach to the study of pain.

CHAPTER FOUR

STUDY 1: TESTING THE INSTRUMENTS AND DETERMINING HYPOTHESES

4.1 Introduction

This first study is by nature exploratory, given the paucity of information available about the psychological health of people with chronic back pain from the wider community. It is clear from the literature review that the knowledge base about the relationship between chronic pain, depression and associated demographic and cognitive-behavioural correlates is chaotic. According to the literature, a cognitive-behavioural perspective has the best research support in terms of testable models.

When the present research was initiated, the only empirical demonstration of a testable model, seeking to explain the relationship between chronic pain and depression, was the 'cognitive-behavioural mediation model', reported by Rudy et al. (1988). These researchers concluded that chronic pain and depression are mediated by two specific cognitive constructs, which they called 'perceived interference' and 'perceived life control'. However, the literature review has also revealed that there are several other correlates worthy of investigation within a similar context.

These include self-efficacy, activity, social support, anger and anxiety. In addition, they, along with perceived pain severity, depression, interference and control, represent sensory, cognitive, behavioural and affective aspects of chronic pain. These variables have been recognized as fundamental to explaining chronic pain and depression from a multi-dimensional, cognitive-behavioural perspective. Therefore, the present research represents a comprehensive investigation into cognitive-behavioural functioning in people with chronic pain. It also partially replicates the Rudy et al. (1988) research by developing and testing a cognitive-behavioural model that might more readily explain the relationship between chronic pain and depression in non-clinical people with chronic back pain.

It should be noted that throughout this thesis, when constructs such as 'perceived pain', 'depression', 'anxiety' 'interference' and 'control' are mentioned they will not be placed in quotes unless the placement of these names in the text might cause confusion or where they relate directly to a specific study, in which case quotes will be used.

4.2 Design

Most chronic pain research is of a cross-sectional design, with a few longitudinal studies. The latter is considered more desirable, given that 'time' is a key element in chronic pain, but is often more difficult to implement. This study is the first of three studies spanning a period of five years. This design allows for both cross-sectional and longitudinal analyses, which may prove useful in disentangling the association between chronic pain and psychological consequences.

The design of the present research uses correlations, t-tests, multiple regressions and path analysis techniques. Correlations are used to determine which significant variables are suitable to be entered into multiple regressions to determine those that might better predict the outcome variable, depression. The t-tests are used to compare groups, such as high versus low risk depressed, pain versus no-pain and so forth. Path analysis extends regression analysis and provides additional information by using standardized regression coefficients to estimate the strength of hypothesized causal pathways between variables.

4.3 Method

As mentioned, this study is a pilot or exploratory study, designed to test instruments and determine relevant hypotheses in a small group of people with chronic pain. This was considered a necessary preliminary step to determine the extent to which this type of research is feasible, given the physical limitations experienced by people with chronic back pain. Chronic back pain is physically challenging and demanding and initial discussions with participants indicated that to sit for any length of time to be interviewed or to complete

questionnaires might be too difficult. The interviews were important to develop rapport with participants as many people with chronic pain appear reluctant to discuss their condition due to the social stigma they perceive to be associated with it.

4.3.1 Participants

The participants were 30 (8M; 22F) people with chronic back pain, who met the inclusion criteria. These were as follows: a) aged 18 years or over; b) English speaking; c) upper and/or lower back pain for most of each week (i.e. on 4 days out of 7) for the previous 6 months or more; d) not presently attending and have not attended a pain clinic or medical pain management program; e) not experiencing chronic pain in multiple body sites; f) no other diagnosed major medical or psychiatric disorder and g) pain condition not related to worker's compensation/litigation process. The final participants ranged in age from 23 to 75 years with a mean age of 47 years, 8 months (SD=9 years, 2 months).

4.3.2 Measures

Participants were first interviewed in person by the author using the Structured Interview Guide for Chronic Pain Patients (Nicholas, 1994; Appendix A). This schedule consists of 32 items that can be modified where appropriate. This was considered most appropriate for the author who was a trainee psychologist at the time. The interviews were also audio-taped with the permission of each participant. Three people asked not to be taped. After the interview, participants were given a packet of questionnaires (stapled together to make one document for ease of administration, postage and scoring) to take away and complete at their own leisure. The first of these was the Patient Information Questionnaire (Appendix B), which is a modified version of that used in the Royal North Shore Hospital and University of Sydney Pain Management and Research Centre. The remainder of the questionnaires in the packet were used to measure the variables of interest. 'Variables' in this context refer to specific constructs such as 'depression', 'pain severity', 'pain self-efficacy' and the like, commonly discussed in the literature. These are

not tangible or observable entities but refer to scores on self-report instruments, which, generally, have been widely used and are reliable and valid. Most of the questionnaires used in this research are either protected by copyright or are in the public domain. They are only included in the Appendices of this thesis if they are in the latter category but not known by the author to be freely available.

In the following sections, the questionnaires and the relevant variables are described.

The variables[4] assessed are:

1) demographics (PIQ)
2) pain severity
3) pain duration
4) interference
5) life control
6) social support
7) punishing, solicitous and distracting responses;
8) general activity
9) pain self-efficacy
10) anger (state, trait, suppressed, expressed)
11) anxiety (state, trait)
12) depression.

(i) Patient Information Questionnaire (PIQ)

This is a six page questionnaire modified from a similar measure used by the Royal North Shore Hospital and University of Sydney Pain Management and Research Centre (1994). It is used to assess demographic information including gender, age, birthplace,

[4] Internal health locus of control and general self-efficacy measures are not introduced until Study 2 for the following reasons. In the Study 1 interviews, many participants expressed beliefs that they were at the mercy of 'fate' or medical professionals in terms of their pain experiences and treatment. Some also expressed little confidence in their abilities to manage daily living or their pain condition. A subsequent literature review undertaken after Study 1 had been conducted revealed that the two most relevant constructs related to these issues were 'internal health locus of control' and 'general self-efficacy'. It was decided that these issues warranted further investigation and so relevant questionnaires were introduced into Study 2. These constructs were discussed in the literature review and the questionnaires will be detailed in the Measures section, 5.3.2, Study 2.

marital status, children, living status, education level, occupation, employment status, pain history and medication usage (see Appendix B for information on direction and range of scores for each variable).

(ii) Multidimensional Pain Inventory (MPI)

The Multidimensional Pain Inventory (MPI: Kerns et al., 1985) is used to measure several of the variables of interest (see underlined in following section/s). It is a self-report measure consisting of three parts comprising a total of 60 items, which produce 13 empirical scales. These are prefaced by one item assessing pain duration, "When did your pain first start?" with provision for month and year. The first and second parts of the questionnaire relate directly to psycho-social factors, such as appraisals of pain, the extent to which pain has impacted on various domains of their lives and how they perceive that significant others respond to their suffering and distress. The third part relates to behavioural consequences of pain, and measures the frequency of general activities, which are or are not performed due to the pain. Ratings on each scale item range from 0 to 6. These end-points vary from 'not at all', 'no change', 'never' type statements to 'extreme change', 'very often' type statements, respectively.

Part 1 specifically measures a) reports of pain severity and suffering; b) perceptions of the extent to which pain is an interference in daily life, ie. including family, marital, work, social functioning; c) level of satisfaction with functioning in those areas listed in b); d) perceived life control (perceived problem solving ability plus feelings of confidence); e) appraisal of perceived social support (spousal, family and/or significant other); and f) affective distress, i.e. depression, tension and irritability. Further details of these measures are as follows:

Pain severity is measured with 3 items that assess severity of pain during the past week, the amount of suffering experienced due to pain and the current pain severity.

Interference is a composite of 11 items relating to "interference in family and marital function, work and social-recreational activities and satisfaction with present levels of

functioning in each of these areas" (Rudy, 1987, p. 38). There are 2 versions of this scale, one with 9 items and one with 11 items. In the present research, the latter scale from MPI: Version 2 is used and is treated as a combined scale.

Control is assessed by the life control sub-scale which measures perceived control over the pain condition and life events, including ability to solve problems as well as "feelings of personal mastery and competence" (Kerns et al., 1985, p. 347). The scale is a composite of 4 items.

Social support sub-scale consists of 3 items assessing how the person with chronic pain appraises "the amount of support received from a spouse or significant other" (Rudy, 1987, p. 38).

Part 2 is completed only if the person with chronic pain has a 'significant other' and consists of 3 scales derived from 14 items. The scales assess punishing (ignores, expresses irritation, frustration or anger), solicitous (gives food, medicine, takes over chores, asks how can help) and distracting responses (encourages diversions, e.g. hobbies). (In the literature, the former has sometimes been referred to as 'negative' social support while the latter may be referred to as 'positive' social support).

Part 3 consists of 5 scales, the first 4 measuring household chores, outdoor work, activities away from home and social activities. The fifth scale, general activity, is based on these four scales including a total of 18 items assessing the extent to which the person with chronic pain engages in typical activities from several domains. These include 1) household tasks (5 items); 2) outdoor work (5 items); 3) activities which occur away from the home (4 items) and 4) social activities (4 items).

Over the last decade, the MPI has become one of the most widely used assessment tools for chronic pain (Keefe et al., 1992). The MPI was specifically designed to measure perception of psycho-social and behavioural consequences of pain and is "theoretically linked to a cognitive-behavioural perspective" (Kerns et al., 1985, p. 346). The scales of this instrument have been reported as reliable, "with coefficient alphas greater than or equal to 0.70 and/or test-retest correlations of greater than or equal to 0.65" (Rudy et al., 1988, p.

131). In terms of external construct validity, the MPI has been found to correlate with other "well-known and established questionnaires" (Kerns et al., 1985, p. 348), such as the McGill Pain Questionnaire (MPQ: Melzack, 1975), the BDI, the Depressive Adjective Check List (DACL: Lubin, 1965) and the Multi-dimensional Health Locus of Control scale (MHLC: Wallston & Wallston, 1978). There is a substantial amount of psychometric support for these scales, including some normative data (Rudy, 1987).

(iii) Pain Self-Efficacy Questionnaire (PSEQ)

This instrument developed by Nicholas (1989) is used to assess self-efficacy related to pain beliefs. The PSEQ is a relatively brief, easy to administer instrument of 10 items reflecting ten different activities that are often difficult to perform for people who suffer chronic pain. Respondents are reminded in each item that they are required to indicate their confidence level in performing the particular behaviour, *despite the pain*. For example, item 1 states "I can enjoy things, despite the pain". The items each have a rating scale ranging from 0 (not at all confident) to 6 (completely confident). A total score is calculated by summing the scores for all ten items with a possible maximum of 60.

The PSEQ has been tested mainly with people with chronic pain and chronic low back pain prior to admittance to pain management programs, in both the UK and Australia. Nicholas (1994) reports that the PSEQ is reliable with a Chronbach's coefficient alpha of 0.92, with item-total correlations ranging from 0.67 to 0.84. A test-retest correlation of 0.79 has also been demonstrated. In terms of validity, Nicholas (1994) points out that the literature lacks any 'gold standard' measure with which the PSEQ can be compared. However, it would be expected that the PSEQ would correlate highly with measures of activity, disability measures, pain coping measures and measures of other pain beliefs. Consistent with this the PSEQ has been found to have a significant inverse correlation with the Sickness Impact Profile (SIP: Bergner, Bobbitt, Carter, & Gilson, 1981), the BDI and the State-Trait Anxiety Scale (STAI: Spielberger, Gorsuch, & Luschere, 1970). It has also correlated significantly in a positive direction with the Coping Strategies Questionnaire score

(CSQ: Rosenstiel & Keefe, 1983). Correlations between the PSEQ and measures of somatic perception would be expected to be weaker given that they tap different domains. According to Nicholas (1994), the PSEQ has not correlated significantly with the MPQ or average pain ratings used in chronic pain research. Gibson and Strong (1996) used the PSEQ to assess people with chronic back pain involved in rehabilitation. They reported the PSEQ as reliable with a Chronbach's coefficient alpha of 0.94, and correlations between the PSEQ and perceived capacity for work-related tasks of r = 0.78 and another self-efficacy measure as r=0.63. Asghari and Nicholas (2001) reported a Chronbach's alpha of 0.92 in a study with 145 chronic pain patients. Nicholas et al. (1992) and Williams et al. (1999, 1996) have also reported that the PSEQ is a sensitive measure, for example, to treatment effects when disability levels were reduced and functional activities improved. The PSEQ can be seen in Appendix C.

(iv) The Spielberger Anger Expression Inventory (STAXI)

The STAXI is a 44-item questionnaire developed by Spielberger et al. (1985). It is divided into 3 sections; 1) how I feel right now, 2) how I generally feel and 3) [how I feel] when angry or furious. Each section contains statements that individuals might use to describe their feelings and behaviour at these times. Section 1 and 2 assess state anger and trait anger respectively. Section 3 yields four sub-scales. "Anger In" assesses the extent to which angry feelings are held inside or suppressed. "Anger Out" measures how often anger is expressed toward the external environment i.e. people or objects. "Anger Control" measures the extent to which a person might try to control expressed anger. "Anger Expression" is a measure of the degree to which angry feelings are expressed, irrespective of the direction of expression i.e. internally or externally (Duckro et al., 1994).

In the present research, only state, trait anger and the anger in and the anger out sub-scales are used. State anger is measured by 10 items that assess "how I feel right now". Trait anger is measured by 10 items that assess "how I generally feel". Anger In is measured by 8 items which reflect the degree to which angry feelings are felt but not

expressed (e.g., "I keep things in"; "I boil inside but I don't show it"), while the 8-item Anger Out sub-scale assesses how often feelings of anger are expressed as aggression (e.g., "I make sarcastic remarks to others"; "I do things like slam doors"). Responses to the 44 items range from 1 (not at all or almost never) through to 4 (very much so or almost always). The first response example here is used in part 1 while the second is used in parts 2 and 3. The total score may range for each section from 10 to 40 (parts 1 & 2) and 8 to 32 for each sub-scale in part 3. Higher scores in parts 1 and 2 indicate higher state and trait anger. Higher scores in part 3, for anger in and anger out, indicate higher suppressed anger and higher expressed anger, respectively.

Adequate internal consistency coefficients for the STAXI have been reported as well as evidence of the validity of the anger scales with respect to other personality and anger measures (refer to Spielberger et al., 1985, for full details). Alpha coefficients of .93 are reported for state anger while alpha coefficients for trait anger have ranged from .70 to .89. Moreover, alphas for the anger in and anger out sub-scales range from .73 to .84. Other researchers have reported on the STAXI's validity in various health related research (Schneider, Egan, Johnson, Drobny, & Julius, 1986; Vogele, 1993).

(v) The Spielberger State-Trait Anxiety Inventory (STAI)

The STAI, developed by Spielberger et al. (1970), comprises two 20-item scales designed to assess both state and trait anxiety. The state anxiety scale reflects the extent that one feels anxious in the present moment, while the trait scale measures a person's propensity to experience feelings of anxiety on a general level across a diverse range of situations. The scales are relatively brief and easily administered. Responses to the 20 items on each scale are made on 4-point scales that range from 1 (not at all) to 4 (very much so) for the state sub-scale and 1 (almost never) to 4 (almost always) on the trait sub-scale. The item scores are summed and range from 20 to 80 with higher scores indicating higher anxiety on both sub-scales. This test is widely used and reported to demonstrate "good normative and psychometric data" (Prowse & Wilson, 1992, p. 75).

(vi) The Center for Epidemiologic Studies Depression Scale (CES-D)

The CES-D is a 20-item self-report instrument designed to assess six different components of depression (Radloff, 1977). These are identified as: depressed mood, feelings of guilt and worthlessness; feelings of hopelessness and helplessness; psychomotor deficits; poor appetite and sleep disturbances. Four of the items are reversed in order to assess positive affect as well as to avoid response set. Subjects are asked to indicate how often these depressive symptoms occurred in the previous week. These responses range on a four-point scale from 'rarely or none of the time' (score=0; less that 1 day) to 'most or all of the time' (score=3; 5-7 days). The scoring is opposite for the four reversed items. The CES-D score may range from 0 (i.e. no depression) to 60 (i.e. maximum depression), with severity indicated by increased numbers of symptoms endorsed and greater frequency or duration.

This instrument was specifically designed for community survey research (Plutchik & Conte, 1989). It was designed to assess depressed mood in non-clinical groups, rather than as a clinical diagnosis tool such as the BDI. The CES-D is considered to be "the most generally useful self-report test" (Turk & Okifuji, 1994, p. 11) in comparison to other tests such as the BDI, the Zung and the MMPI Depression Scale (Plutchik & Conte, 1989). It has also been widely used to assess depressed mood in chronic pain patients (Fishbain et al., 1997). It has been reported that the validity of the CES-D is not seriously threatened by physical impairment, such as is typical of chronic pain (Berkman, Berkman, Kasl, Freeman, Leo, Ostfeld, Cornoni-Huntley, & Brody, 1986). As the CES-D was developed especially for non-psychiatric patients, it is considered to be more useful than the BDI for those in chronic pain as it contains few items dealing with somatic symptoms. This is particularly relevant for chronic pain patients who may experience physical problems that are not necessarily related to depression.

There is some debate over the optimum cut-off point, which relates to the sensitivity and specificity of the measure (Fechner-Bates, Coyne, & Schwenk, 1994). 'Sensitivity' refers to cases that score above a specified cut-off and are correctly identified as

depressed. 'Specificity' relates to those who score below the cut-off point and are correctly identified as non-depressed. Sensitivity is considered more important as it is better to 'err on the side of caution' and label someone as depressed when they are not rather than vice versa (Turk & Okifuji, 1994). While the 'specificity' of the CES-D has been reported as somewhat low, the more important 'sensitivity' appears to be comparable to other instruments used with people with chronic pain. While a score of 16 has been suggested as a general cut-off score for clinical depression (Radloff, 1977), for subjects with physical conditions that may confound the score, a cut-off point of 19 is indicated (Turk & Okifuji, 1994). Other researchers have also suggested both 16 and 20 as cut-off scores (Magni et al., 1994; Murrell, Himmelfarb, & Wright, 1983). In the present research, scores of 19 or more are used as the cut-off point in order to distinguish between high and low risk scorers. However, in the statistical analyses, the whole range of scores is used, except in comparison tests between high and low risk scorers.

While there is some criticism that use of such self-report depression measures may result in an increase in false-positive diagnoses of depression (i.e. diagnosed as depressed when not), the CES-D reportedly discriminates well between false and true positives (Turk & Okifuji, 1994). The CES-D is reported as a valid measure with high internal reliability, with Chronbach's alpha ranging from 0.85 to 0.90 (Radloff, 1977), and 0.85 to 0.91 (Himmelfarb & Murrell, 1983). Moreover, split-half reliability has been reported as ranging from 0.76 to 0.85 (Radloff, 1977). It has reported high sensitivity for detecting depression in diverse groups, such as alcoholics, schizophrenics, stroke patients and primary care patients (Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977). It has also been found to be reliable and valid specifically with chronic pain subjects (Brown, 1990; Turk & Okifuji, 1994; Turner & Noh, 1988), people with arthritis (Schiaffino & Revenson, 1995) and the elderly (Turner & Noh, 1988). The CES-D also compares favourably with other depression measures such as the DACL (Radloff, 1977). It has also been found to correlate well with depression diagnoses based on DSM-III criteria, with correlations about 0.77 (Parikh, Eden, Price, & Robinson, 1988; Shinar, Gross, Price, Banko, Bolduc, & Robinson, 1986).

4.3.3 Procedure

While chronic back pain seems to affect a high proportion of the general public, acquiring people to take part in the research was difficult. It was not possible to obtain a demographically accurate random sample of people from the chronic back pain population due to time and resource constraints. Volunteers were sought in as many ways as possible in metropolitan Adelaide. Posters (Appendix D) were placed in public places such as shopping centres, public transport boarding platforms, university and college campuses and advertisements were placed in a regional community newspaper, *The Messenger.* These advertisements asked for volunteers who suffered from long-term or persistent back pain to take part in questionnaire research. Overall, however, the response was poor and it was felt that this was possibly due to the time of year that volunteers were sought, i.e. just before Christmas.

Fifty-eight people made contact but 18 were eliminated as they failed to meet the inclusion criteria (refer to Participants, Section 4.3.1). The reasons for exclusion were: had been attending or were being referred to a pain clinic (5); were being treated for a major psychiatric disorder (4, i.e. anxiety=1; depression=3); or had multiple (neck, shoulder, back, knees) chronic pain sites (9). Five, although willing, were constrained by their lack of mobility or resources and subsequently could not be contacted by telephone or mail service. Of the 35 people who did meet the inclusion criteria and were interviewed, 5 failed to complete or did not return the questionnaires. These 5 people were involved in work-related litigation at the time and despite their interest in the research, expressed considerable concern about anonymity and confidentiality during the interview. Although they were assured of both, they failed to provide completed questionnaires and did not respond to two rounds of follow-up letters.

Most of the final 30 participants were either interviewed in their own homes, if they had mobility problems, or at Adelaide University. These interviews were part of a process to develop rapport with the participants and the resulting transcriptions represent qualitative data not specifically addressed in this thesis, although some of the information may be

presented elsewhere. Prior to the interview, each participant was supplied with a participation consent form and an information and instruction sheet (Appendix E). They were also provided with an anonymity number to maintain confidentiality. Upon completion of the interview they were provided with the remainder of the questionnaires to complete at leisure and return by post.

Data were analysed using the Statistical Package for the Social Sciences (SPSS) version 9.0 for Windows. Scales from the MPI (Kerns et al., 1985) were computed via the MPI program which is a "MS-FORTRAN77 (Version 4.10) microcomputer program designed to read [MPI scores], compute raw scale and T-scores on the 13 [MPI] scales and [to also..] compute multivariate profile classification statistics based on the University of Pittsburgh Multiaxial Assessment of Pain (MAP : Rudy et al., 1989; Turk & Rudy, 1986; Turk & Rudy, 1988) empirically derived taxonomy system" (Rudy, 1987, p. 1). According to a recommendation by Rudy (1987), version 2 was used for the present study. Only the raw scale scores for the relevant MPI variables are used for the SPSS analyses. Analyses of all other questionnaires are also based on raw score values.

4.4 Results

4.4.1 Analyses and assumptions

The variables in this thesis were assumed to be measured at interval level, even if they were ordinal. Such variables are routinely treated as interval variables in the social sciences (Bryman & Cramer, 1997). According to Labovitz (1970, cited in Bryman & Cramer, 1997), the small amount of error that might occur is generally outweighed by the advantages of being able to use parametric tests, such as correlation and regression, which are more powerful than non-parametric tests.

Several of the variables in this research also have skewed distributions. Many research psychologists argue that psychological variables are inherently likely to be skewed. Some suggest that non-parametric tests are therefore more suitable for use with such variables. However, Bryman and Cramer (1997) argue that, with psychological

variables, such as beliefs, parametric tests often produce results similar to that produced by the less powerful non-parametric tests, despite a skewed distribution[5]. Therefore, in the analyses presented in this thesis, parametric tests are the statistics of choice, unless stated otherwise.

Power analysis was conducted wherever appropriate throughout the statistical analyses of this thesis (refer to Cohen, 1992, for more detail). This involved calculating effects sizes (ESs) for all samples used in calculations, to determine the adequacy of sample sizes. All tests are conducted with alpha set at 0.05, unless otherwise specified. The main tests used in this thesis, for which ESs are calculated are t-tests, correlations and multiple regressions. The ES index for each of these is *d*, *r*, and f^2, respectively and they are calculated according to Cohen (1992). Small, medium and large ESs for each of these are [*d*] .20, .50, .80; [*r*] .10, .30, .50 and [f^2] .02, .15 and .35, respectively. When an effect is large, fewer cases are required to show it compared to a small effect size, which requires a greater number of cases to be demonstrated (Cohen, 1992). Cohen (1992) also provides a table of Ns for small, medium and large ESs at Power = .80, for alpha set at .01, .05 and .10. These were referred to where appropriate.

Table 4.1 shows demographic and pain related variable responses for N=30. Although examination of gender differences represents one of the aims of this research, this pilot group was considered too small to be able to do this, as there were only 8 males, therefore gender differences are not examined.

[5] Tabachnik and Fidell (1989) suggest that with skewed distributions the scores can be inverted and square rooted to try to improve the shape of the distributions for later analyses. This was attempted with the variables in this research, but in no case was the shape improved and in the case of some, i.e. depression, it was more skewed. The variables were therefore retained in their original form.

Table 4.1. Demographic and pain-related characteristics of the chronic back pain participants (N=30).

Demographic characteristics	Chronic Back Pain participants (N=30)	
	n	%
Gender		
Male	8	26.6
Female	22	73.3
Marital Status		
Married/Defacto	20	66.7
Single	10	33.3
Education		
≥Year 12	14	46.7
<Year 12	16	53.3
Employment Status		
Full-time	6	20.0
Part-time/volunteer	3	10.0
Unemployed	10	33.3
Retired/home-duties	11	36.7
Drug use		
Prescription(including nsaids°)	16	53.3
Otc°° (incl. nsaids)	8	26.6
No drugs	6	20.0
Back operations		
No	23	76.7
Yes	7	23.3
	Mean	**SD**
Age (years)	47.67	9.18
Pain Duration	13.2	9.0

°non-steroidal anti-inflammatory drugs; °°over-the-counter

As can be seen from Table 4.1, this group of participants was predominantly female (n=22, 73.3%) and aged from 23 to 75 years (m=47.7; sd=9.2). Pain duration ranged from 1 year, 7 months to 34 years (m=13.2; sd=9 years). Almost half of the participants had been educated to year 12 or more. Twenty were currently married or living with a partner, while the other 10 were living alone as single, divorced, separated or widowed persons. Only 6 were employed full-time, 3 worked part-time or volunteered, while 11 were retired or performing home duties. Most of them had not had back-related surgery.

When asked about drugs, 14 were taking mainly *prescription* drugs for pain on a regular basis. These included narcotic analgesic drugs (e.g. panadeine forte, capadex, digesic, mersyndol forte) and nsaids or non-steroidal anti-inflammatory drugs (e.g. voltaren, brufen, naprosyn) taken on a regular basis. This varied from routinely at night before bed to 8 tablets a day to only 'as needed. Ten people were mainly taking *over the counter* analgesic drugs (e.g. panadeine, mersyndol, aspirin) and/or nsaids (e.g. nurofen) when needed. Of the 30 participants, 7 took medication as needed, while the rest took it daily (6, once a day; 4, twice a day; 2, 4 times a day and one person took medication 8 times a day). Six reported that they took neither prescription or over the counter drugs for pain because they avoided all drugs. If needing urgent relief these 6 people all said they used some type of anti-inflammatory cream but only if absolutely necessary, preferring to use hot packs, spas, hot baths or showers to provide immediate relief. Fourteen of the participants used physiotherapy, chiropractic treatment, massage and/or acupuncture on a regular basis (i.e. 8 used one of these about once a month and 5 reported that they used one of these occasionally, by which they meant less than once a month) to relieve pain.

Results from the assessment of cognitive-behavioural functions can be seen in Table 4.2. Means and standard deviations are reported for the sample and are compared to normative samples. A t-test was conducted for each variable to determine if the responses from the present research sample differed significantly from responses by other samples of pain subjects or samples of normal adults (Bryman & Cramer, 1997). While two sample or unrelated t-tests may be more appropriate here, the available normative data did not

provide sufficient information to run such tests. That is, only the means and standard deviations were available, not the individual scores, so unrelated sample t-tests are not appropriate. However, under such circumstances, it is appropriate to use the one-sample t-tests (Bryman & Cramer, 1997; pers. comm. BW). The statistics package (SPSS) has the facility to carry out one-sample t-tests when only the means and standard deviations of normative data are available. Scores for the pain related responses are compared to normative data obtained from Rudy (1987) and are based on a sample of 150 chronic low back pain patients. The pain self-efficacy norms are based on previous research with 103 chronic low back pain patients (Nicholas, 1994). The anger and anxiety scores are compared to normative non-pain data obtained from Spielberger (1988) and Spielberger et al. (1970). These are based on two United States working adult samples of and 4,062 and 1,838, respectively. The normative data for the CES-D was obtained from Radloff (1975) and based on a randomly selected community sample (N=1,173). The normative sample sizes are indicated in the table.

Table 4.2. Means and standard deviations of cognitive-behavioural functioning in chronic back pain participants compared to normative data from pain samples and non-pain samples.

Variables	Chronic Back Pain participants (N=30)		Normative Data Pain Samples		Normative Data Non-Pain Samples			
	Mean	SD	Mean	SD	Mean	SD	t	d
			(N=150)					
Pain Severity	3.77	1.13	4.64	1.00			-4.23***	.85
Interference	4.16	1.13	4.79	0.98			-2.84**	.62
Perceived Control	3.42	1.03	3.09	1.52			1.73	.22
Social Support	3.78	1.64	4.59	1.43			-2.72*	.55
Punishing Rs	1.55	1.31	1.72	1.62			-0.71	.11
Solicitous Rs	2.98	1.59	3.61	1.55			-2.10*	.40
Distracting Rs	1.63	1.27	2.47	1.46			-3.60**	.59
Activity	2.41	0.93	1.96	0.91			2.65*	.49
			(N=103)					
Pain self-efficacy	30.13	11.99	25.80	12.40			1.98	.35
					(N=4062)			
State Anger	13.43	4.92			12.04	4.00	1.54	.34
Trait Anger	18.67	5.63			19.05	4.96	-0.37	.07
Suppressed Anger	17.57	5.18			15.53	4.08	2.15*	.50
Expressed Anger	14.33	3.35			14.67	3.68	-0.55	.09
					(N=1838)			
State Anxiety	44.73	14.56			35.96	10.79	3.30**	.81
Trait Anxiety	43.97	15.06			35.04	9.10	3.61**	1.07
					(N=1173)			
Depression	17.60	10.88			9.92	9.32	3.87**	.82

*p<0.05; ** p<0.01; ***p<0.001; d=Cohen's d (the standardized difference between 2 group means).

Table 4.2 shows that respondents in this study scored significantly less than the normative pain sample on perceived pain severity, interference and social support. As can be seen in the table, the ESs for these were large (Cohen, 1992). They reported

significantly less solicitous (small to medium ES) and distracting responses (medium ES) and significantly more activity (medium ES) than the norm. They reported more control and pain self-efficacy (small ES) than the pain sample, although these differences were not significant. Twenty-two respondents (73%) scored below 39 on the PSEQ, the recommended cut-off score for low pain self-efficacy (Nicholas, 1997).

The respondents did not differ from the norm on anger responses except for suppressed anger (medium ES), which they reported was significantly greater. Also, they appeared to be significantly more anxious (large ES) and depressed (large ES) than normal as 14 respondents (47%) scored 19 or more on the CES-D.

With respect to effect sizes, it can be seen that the pain group, compared to the normative pain group, was significantly less dysfunctional on six of the pain measures. That is, they reported less pain, interference, solicitous and distracting responses and more activity, although they also reported less social support. Data also shows that this group suppressed significantly more angry feelings and was significantly more anxious and depressed than those without pain.

4.4.2 Exploring the data

To begin with, correlations (see Table 4.3) were used to establish the degree of inter-relationship between the variables. Significant correlations are shown in bold type. A legend showing abbreviations used in the correlation matrix, is presented in Table 4.4.

Table 4.3 Matrix of Pearson's product moment correlations among cognitive variables for chronic back pain participants (N=30).

	PSv	Int	LC	SS	PR	Sol	DR	Activ	PSE	SAng	TAng	AngIn	AngOut	SAnx	TAnx	Dep
PSv		**.68*****	-.12	**.49****	.28	.28	.20	-.13	**-.64*****	.33	**.41***	.34	-.11	.27	.19	.31
Int			-.19	.32	.28	.13	.18	-.34	**-.66*****	.32	**.51****	**.47****	.01	**.51****	**.43***	**.44***
LC				-.07	**-.36***	-.11	.06	.33	**.37***	-.01	-.20	-.36	-.04	**-.54****	**-.48****	**-.49****
SS					.17	**.79****	**.61****	.25	-.17	.22	.30	.05	.16	.21	.03	.21
PR						-.03	-.07	-.07	-.17	.27	-.02	**.37***	.09	.18	.25	.25
Sol							**.64****	.27	-.13	.02	**.42***	.05	.12	.04	-.11	.03
DR									-.28	.19	.25	-.12	.25	.13	.03	.24
Activ									**.37***	-.22	-.03	.05	.01	**-.48****	**-.41***	**-.52****
PSE										**-.40***	**-.43***	-.35	-.07	**-.53****	-.33	**-.47****
SAng											.15	**.37***	.09	**.46***	.32	**.43***
TAng												**.36***	**.45***	.15	.18	.08
AngIn													-.24	**.44***	**.43***	.18
AngOut														-.10	.04	.05
SAnx															**.69*****	**.75*****
TAnx																**.63*****

*** Correlation is significant at the 0.001 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 4.4. Legend of abbreviations used for variables in Table 4.3.

Abbreviation	Variable
PSv	Pain Severity
Int	Interference
LC	Life Control
SS	Social Support
PR	Punishing Responses
Sol	Solicitous Responses
DR	Distracting Responses
Activ	Activity
PSE	Pain self-efficacy
SAng	State Anger
TAng	Trait Anger
AngIn	Suppressed Anger
AngOut	Expressed Anger
SAnx	State Anxiety
TAnx	Trait Anxiety
Dep	Depression

In a correlation matrix, *r* represents the effect size and small, medium and large ESs are .10, .30 and .50, respectively. As the main focus of this research is the relationship between chronic pain and depression, the correlation between chronic pain, as measured by pain severity, and depression, is of interest. For this group of 30, the correlation is positive suggesting that higher pain severity is associated with greater depression, although the correlation is not significant (r=0.31). There are other noteworthy correlations between these key variables and others in the matrix. For example, pain severity is significantly and positively correlated with interference, social support, and trait anger while significantly and negatively correlated with pain self-efficacy. These results suggest that increased pain levels are related to perceiving that pain is a great interference, more social support, a greater proneness to anger and less self confidence in dealing with pain. Likewise, depression is significantly and positively correlated with interference, state and trait anxiety and negatively correlated with life control, activity and pain self-efficacy. This suggests that increased depression is associated with greater perceived interference, increased state anxiety and proneness to anxiety, reduced feelings of control and confidence in dealing with

pain and lower activity levels. It can also be seen that the ESs are mainly large, indicating that this sample size is adequate to provide reasonable effect sizes.

In terms of other correlations, Table 4.3 demonstrates that there is a high level of inter-relatedness between particular pairs of clusters of variables. For example, social support is closely related to solicitous responses (r=0.79). There are broader clusters also, which is expected given that most of these variables have been shown to be important in the chronic pain-depression relationship. It is also clear that, apart from depression, pain self-efficacy and state anxiety correlate most with other variables across the matrix.

As depression is the main outcome variable of interest, a linear multiple regression was performed to determine which of the significant variables might be accounting for most of the variability of the depression scores. Some authors recommend that there should be at least 10 cases per variable entered into a multiple regression (especially with the stepwise procedure, see below) (Tabachnik & Fidell, 1996)[6]. With a sample of 30 participants only 3 variables can reasonably be entered into the regression equation. Therefore, only those 3 variables with the highest correlations with depression were chosen. These were state anxiety, trait anxiety, and activity. The stepwise procedure for linear multiple regression was adopted as this procedure is recommended by Bryman & Cramer, 1996. However, according to Bryman and Cramer (1996), its use is controversial as it only enters independent variables into the equation if they meet specified statistical criteria and some researchers maintain that theoretical criteria should take precedence over statistical criteria. This controversy is noted and the author opted for the stepwise procedure because it does emphasise statistical priority. To implement this technique the variable with the highest correlation to the dependent variable would be entered on the first step and so forth. Order of entry is determined by the degree of contribution that each variable makes to the explained variance in the dependent variable (Bryman & Cramer, 1997).

[6] It should be noted that other advice (Winefield, 1998) indicated that this regression could be done with 6 variables. When this was carried out, the results were the same as with 3 variables (to 3 decimal places).

Table 4.5. Multiple regression analysis: Significant predictor of depression for chronic back pain participants (N=30).

Variable In	B	Beta	CI [95]	t	R^2	AdjR^2	F(1, 28)
Anxiety (state)	.56	.75	.37, .75	5.99***	.56	.55	35.96***

***p<0.001. Note: no further variable achieved a significant independent contribution to the regression (α=.05).

As can be seen from Table 4.5, state anxiety was the only significant contributor to the regression equation for depression, accounting for 56%[7] (55% adjusted; F(1, 28) =35.95, p<0.001). In addition, the effect size was large (Cohen, 1992; f^2[8]). All other variables were excluded from the analysis, as they did not contribute significantly over and above the variance already explained by state anxiety. Previous research has questioned the difficulty of clearly differentiating anxiety from depression in self-report inventories (Craig, 1995; Gulley & Nemeroff, 1993; Preskorn & Fast, 1993; Tyrer, 2001), so this finding is problematic. In statistical texts, the problem is called "multicollinearity" (Bryman & Cramer, 1997; Lewis-Beck, 1980). Moreover, experiencing anxiety when filling out forms might be considered a likely and usual response, especially by someone suffering persistent pain and depression.

The finding that pain severity and depression are positively related but not significantly so, suggests that depression is influenced by behavioural and cognitive reactions to pain, rather than directly by the pain severity itself. This is also consistent with Rudy et al.'s (1988) suggestion that mediating variables may be operating. However, the only variables which are significantly related to <u>both</u> pain severity and depression, a requirement for path analysis, are interference and pain self-efficacy.

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[7] When there are few degrees of freedom (i.e. small sample), the R^2 and Adjusted R^2 may be quite different and as, in such a case, one would be trying to adjust for chance effects, it is more appropriate to report the Adjusted R^2. On the other hand, with a larger sample size and therefore more degrees of freedom, then there is less likely that there will be much difference between R^2 and Adjusted R^2 and usually R^2 is reported. However, in Table 4.5, there is actually little difference (i.e. .56 or .55) therefore it is of little consequence.

[8] The effect size for a multiple regression is calculated as $f^2 = R^2/1 - R^2$.

Given that part of the focus of this thesis is on mediating variables, it is appropriate that these variables should be hypothesized to act as mediators and examined by path analysis. The result is depicted in a simple path diagram. The latter may give a picture of the relationship between chronic pain and depression, and the mediating contribution made by selected variables. Even though the sample is small, and caution should be used in interpreting results, the path diagram may prove illuminating.

To produce a path analysis model with data from this sample, three multiple regressions were performed[9]. In the first two equations each potential mediator (interference and pain self-efficacy) was predicted from the independent variable (pain severity). The resulting beta values from these first two equations are demonstrated on the left-hand paths of the path diagram to follow. In simple cases, such as this where there are only two variables involved, the regression or beta coefficients will be equal to the correlation values. In the third equation, the dependent variable (depression) was predicted from each potential mediator variable (interference, pain self-efficacy) and from the independent variable (pain severity)(see Baron & Kenny, 1986, for more detail). The resulting beta values from this third equation are depicted on the right-hand paths of the path diagram to follow.

[9] See Bryman and Cramer (1997) for full explanation of path analysis techniques.

Table 4.6. Multiple regression analyses for path diagram (N=30).

Predictor Variables	**B**	**Beta**	**CI [95]**	**t**	**R^2**	**AdjR^2**	**F (df)**
Equation 1 **DV: Interference**							
Pain severity	.74	.68	.44, 1.05	4.93***	.46	.45	24.31*** (1, 28)
Equation 2 **DV: Pain self-efficacy**							
Pain severity	-6.8	-.64	-10.00, -3.69	-4.44***	.41	.39	19.73*** (1, 28)
Equation 3 **DV: Depression**							
Interference	2.63	.30	-1.93, 7.19	1.19	.26	.17	3.03* (3, 26)
Pain self-efficacy	-.32	-.35	-.77, .13	-1.48			
Pain severity	-1.21	-.12	-6.11, 3.70	-.51			

*p<0.05, ***p<0.001; B = unstandardized coefficient; CI = Confidence Intervals.

The regression output which determined the path coefficients for depression showed that the three independent variables, interference, pain self-efficacy and pain severity together accounted for 17% of the variance ($F(3, 26) = 3.03$, $p<.05$), as shown in Table 4.7. The path diagram, Figure 4.1, illustrates the relationships. All path values in the diagram are beta values.

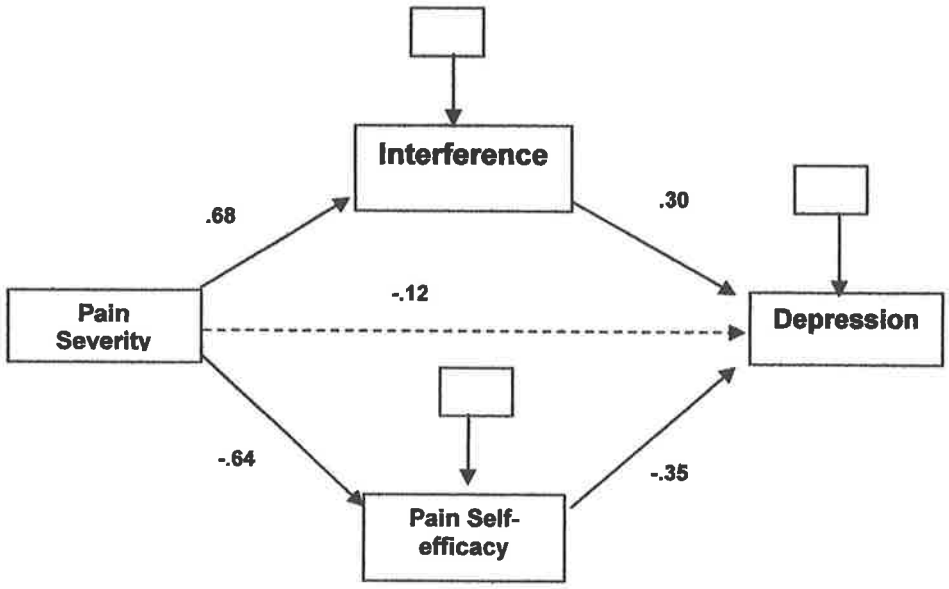

Figure 4.1. Cognitive-behavioural mediation model of chronic pain and depression (N=30).

The direct effect of pain severity on depression is negligible (-.12). The indirect possible effects of pain severity via increased interference to daily life and via reduced pain self-efficacy are small (.68*.30) = 0.20, and (-.64*-.35) = 0.22, respectively. Although some of the separate beta values are not significant, this does not indicate that there is no relationship nor that the model is not supported. If the R^2 of the overall model is significant (i.e. see Equation 3), then the model has some validity. Individual beta values reflect the unique contribution of variables while the R^2 reflects the overall significance of the model. The non-significant beta values merely indicate that it is difficult to disentangle the effects of some of the individual variables.

In this particular case, although the model is significant, the actual p value for F =3.03 was 0.047, which explains why the separate beta values in the final equation are not

significant. As this sample was small, there was concern that a Type II error could have been made. However calculations, according to Cohen (1992), showed that the effect size for this analysis (f^2) = 0.35, which is a large effect for this test, suggesting that the sample size was adequate.

It is also acknowledged that 'error variance' or the amount of variance not explained by the variables is present, as depicted in the path diagram, for interference, pain self-efficacy and depression. These error terms indicate that there are other, unknown, variables which contribute to the outcome and mediator variables but which are not represented in the path diagram (see Bryman & Cramer, 1997, for more detail). In this thesis, error terms are not used in predictions, therefore, as suggested by Bryman and Cramer (1997), they are ignored.

With further reference to the correlation matrix presented in Table 4.3, it can be seen that the original correlation between pain severity and depression was 0.32, but the direct effect, after allowing for the two mediating variables, has been reduced from 0.31 to -0.12. The beta values in the path diagram suggest that high pain severity may lead to increased perceived interference of pain in daily life and decreased pain self-efficacy which may then contribute to elevated depression.

4.5 Discussion

There are limitations to this first study. Although there was concern that the sample would be too small to make firm conclusions, given the difficulty of acquiring subjects, power analysis showed that this was not the case.

The basis of this first study was cross-sectional data which cannot support a cause-effect relationship possible with longitudinal data, but this is addressed with the later research. This study relied on the use of self-report measures which have been criticized for their subjectivity, implying that such measures lack scientific credibility and validity (Turner & Jensen, 1993). However, many researchers consider that self-report is valid especially with respect to pain intensity and levels of activity (White & Strong, 1992; Yang et

al., 1991). Furthermore, self-reports are relatively inexpensive to produce and easy to obtain. Despite continued debate, the self-report is generally accepted as an important measure of in pain research, especially given that there is no objective measure (Von Korff et al., 1992).

This study reports the investigation of a small group of, predominantly, females with chronic back pain. These participants were mostly middle-aged and had experienced chronic pain for a considerable period of time, about 13 years on average. The group was reasonably well educated and the majority did not live alone. Most of them did not work full-time and had not had surgery for their chronic pain condition. About half of this group were taking regular prescription medication for pain relief and about the same number regularly sought relief from allied health practitioners, such as physiotherapists and chiropractors.

The sample is described as a community sample. In this research, 'community sample' was defined as those who had not attended or were not attending a pain clinic. It is acknowledged that some of these people had sought help from health care workers etc., but were not considered severe enough by health care workers to be part of a formal treatment program or to be referred on to a pain clinic, therefore they were accepted as a non-clinical or a community group. The issue of how 'non-clinical' the sample will be referred to in later discussion of the representativeness of the study subjects.

There were limited relevant normative data with which to compare these people in terms of cognitive responses to pain. That is, the number of clinical studies with which they could be compared, in terms of measures used and sample characteristics, was small. Compared to other pain groups they reported less pain severity, interference and social support. Of the social support they did receive, they experienced less solicitous and distracting responses, at least compared to normative data from clinical pain patients. They also received less punishing responses, although this was not significantly so. As a group, they reported greater control and pain self-efficacy than other people with chronic pain, although, again, not significantly so. Even so, a high percentage (73%) scored low pain self-efficacy, which may be some cause for concern. These early results show that this

group differed on key measures to clinical patients. According to the literature, most clinical subjects are in the minority and do not represent the rest of the community (Elliott et al., 1999; Magni et al., 1994), so this is an interesting finding. They differed in directions that would be expected if they were not as severely affected by pain as those cases that are referred on by general practitioners for specialist treatment, i.e. pain severity and interference were low, while control and pain self-efficacy were high.

They did not differ on anger measures from non-pain subjects, except that they reported more suppressed anger. This is consistent with previous research, especially work by Hatch et al. (1991) and Kerns et al. (1994), that reported that headache patients and mixed chronic pain patients, respectively, reported more suppressed anger than did control non-pain subjects. The literature is not consistent about whether predominantly female groups are likely to report more suppressed anger than expressed anger, either in pain or non-pain groups (Okifuji et al., 1999a).

The sample was also significantly more anxious (state and trait) and more depressed than normal and a high percentage (47%) were classified as 'depressed'. As with anger, these were compared to non-pain subjects, due to a lack of strictly comparable clinical studies. According to previous research, those in chronic pain tend to be more anxious than among the general public (Asmundson et al. 1999; Asmundson & Taylor, 1996; Craig, 1994). The present findings are also consistent with previous research. For example, Magni et al. (1993) found that depression in the general population ranged from 6% in those without chronic pain to 16% among those with chronic pain, while Banks and Kerns (1996), concluded that depression was much higher in those with chronic pain, ranging from 30% to 54%.

Initial correlational analyses suggested that in this sample of participants, those who reported high pain levels were more likely to perceive their pain as a great interference in daily living. This is consistent with much of the previous research. For example, Turk and colleagues (Rudy et al., 1989; Turk et al., 1995; Turk & Rudy, 1990) reported that patients classified as 'dysfunctional' reported higher than average pain severity and were far more

likely to perceive that pain disrupted their daily lives than other people with chronic pain. Maxwell et al.'s (1998) findings also concur with this. Those with high pain were also more likely to perceive that they had considerable social support. This is consistent with the operant view, that more attention and sympathy may result in more pain because they reinforce the individual's pain expressions (Turk et al., 1992a). Although it might be expected that solicitous responses would also therefore be significantly correlated to pain severity, they were not, although they were in a positive direction. As most of the sample was female there could be gender effects, however, there is little information in the literature with which to compare this result.

A greater proneness to anger was also a feature of those who reported high pain. This is consistent with previous research with clinical patients that has found that anger tends to be common amongst those with chronic pain (Okifuji et al., 1999a), although most research fails to specify whether state or trait anger are being investigated (Wade et al., 1990). Moreover, while there appear to be reported gender differences in anger management styles, i.e. suppressed or expressed anger, there is little information in the literature with regard to trait anger and gender differences. Those in greater pain were also less confident in their ability to perform normal activities in spite of their pain. This is consistent with previous research. For example, O'Leary et al. (1988) and Lorig et al. (1989) reported that high self-efficacy before treatment was related to less reported pain intensity after treatment.

Depression, however, was closely related to anxiety and regression analysis showed that most of the variance in depression was predicted by 'state' anxiety. Fordyce and Steger (1979) reported that anxiety in chronic pain is increasingly likely when it becomes clear to the individual with pain that treatment is not succeeding and that increased pain severity is more probable. However, state anxiety represents a 'transient' state so the notion that it was predicting depression as a long-term chronic condition appears questionable. It seems possible to conclude that it was reflecting a situational state of

distress related to discussing a distressing medical condition with a stranger, rather than a long-term stressor.

The relationship between depression and anxiety is consistent with other research that suggests that discriminating between anxiety and depression can be problematic. Anxiety is difficult to define and measure given its overlap with depression (Gulley & Nemeroff, 1993; Preskorn & Fast, 1993). Furthermore, as most research fails to discriminate between state and trait anxiety, making firm conclusions and comparisons is difficult. Finding that anxiety was significantly related to depression, but was not related to pain severity, may provide some support for the theory that anxiety and depression can sometimes occur together as "cothymia" (Tyrer, 1989). It is also consistent with previous research suggesting that anxiety and depression occurring together reflect a co-morbid association operating independently of chronic pain (Ackerman & Stevens, 1989).

It may be argued that measures other than the ones chosen to assess depression and anxiety in the present research may have better discriminated between the disorders. However, the CES-D and the STAI measures have been used consistently with pain subjects and there was no reason to think they would not be suitable in this case. Certainly, using an instrument such as that developed by Lovibond and Lovibond (1995), for example, may have been more useful but as that measure has not been commonly used in pain research, it was not considered an option. The extent of inter-relationship between depression and anxiety, especially with respect to chronic pain, remains contentious.

Overall, it was decided that incorporating anxiety into future research would continue to be problematic, given that so many other variables were being investigated, and it was omitted from the subsequent studies. This decision was not only based on the apparent "multicollinearity" of the depression and anxiety measures, but also because it was 'state', rather than trait anxiety which predicted most of the variance in depression. As mentioned, being anxious while filling out forms about one's pain and suffering could be considered a likely and usual response. The subjects could have been experiencing this 'form-filling anxiety' rather than a proneness to anxiety. Such information is not especially informative in

terms of predicting adjustment to chronic pain. If trait anxiety, on the other hand, had been the main predictor, this may have been an issue worthy of further investigation. On the basis of these considerations, the decision was made to omit the anxiety measures.

Data analysis provided some support for the theory that perceived pain severity and depression were not directly related in this group. The analysis indicated that it was more likely that other variables, consequential to pain, made significant contributions to depression, and need to be acknowledged in trying to explain the development of depression in the person with chronic pain.

As this was an exploratory study and as pain severity and depression were not significantly related, it was decided to conduct further analyses to determine if there were indirect relationships, a situation suggested by previous research (Maxwell et al., 1998; Rudy et al., 1988; Turk et al., 1995). Analysis revealed that pain severity and depression might be indirectly rather than directly related, via the effects of perceived interference and pain self-efficacy. This is not completely consistent with previous researchers who had found that perceived life control was an important mediator (Maxwell et al., 1998; Rudy et al., 1988; Turk et al., 1995).

Although research into pain self-efficacy is relatively recent, it has been reported as a key cognitive variable in terms of adjustment to chronic medical syndromes, including chronic pain (Asghari & Nicholas, 2001; Arnstein et al., 1999; Bandura et al., 1987; Nicholas, 1994). Also, there has been some suggestion that it may play a pivotal role in the cognitive-behavioural model of chronic pain (Lin, 1998; Rudy et al., 1988; Turk & Rudy, 1992). The finding that pain self-efficacy was prominent in the correlation matrix is consistent with the cognitive-behavioural explanations of how elements of the chronic pain experience interact. For example, the degree to which chronic pain interferes in daily life is reflected in an erosion of confidence or self-efficacy in ability to deal with the condition. Believing that an activity will lead to pain is likely to undermine the confidence one has to actually perform the behaviour (Council et al., 1988). The consequence of this is that the individual is less likely to engage in the behaviour. This affects both self-efficacy and

activity levels. If self-efficacy is low to begin with and activities are avoided, pain is seen or believed to be less severe as a result of decreased mobility. This confirms to the individual that the right decision was made, the general activity level falls and muscles atrophy. Thus when some activity is undertaken and pain results, this confirms to people with chronic pain that they should have avoided the activity in the first place. The ongoing dysfunction may then lead to increased pain severity and depression in a cyclical pattern (Turk & Rudy, 1992). These findings are discussed more fully in Chapter 7 in the context of the overall research. In sum, this chapter has described methodological issues related to the present research, as well as detailing the initial study. The first study was conducted in order to determine the extent and type of psychological dysfunction present in a small group of people with chronic back pain, to explore test instruments and determine useful hypotheses. This study has been a useful demonstration of statistical techniques that have the potential to be even more useful with a larger sample. The findings have shown that conclusions drawn from clinical research are not necessarily applicable to people with chronic back pain from the wider community. This research will be extended with a larger sample.

4.6 Hypotheses for further investigation

Findings from this study, along with evidence outlined in the literature review, suggest the following hypotheses are suitable for testing with a larger sample, to be presented in Chapter 5.

In accord with the first aim of this research, Hypotheses 1 addresses the typical nature of the chronic back pain group, in terms of clinical profile classification. In line with the second aim, Hypothesis 2 addresses the comparison between people with chronic back pain and an otherwise healthy control group. In accord with Aim 3, Hypotheses 3 and 4 are concerned with the comparison between sub-groups of depressed and non-depressed people with chronic back pain. Finally, in line with the fourth aim of the thesis, Hypotheses 5 and 6 address the relationship between chronic pain and depression in the entire sample and between males and females, respectively.

1. The chronic pain profile classification system (Kerns et al., 1985) will be replicated on this non-clinical sample of people with chronic back pain, such that at least 90% of the sample can be classified as either dysfunctional, interpersonally distressed or adaptive coper.
2. The chronic back pain group will report higher depression and trait anger scores and lower internal health locus of control and general self-efficacy scores than the non-pain healthy control group.
3. The depressed persons with chronic back pain will be female, single, older, have less education, be unemployed, taking more prescribed medication, have had more back operations and have been in chronic pain longer, compared to the non-depressed persons with chronic back pain.
4. The depressed people with chronic back pain will demonstrate higher levels of perceived interference and state anger and lower levels of internal health locus of control, pain self-efficacy and general self-efficacy, compared to the non-depressed.
5. The direct correlation between chronic pain severity and depression will be positive but small and cognitive-behavioural variables will act as mediators between pain severity and depression. These cognitive-behavioural variables will include interference and pain self-efficacy.
6. There will be gender differences in the relationship between chronic pain severity and depression. For males, chronic pain and depression are more likely to be indirectly related and mediated by cognitive-behavioural variables, particularly interference. For females, the relationship is more likely to be direct such that chronic pain severity and depression will be positively and significantly correlated.

CHAPTER FIVE

STUDY 2: COGNITIVE-BEHAVIOURAL FUNCTIONING OF CHRONIC BACK PAIN PARTICIPANTS COMPARED TO HEALTHY CONTROLS

5.1 Introduction

This chapter presents Study 2, the general purpose of which was to investigate the extent of psychological dysfunction in a large group of people with chronic pain. This investigation was based on the findings of the first study, as well as previous research outlined in the literature review. This second study was based on a community survey conducted in urban and rural South Australia during 1997 and 1998.

Results of the first study provided grounds for continuing the search for an explanation of the chronic pain-depression link with path analysis modelling techniques. An important finding from Study 1 was that chronic pain and depression were not directly linked in the sample of 30 chronic back pain participants. Path analysis revealed that interference and pain self-efficacy were possibly mediating a relationship. This result is partly consistent with previous research (Kerns & Haythornthwaite, 1988; Maxwell et al., 1998; Rudy et al., 1988; Turk et al., 1995). Study 2 was designed to test hypotheses listed at the end of Chapter 4 with a large sample of chronic back pain participants.

5.2 Design

This study used correlations, multiple regressions, t-tests and path analysis techniques. Initially it was planned that Structural Equation Modeling (SEM) would be used to test a cognitive-behavioural mediation model of chronic pain and depression as this was the technique used by Rudy et al. (1988), on which this research was partly based. SEM has become very popular with social scientists over the past decade or so, but more recently there has also been criticism of its misuse as well, given that it is not suitable for all

types of data (Pedhazur & Schmelkin, 1991). Other researchers who also used the Rudy study as a basis for similar research did not use SEM, but rather used correlation, regression and/or path analysis to analyse data (Maxwell et al., 1998; Turk et al., 1995). It is not clear why the subsequent studies did not use SEM, although small sample size may have been a contributing factor. According to more recent information about using SEM, at least 200 participants are required to make reliable conclusions using this technique (Hoyle, 1995; Loehlin, 1998; Mueller, 1996). With respect to the present research, acquiring participants for Study 2 proved to be as difficult as for Study 1, and the final number was 105 participants. Based on this, the author was therefore advised that path analytical techniques would be more appropriate (Willson, 1999).

5.3 Method

The hypotheses presented at the end of Chapter 4 are the focus of this chapter. A comprehensive investigation of the psychological functioning of the chronic back pain groups is conducted to test Hypotheses 1, 3 and 4. The chronic back pain group and non-pain healthy controls are compared on several measures to test Hypothesis 2, concerning differences in depression, trait anger, internal health locus of control and general self-efficacy. Hypotheses 5 and 6 are concerned with the role of cognitive appraisal in the relationship between chronic pain and depression, for the total sample of participants and for each gender, respectively.

5.3.1 Participants

The participants of this study (N=171) consisted of 105 people with chronic back pain and 66 non-pain healthy controls. These people were recruited from the general community via posters and newspaper advertisements requesting volunteers (see Procedure, Section 5.3.3, for more detail). The mean age of the chronic back pain participants was 45 years, 5 months (SD = 12 years, 10 months) and 64 (60.9%) of them

were female. Of the participants in the control group, the mean age was 42 years, 3 months (SD = 12 years, 10 months) and 40 (60.6%) were females.

5.3.2 Measures

The variables investigated in this study were assessed by several questionnaires, which were stapled together to form one document, as in Study 1. However, the sequence of questionnaires was different to that used in Study 1. Instead of being used for face-to-face interviews, the Structured Interview Guide for Chronic Pain Patients (Nicholas, 1994) was used, in modified format, in the telephone interviews with volunteers. These were used to screen volunteers as well as to develop rapport with potential participants. Information from these interviews was of a qualitative nature and will not be reported in this thesis. As before, the first part of the questionnaire document sent to participants was the Patient Information Questionnaire, based on that used at the Royal North Shore Hospital and University of Sydney Pain Management and Research Centre (1994). It includes demographic and pain history details.

As mentioned, as a result of findings made in Study 1, of the strong overlap between anxiety and depression measures, the anxiety measures used in the first study were omitted, and two new measures, health locus of control and general self-efficacy, were introduced. The resultant sequence of questionnaires for the chronic back pain participants was therefore as follows: measures 1 to 9 and 12 as detailed in chapter 3; measures 10 and 11 are described following a listing of all measures. The control participants completed a Patient Information Questionnaire, modified for non-pain participants, and the other non-pain specific questionnaires to assess depression, anger (state, trait, suppressed and expressed), internal health locus of control and general self-efficacy (9 to12 below).

1) *Perceived pain severity* as assessed by the pain severity scale from the MPI (Kerns et al., 1985).

2) *Pain duration* was measured with one question regarding date and year of commencement of pain, in the MPI (Kerns et al., 1985).

3) *Perceived life interference* was assessed with the composite Interference scale from the MPI (Kerns et al., 1985). This consists of a) social, b) work, and c) family interference scales.

4) *Perceived life control* was measured by the life control scale from the MPI (Kerns et al., 1985).

5) *Perceived social support* was measured by the Support scale from the MPI (Kerns et al., 1985).

6) *Punishing, solicitous and distracting responses* were assessed with three scales from the MPI (Kerns et al., 1985).

7) *Activity level* was measured by the General Activity scale from the MPI (Kerns et al., 1985).

8) *Pain self-efficacy* was measured by the Pain Self-Efficacy Questionnaire (Nicholas, 1988).

9) *Anger* was measured by the Spielberger State-Trait Anger Inventory (Spielberger et al., 1985).

10) *Internal health locus of control* was measured by the Multidimensional Health Locus of Control Inventory (Wallston & Wallston, 1978).

11) *General self-efficacy* was measured with the General Self-Efficacy Scale (Sherer et al., 1982).

12) *Depression* was measured by The Center for Epidemiologic Studies Depression Scale (Radloff, 1977).

(i) Details of additional measures: Internal Health Locus of Control and General Self-efficacy.

Internal health locus of control was measured by the Multidimensional Health Locus of Control Inventory, Form A (MHLC: Wallston & Wallston, 1978). This is an 18-item, 6

point Likert-type instrument designed to assess the way people view specific key health-related matters. Each item is a belief statement with which the respondent agrees or disagrees. The items each have a rating scale ranging from 1 (strongly disagree) to 6 (strongly agree). The 18 items form 3 sub-scales of 6 items each, for Internal, Chance and Powerful Others locus of control. The maximum score for each scale is 36. Only the Internal sub-scale was used in the present research following the suggestion of Rudy et al. (1988). Previous research has reported that this scale is reliable, with Chronbach's alpha of at least 0.70 and test-retest correlations of at least 0.65 (Rudy et al., 1988).

General self-efficacy was measured by the General Self-Efficacy Scale (Sherer et al., 1982). The original scale consisted of two sub-scales, a general self-efficacy sub-scale and a social self-efficacy sub-scale. Only the general self-efficacy sub-scale was used in the present study. This is a 17-item scale that reflects efficacy as it relates to general abilities, as opposed to the social self-efficacy sub-scale which reflects efficacy expectations in social situations. The items have a rating scale ranging from 1 (strongly disagree) to 7 (strongly agree). Eleven of the items are reverse scored. A total score is calculated by summing the scores for all 17 items with a possible maximum of 119, which indicates high general self-efficacy.

This measure has been used only in limited research, but has been found to meet reliability and validity criteria. Reliability has been reported with Chronbach's alpha coefficients of 0.84 and 0.86 (Sherer et al., 1982; Woodruff & Cashman, 1993). Construct validity has been demonstrated with the scale correlating well with other measures of personality. These include Rotter's (1966) Internal-External Control Scale, Crowne and Marlowe's (1964) Social Desirability Scale, Holland and Baird's (1968) Interpersonal Competency Scale and Rosenberg's (1965) Self-esteem scale (see Sherer et al., 1982, for details). It has also correlated well with the Minnesota Multiphasic Personality Inventory (MMPI: Graham, 1977) and masculinity (Bem, 1974) and assertive measures (Rathus, 1973; see Sherer & Adams, 1983, for details). As predicted, general self-efficacy has been found to correlate well with general adjustment, as implied by these measures. Sherer et al.

(1982) and Woodruff and Cashman (1993) have also reported criterion validity. The GSES can be seen in Appendix F.

5.3.3 Procedure

To acquire respondents for the present study, a similar but more extensive media campaign was conducted than for Study 1. All the participants were accepted into the study according to inclusion criteria. For the chronic back pain participants, these were: a) aged 18 years or over; b) English speaking; c) upper and/or lower back pain for most of each week (i.e. on 4 days out of 7) for the previous 6 months or more; d) not presently attending and have not attended a pain clinic or medical pain management program; e) not experiencing chronic pain in multiple body sites; f) no other diagnosed major medical or psychiatric disorder and g) pain not related to worker's compensation/litigation process. For the non-pain controls, the inclusion criteria were: a) aged 18 years or over; b) English speaking; c) no persistent back pain for at least the previous 6 months or at any previous time in their lives for more than 6 months and d) no other diagnosed chronic pain condition, major medical or psychiatric disorder.

As before, participants were recruited from the general community via newspaper advertisements and posters, which described the research and requested volunteers. The advertisements were carried in *The Messenger*, a local newspaper which covers a wide area of Adelaide and the surrounding rural areas. Posters were displayed at local transport stations and at Adelaide and Flinders Universities. They were also displayed at shopping centres in diverse metropolitan suburbs such as Modbury, West Lakes and Marion in order to cover as much of the city area as possible. Posters were not displayed in any health care centre in an effort to reduce the number of participants who were clinical patients. The author's principal supervisor also spoke about chronic back pain on a local radio program. The process to acquire volunteers for this study took approximately 6 months from inception until all of the questionnaires were received. Meanwhile, a third media campaign to acquire

control participants had limited success, and a fourth was totally unsuccessful. Subsequently, it took more than 9 months to recruit the non-pain participants.

Two hundred and fifty-one people volunteered to take part in the research, 152 persons with chronic back pain and 99 otherwise healthy adults who did not have chronic back pain. Due to time and resource constraints it was decided to forego face-to-face interviews as were conducted in the first study. Instead, participants were screened over the telephone with a modified interview format based on that used in the first study.

Of the 152 pain participants who volunteered to participate in the research, 124 were found to fit the selection criteria and were subsequently sent questionnaire packets with reply-paid envelopes. These contained an information sheet, participation consent form and instruction sheet and the questionnaire. Of these 124 people, 19 failed to return the questionnaires, despite 3 rounds of follow-up letters, or they withdrew for various reasons. Finally, 105 complete sets of data for chronic back pain participants were received. Of the 28 people who did not fit the selection criteria, 3 had not been in pain for at least the previous 6 months; 3 had been referred to a pain clinic; 7 complained of multiple chronic pain sites; 5 had been diagnosed with a depressive disorder; 1 was being treated for an anxiety disorder and 9 were involved in workers' compensation cases.

For the control group, 99 otherwise healthy individuals who did not suffer chronic back pain volunteered to participate in the research as a result of a poster (Appendix G) and advertisement campaign similar to that for the pain participants. Each package sent to a control participant contained a participation consent form and an information and instruction sheet (Appendix H) and the questionnaires. They also received a Patient Information Questionnaire, modified from that sent to the pain participants. Sixty-six of these people, who met the selection criteria, were matched for age, gender marital and employment status to the pain participants and provided complete data. The final number of total participants who completed the study was 171, which represents a return-rate of 68% of the original 251 persons who had expressed interest in the research.

5.4 Results

In the following sections, demographic characteristics are reported along with responses to personal history questions. Subsequently, cognitive-behavioural responses to chronic pain and results of hypothesis testing are reported.

5.4.1 Chronic back pain: Demographic characteristics

As mentioned, of the 105 chronic back pain participants who completed the study, 41 (39%) were males and 64 (60.9%) were females. They were aged between 20 and 74 years with a mean age of 45 years, 5 months (SD = 12 years, 10 months). Chronic pain duration ranged from 8 months to 54 years, 9 months (M=14.9; SD=11.5). Table 5.1 summarises the demographic and pain related variables of the chronic pain participants for this group (N=105).

It can be seen from Table 5.1, most of the sample (67%) was married or living with significant other/s and a majority had been educated to at least year 12 (56%). Approximately a third worked full-time (31%), with the remainder engaged in part-time or volunteer work (25%) or retired and/or home-makers (28%).

Information on drug use showed that a majority (53%) were taking prescription drugs[10], including non-steroidal anti-inflammatory drugs (nsaids), for their pain. Dosage varied from routinely at night before bed up to 6 - 8 tablets a day with a few taking medication only as needed. These included narcotic analgesic drugs (panadeine forte, capadex, digesic, mersyndol forte) and non-steroidal anti-inflammatory drugs (voltaren, brufen, naprosyn) taken on a regular basis. Thirty-five people (33%) were mainly taking *over the counter* analgesic drugs (panadeine, mersyndol, aspirin) and/or nsaids (nurofen) when needed. Only 14 (13%) reported that they avoided prescription or over the counter drugs for pain most of the time and that if urgent pain relief was required, they tended to resort to heat treatments, hot baths or showers and skin balms or anti-inflammatory creams. Most of the sample (80%) had not had surgery for back pain.

[10] Information about script-takers is presented after Table 5.2.

Table 5.1. Demographic and pain-related characteristics of the chronic back pain participants for Study 2 (N=105).

Demographic characteristics	Participants	
	n	%
Gender		
Male	41	39.0
Female	64	60.9
Marital Status		
Married/Defacto	70	66.7
Single	16	15.2
Div/sep/wid	19	18.1
Education		
≥Year 12	59	56.2
<Year 12	46	43.81
Employment Status		
Full-time	32	30.5
Part-time/volunteer	26	24.8
Unemployed	18	17.1
Retired/home-duties	29	27.6
Drug use		
Prescription (including nsaids°)	56	53.3
Otc°° (incl. nsaids)	35	33.3
No drugs	14	13.3
Back operations		
No	84	80.0
Yes	21	20.0
	Mean	**SD**
Age	45.45	12.85
Pain Duration	14.9	11.5

°non-steroidal anti-inflammatory drugs; °°over-the-counter

5.4.2 Chronic back pain: Personal history

Participants were asked a range of other questions about their personal history and more specific questions about their pain history, as follows:

If you have children, how many do you have?

Seventy-three percent of the participants had at least one child; including 34% who had 2 children.

What was your country of birth?

Seventy percent of participants were born in Australia and had lived all their lives here. Twenty percent were United Kingdom born while 5% were born in Europe. The remaining 5% were born in other regions, including Asia.

What was your main occupation before your pain/injury?

Twenty-one percent were nurses, while 18% were employed in manual labour. Of the remainder, 15% had been working in sales or clerical positions and 15% classed their main occupation as 'student'. Nine percent were engaged in 'home duties' and 7% endorsed the 'shop assistant' category. There were only 11% in 6 other categories including teacher/lecturer, manager, medical/science technology, doctor, lawyer and mental health professionals. The remainder did not answer the question.

If working now, is your work restricted due to pain?

Twenty-nine percent of participants reported that their work was restricted by pain, while 24% said it was not. However, 47% reported that this was not applicable, as they were not engaged in paid work.

How did your pain begin: (tick <u>one</u>; if more than one applies, tick the one which applies <u>best</u>).

Twenty-four percent reported that they did not know how their pain began while 19% reported that it was an 'accident at work'. It should be noted here that during the initial screening telephone interview, people had been asked if they had been or were currently involved in worker's compensation cases and they were not included if so. Therefore it is

understood that those who reported that they believed their pain began because of an accident at work did not seek compensation for (unknown) reasons. Sixteen percent described their origin of pain as 'other' but did not expand on this. Fourteen percent reported that their pain began 'at work, but not accident'. Most of these had suggested during the interview that it was the type of work they did, i.e. involving much bending and lifting, that they felt had contributed to their pain over a long period of time, rather than any single incident. Of the remainder, 11% cited 'accident at home', 8% cited 'car accident' and 8% failed to answer the question.

What is the best description of your pain?

All participants had met the criterion for pain that was present for 'most of each week for more than the previous 6 months'. Within that category, however, their descriptions of pain varied. Many (38%) described their pain as 'always present, intensity varies'. The second largest percentage (27%) described their pain as 'usually present, with short periods without pain'. Twelve percent described their pain as 'often present, but pain-free most of day'. Ten percent reported that pain was 'often present with longer pain-free periods'. The remainder (13%) indicated that pain was 'occasionally present for brief periods, but not every day' or they did not describe their pain.

What makes your pain worse?

Participants were asked to tick more than one category if necessary. The main factors that made pain worse were bending (75%), lifting (73%), standing (62%) and sitting (62%). To a lesser degree were tension and stress (40%), walking (36%) and driving (35%). This indicated that, for this group, various physical activities increased their back pain.

What makes your pain better?

Again, they were asked to tick more than one category, if appropriate. The main activities that appeared to relieve pain were lying down (60%), pain medication (56%), massages (53%), hot showers (43%), stretching (42%) and walking (24%). A variety of activities were thus used by the group to relieve pain.

Since your pain began, which of the following people have you seen about it and who has been most helpful?

A list of health professionals was provided. These were:

__Acupuncturist

__Anaesthetist

__Chiropractor

__Homeopath

__Hypnotherapist

__Neurologist

__Neurosurgeon

__Occupational therapist

__Orthopaedic surgeon

__Physiotherapist

__Psychologist

__Psychiatrist

__Rheumatologist

__General practitioner

__Other

Eighty-five percent of the participants indicated that they had visited most of these professionals at least once, although only four were consistently indicated by participants. In order of frequency of indicated helpfulness were: physiotherapists (26%), chiropractors

(20%), general practitioners (11%) and acupuncturists (5%). Participants were also asked to indicate how often and in what time frame they had visited health professional/s for their pain, but this question was poorly responded to. There were not enough data to comment on the issue.

In summary, it appears that most of the participants were parents (73%), and Australian (70%) or British born (20%). As for occupation, many (21%) were working as nurses, with another 18% engaged in some form of manual labour. Combined, these represent the largest category and therefore implied that many of the participants could be classed as involved in labour intensive occupations involving high levels of bending and lifting. The other main occupations included clerical, sales and student. Only a few (12%) were classed in a profession such as teacher, doctor, lawyer and mental health professionals. Forty-seven percent reported that they worked despite their pain or that pain was not an impediment to their working.

The largest category for the reported origin of pain was 'unknown' at 24%. However, as many as 19% reported that the origin of their pain was 'accident at work' and 16% reported that their pain began from some 'other' cause but failed to elaborate on this. Fourteen percent of the sample described the origin of their pain as 'work, but not an accident' and this had been elaborated at many of the interviews to mean that it was the type of work they did rather than a single incident. That is, 'bending' and 'lifting' were cited as being integral to their occupations and thought to be largely responsible for their ongoing problems. Most of the group (65%) reported that their pain was either 'always present' with varying intensity or 'usually present'.

Overall, it appeared that the most troublesome movements for people suffering chronic pain were the obvious ones such as bending, lifting, standing and sitting for extended periods of time. Moreover, pain relief was reported as mainly provided by resting, pain medication, massages and hot showers. While physiotherapists, chiropractors, general practitioners and acupuncturists were mostly cited as the health professionals who

were sought for pain relief, only a minority of the sample actually found them to be helpful and in that order.

5.4.3 Chronic back pain: Sensory, cognitive, behavioural and affective responses

The analyses and assumptions for this study are the same as for Study 1 (refer to section 4.4.1). There are multiple comparison analyses in this chapter and it could be argued that these require appropriate p-value adjustments to compensate for the possibility of an increased risk of Type I errors. This arises from the belief that with increased comparisons there is a greater chance of reporting a significant result that is really due to chance. However, according to Feise (2002), there are a number of reasons why post-hoc correction testing is not necessarily appropriate. For example, correction testing (e.g. Bonferroni) tests a 'family' level of alpha when the real interest is individual differences and while correction testing may reduce Type I errors, it increases the chance of making Type II errors. In addition, correction testing may imply that statistical significance is more important than the quality of the research. Effect size is just as important as significance testing. Furthermore, multivariate testing can be problematic, i.e. interpretation of results, such that univariate testing may still be required. Therefore, correction testing was not performed on the data.

In accord with the aims of this thesis, the data for the entire group of participants (N=105) will be initially addressed to allow for comparisons between much of the previous research that has not examined group differences, such as those that may be related to gender. Gender differences will be examined subsequently. The means and standard deviations of responses to questionnaires measuring cognitive-behavioural variables are detailed in Table 5.2. Previous research with pain and non-pain samples has also provided means and standard deviations for these measures (see Study 1, Table 4.2 for details) and these were compared to the results from the present study.

One sample t-tests were conducted, as with Study 1, to determine whether the present sample was significantly different from the normative data and these results are

also presented in Table 5.2. As in Study 1, the normative data available was not detailed enough to conduct two sample t-tests. That is, only the means and standard deviations were available, not the individual scores, so two sample t-tests were not appropriate. Under such circumstances, it is therefore appropriate to use the one sample t-tests (Bryman & Cramer, 1997; pers. comm. Willson, R). The statistics package (SPSS) has the facility to carry out one sample t-tests when only the means of normative data are available. Normative data for the two new measures, internal health locus of control (N=122: mixed non-pain adult sample) and general self-efficacy scales (N=101: non-pain adult students) were obtained from Wallston et al. (1983) and Sherer and Adams (1983), respectively. The sample sizes for the normative data are presented in the table for easy reference.

Table 5.2. Means and standard deviations of cognitive-behavioural functioning in chronic back pain participants compared to normative data from pain samples and non-pain samples.

Variables	Chronic pain group (N=105)		Normative Data - pain samples		Normative Data - non-pain samples			
	Mean	SD	Mean	SD	Mean	SD	t	d
			(N=150)					
Pain severity	3.15	1.44	4.64	1.00			-10.67***	1.24
Interference	3.37	1.49	4.79	0.98			-9.76***	1.16
Life Control	3.54	0.88	3.09	1.52			5.21***	.35
Social Support	3.26	1.83	4.59	1.43			-7.49***	.82
Punishing Rs	1.56	1.56	1.72	1.62			-1.05	.10
Solicitous Rs	2.72	1.73	3.61	1.55			-5.20***	.55
Distracting Rs	1.62	1.39	2.47	1.46			-6.23***	.59
Activity	2.90	0.84	1.96	0.91			10.14***	1.06
			(N=103)					
Pain self-efficacy	38.90	13.23	25.8	12.4			11.49***	1.02
					(N=122)			
Internal Health locus of control	25.42	5.08			25.10	4.89	0.63	.06
					(N=101)			
General self-efficacy	86.70	15.59			64.31	8.58	14.72***	4.14
					(N=4062)			
State Anger	11.88	4.14			12.04	4.00	-0.41	.04
Trait Anger	17.75	5.89			19.05	4.96	-2.26*	.26
Suppressed Anger	16.30	5.07			15.53	4.08	1.55	.18
Expressed Anger	13.99	4.14			14.67	3.68	-1.68	.18
					(N=1173)			
Depression	15.78	10.27			9.92	9.32	5.87***	.62

*p<0.05; ***p<0.001

As can be seen from Table 5.2, the present sample reported significantly less pain severity, interference and social support and significantly more life control, pain self-efficacy and activity than normative pain samples from previous research. For four of the scales, effect sizes are large, two are medium and one is small to medium. Compared to the non-pain samples, they did not differ on internal locus of control, but reported significantly more general self-efficacy, with a large ES. They were also significantly more depressed than the non-pain sample and the ES was medium to large. In this study, the cut-off score of 19 is used with the main intent to identify 'depressed' members of the chronic pain group. Although not shown in the above Table, 34.3% (N=36) of the chronic back pain group scored 19 or more on the CES-D (Radloff, 1977). The mean score for this group was 27.83 (sd=6.26), which was significantly greater than the average score of 9.52 (sd=4.93) reported by the non-depressed group (t=-16.43, df = 103, p = 0.001). With regard to the anger measures, they did not differ from the normal non-pain group except for reporting significantly less trait anger, although the ES was small. Note that this sample will also be compared to non-pain controls specifically acquired for this research, in the testing of Hypothesis 2.

Although not presented here, the sample was also divided into those who were taking mostly prescription medication for pain (n=56) versus those who were not (n=49). They did not differ on any of the demographic characteristics, except that the script-takers had significantly more back operations (χ^2 = 11.06; df = 1; p < 0.001). Close inspection of the data showed that the script-takers were also more inclined to report general practitioners of greatest help, while the rest were more inclined to report that chiropractors were of most help. However, the numbers in each group were too small to provide a significant result.

(i) Testing the Hypotheses

In the following sections, the hypotheses presented at the end of Chapter 4 are detailed and tested. To begin with, the present sample of people with chronic back pain are

investigated to determine the extent to which they resemble clinical profiles, given that one of the aims of the thesis is to investigate a non-clinical sample. The participants are then compared to a non-pain otherwise healthy group of people to determine how persistent pain may interfere with mood states such as depression, emotions such as anger and specific beliefs about responsibility of health behaviour and general confidence in daily functioning. The entire group of participants is then sub-divided into those who scored as 'depressed' on the depression inventory and those who did not. These sub-groups are then investigated to determine whether they differ on demographic characteristics and cognitive functioning. Finally, cognitive-behavioural mediation models of chronic pain and depression are tested, based on data from the whole sample and from male and female participants, separately.

Hypothesis 1, concerned with the profiles of people with chronic pain, is based on evidence detailed in the literature review. To briefly reiterate, Turk and Rudy (1988) identified 3 patient subgroups, determined by cluster analysis, based on patient responses to the MPI. These are automatically provided as part of the printout of each participant's responses. They are empirically derived groups of chronic pain patients that have been labelled as 1) 'dysfunctional', characterised by high pain severity, psychological distress and interference with low control and activity levels; 2) 'interpersonally distressed', characterized by low perceived support and high levels of negative responses from significant others; and 3) 'adaptive copers', characterized by low levels of psychological distress and high activity levels compared to the other two groups. Other research has replicated these subgroups (Talo, 1992; Turk et al., 1995; Turk & Rudy, 1990) and in these previous studies, 100% of clinical samples have been classified according to these 3 categories. In addition, most clinical studies have found that about 40% of patients can be classified as dysfunctional and 30% as either interpersonally distressed or adaptive copers.

There are 3 other possible classifications which Rudy (1987) identified and which the MPI automatically selects, although they are not typically mentioned in research studies. If a case has too much missing data, (i.e. more than 2 of the 9 scales have missing data), it is classified as 'unanalysable'. There is also the 'hybrid' classification, which means the "MPI

scale scores represent aspects of more than one prototypic profile" (Rudy, 1987, p. 40), and the 'anomalous' category, which means "the MPI scale scores make no sense according to established theory" (p. 40). This implies that the participant has not responded in any recognised way, which could reflect their difficulty interpreting the questionnaire, faking responses or random responses, etc.

The hypothesis is as follows:

Hypothesis 1: The chronic pain profile classification system (Kerns et al., 1985) will be replicated on this non-clinical sample of chronic back pain participants, such that at least 90% of the sample can be classified as either dysfunctional, interpersonally distressed or adaptive coper.

In order to test the hypothesis, the profiles of the chronic back pain participants were examined. The profile classifications were provided by the MAP computer program as described in section 4.3.3. Table 5.3 shows the numbers and percentages of participants from the present study that could be classified according to the 6 categories. Breakdown according to gender is also shown.

Table 5.3. Profile classification for chronic back pain participants (N=105).

Profile Classification	**Total N (%)**	**Male n=41 (%)**	**Female n=64 (%)**
Dysfunctional	9 (8.6)	1 (2.4)	8 (12.5)
Interpersonally Distressed	28 (26.7)	14 (34.1)	14 (21.9)
Adaptive Coper	25 (23.8)	12 (29.3)	13 (20.3)
Unanalysable	15 (14.3)	5 (12.2)	10 (15.6)
Hybrid	4 (3.8)	1 (2.4)	3 (4.7)
Anomalous	24 (22.8)	8 (19.5)	16 (25.0)

As can be seen from Table 5.3, only 59% of participants could be classified into one of the three main patient subgroups. The remaining 41% could not be readily classified due to missing data, hybrid effect or anomalous responding. Of the 15 who were unanalysable, 10 of these had no 'significant other' with whom they lived. All of those with hybrid profiles had significant others as did 23 of those who produced the anomalous profiles. It is not possible to examine original data to determine more clearly why a profile is categorized as unanalysable, hybrid or anomalous, as these are based upon statistical indicators within the computer program and are inherent to the assessment instrument (Rudy, 1989).

When the participants were divided into males and females, other differences were noted. More males (66%) compared to females (55%) could be classified into the 3 main subgroups. A greater percentage of the females were classified as 'dysfunctional' while more males were classified as 'interpersonally distressed'. In addition, while 29% of the males were classed as 'adaptive copers', only 20% of the females could be classed this way. Twenty-five percent of females and nearly 20% of males could not be classified due to responding that did not make sense according to "established theory" (Rudy, 1987, p. 40). In addition, 12% of males and 16% of females could not be classified due to missing data. Chi-square tests failed to show any significant differences between males and females in terms of profile classification. Hypothesis 1 has not been supported because less than 90% of the participants could be classified according to Kerns et al.'s (1985) system. This suggests that this sample of participants is dis-similar to other clinical patients who have been classified this way, implying that they are characteristically non-clinical.

If chronic pain affects cognitive functioning, then it would be expected that the chronic pain group would demonstrate cognitive dysfunction significantly more than people who do not experience chronic pain. Therefore, the following hypothesis relates to comparisons between the chronic back pain group and an otherwise healthy non-pain control group on a selected set of variables. Clearly, they cannot be compared on pain-related variables but this research does provide an opportunity to determine whether chronic pain may have an effect on cognitive variables not specifically related to pain.

Hypothesis 2: The chronic back pain group will report higher depression and trait anger scores and lower internal health locus of control and general self-efficacy scores than the non-pain healthy control group.

The non-pain control group consisted of 26 males (39.4%) and 40 females (60.6%), who ranged in age from 25 to 81 years, with a mean age of 42 years, 3 months (SD = 12 years, 10 months). As the control group had been matched for age, gender, marital and employment status, there were no significant differences in any of these demographic characteristics and this is demonstrated in Table 5.4. However, they were significantly more educated, post-secondary school (χ^2=5.68, df=1, $p<0.05$). Table 5.5 shows the findings for cognitive-behavioural variables. Independent sample t-tests were conducted on the cognitive-behavioural responses to determine whether the chronic pain group was significantly different from the control group. Only those variables that could be assessed in both groups have been analyzed.

Table 5.4. Demographic characteristics of chronic back pain and control non-pain participants: Nature and significance of between-group differences.

Demographic Characteristics	Chronic back pain group (n=105)		Controls non-pain group (n=66)					
	n	%	n	%	χ^2	df	p	sig
Gender					.002	1	0.96	Ns
Male	41	39.0	26	39.4				
Female	64	60.9	40	60.6				
Marital Status					4.52	2	0.10	Ns
Married/Defacto	70	66.7	53	80.3				
Single	16	15.2	8	12.1				
Div/sep/wid°°°	19	18.1	5	7.6				
Education					5.68	1	0.05	*
≥Year 12	59	56.2	49	74.3				
<Year 12	46	43.8	17	25.8				
Employment Status					4.98	3	0.17	Ns
Full-time	32	30.5	30	46.2				
Part-time/ volunteer	26	24.8	11	16.9				
Unemployed	18	17.1	7	10.8				
Retired/home-duties	29	27.6	17	26.2				
	Mean	**SD**	**Mean**	**SD**	**t**	**df**	**p**	**sig**
Age (years)	45.45	12.85	42.21	12.79	1.61	169	.11	Ns

*p<0.05; °°°divorced/separated/widowed

Table 5.5. Comparisons between chronic back pain and control non-pain participants: Means, standard deviations and t-tests for between-group differences.

Variables	Chronic back pain group (n=105)		Control non-pain group (n=66)			
	Mean	SD	Mean	SD	t	d
Depression	15.80	10.27	9.76	8.22	4.04	.63
State Anger	11.88	4.14	10.91	3.94	1.52	.24
Trait Anger	17.75	5.89	17.32	4.46	.51	.08
Suppressed Anger	16.30	5.07	15.53	3.35	1.09	.17
Expressed Anger	13.99	4.14	14.47	3.20	-.80	.12
Internal Health locus of control	25.42	5.08	26.61	4.34	-1.57	.25
General self-efficacy	86.70	15.59	88.73	13.79	-.87	.14

***p<0.001; d = Cohen's d.

As can be seen from Table 5.5, Hypothesis 2 has been partially supported. The people with chronic pain did report significantly higher depressive symptomatology than did the non-pain healthy controls (t=4.04, df=169, p< 0.001), with a medium to large ES (Cohen, 1992). Furthermore, as demonstrated in the previous table, the chi square tests showed that the control group was significantly better educated than the pain group. Therefore, to determine whether this difference in education accounted for the significant difference in depression scores, an ANOVA was performed comparing depression scores by group with education level covaried. The covariation did not reveal a significant effect and the difference between the groups remained significant [F (1, 168) = 18,06, p<0.001, two-tailed]. The adjusted means for depression after controlling for education were 15.96 and 9.51 for the pain group and control group, respectively. The people with chronic pain did not respond significantly differently from the controls on any of the other cognitive-behavioural variables. Although the controls scored in the expected direction for both internal health locus of control and general self-efficacy, i.e. higher, the mean scores were

not significantly different. The chronic back pain participants scored higher than the controls on all anger measures but none was significantly different.

The following two hypotheses relate to those who scored 19 or more on the CES-D and who are therefore described as 'depressed' persons with chronic pain. They are predicted to differ significantly from those who scored less than 19 on the CES-D, i.e. the 'non-depressed', on several demographic and cognitive variables. The hypotheses are based on previous research findings and the results of Study 1. While the models to be tested later in the thesis will also explore relationships between depression and other cognitive variables, the following hypotheses test aspects of the relationships that are not necessarily addressed by the model testing.

Hypothesis 3: The depressed persons with chronic back pain will be female, single, older, have less education, be unemployed, taking more prescribed medication, have had more back operations and have been in chronic pain longer, compared to the non-depressed persons with chronic back pain.

To test for significant differences in demographic characteristics, medication use or number of back operations between the groups, χ^2 tests were performed. Refer to Table 5.6 for details.

Of the 36 depressed persons, 66.7% (n=24) were female. The gender ratio of this group was not significantly different from that of the non-depressed group. The depressed group was significantly younger than the non-depressed group ($t=2.25$, $df = 103$, $p<0.05$). They ranged in age from 20 to 64 years (M = 41 years, 7 months; SD=11 years, 6 months) compared to the non-depressed group that ranged in age from 23 to 74 years (M = 47 years, 5 months; SD=13 years, 1 month). As can be seen from Table 5.6, there were no significant differences in marital status, education level or employment status, medication use or number of back operations.

Although findings are limited, there has been research that suggests that time in chronic pain is related to depression. However, as can be seen from Table 5.9, pain duration (in months), tested for significance with an Independent t-test, did not differ significantly between the two groups. However, as there has also been speculation that those who experience chronic pain for a very long time are also likely to be more depressed, separate analyses were conducted on those who had been experiencing chronic pain for more than 25 years. While this may seem an arbitrary figure, there was only one piece of published research addressing duration of chronic pain and depression in terms of a specific extended time frame. This was the Swanson et al. (1986) paper that found that such people were significantly more depressed than others who had experienced chronic pain for less than 25 years. Therefore, the present sample was divided into those who had experienced chronic pain for 25 years or longer and those who had not. Refer to Table 5.7 for demographic characteristics of the two groups.

Table 5.6. Demographic characteristics of depressed and non-depressed chronic back pain participants: Statistics and tests of significance (χ^2; t) of between-group differences.

Demographic Characteristics	Depressed group (scored ≥ 19) (n=36)		Non-depressed group (scored < 19) (n=69)					
	n	%	n	%	χ^2	df	p	Sig
Gender					0.75	1	0.39	Ns
Male	12	33.3	29	42.0				
Female	24	66.7	40	58.0				
Marital Status					1.72	2	0.42	Ns
Married/Defacto	21	58.3	49	71.0				
Single	7	19.4	9	13.0				
Div/sep/wid	8	22.2	11	15.9				
Education					0.78	1	0.39	Ns
≥Year 12	15	41.7	35	50.7				
<Year 12	21	58.3	34	49.3				
Employment Status					5.17	3	0.16	Ns
Full-time	6	16.7	26	37.7				
Part-time/ volunteer	11	30.6	15	21.7				
Unemployed	8	22.2	10	14.5				
Retired/home-duties	11	30.6	18	26.1				
Drug use					1.01	2	0.60	Ns
Prescription (including nsaids°)	20	55.6	36	52.2				
Otc°° (incl. nsaids)	10	27.8	25	36.2				
No drugs	6	16.7	8	11.6				
Back operations					0.86	1	0.36	Ns
No	27	75.0	57	82.6				
Yes	9	25.0	12	17.4				
	Mean	SD	Mean	SD	t	df	p	sig
Age (years)	41.61	11.53	47.45	13.12	2.25	103	0.03	*

*p=<0.05; °non-steroidal anti-inflammatory drugs; °°over-the-counter

Twenty percent (n=21) were found to have experienced chronic pain for 25 years or more (m=33 years, 11 months; sd=8 years, 1 months). This was significantly longer (t=-14.97, df=103, p=.001) than that of the remaining 80% (n=84) who had experienced chronic pain for less than 25 years (m=10 years, 9 months; sd=6 years, 8 months). As with the Swanson et al. (1986) study, this group acted as controls for the ancient pain group. The statistical significance of differences in demographic and cognitive measures of the two groups was evaluated using the χ^2 test and the Independent samples t test. Tables 5.7 and 5.8 show the demographic characteristics and cognitive responses to chronic pain for the two groups, respectively.

As expected, the 'ancient' pain group was significantly older than the other group (t=-4.62, df=103, p<0.001), and the age range was much more limited: 42 to 74 years for the ancient pain group, compared with 20 to 73 years for the shorter pain group. There were no significant differences in terms of other demographic characteristics (see Table 5.7). Categories were collapsed where there were less than 5 cases per cell but there were still no significant differences. In the marital status category, for example, 'single' was combined with divorced, widowed and separated people as the numbers per cell were too small to retain individual categories.

Table 5.7. Demographic characteristics of long-term (25 years or longer) and short-term chronic back pain participants: Nature and significance of between-group differences.

Demographic Characteristics	Long-term group (≥25 years) (n=21)		Short-term group (<25 years) (n=84)					
	n	%	n	%	χ^2	df	p	Sig
Gender					.16	1	.69	Ns
Male	9	42.9	32	38.1				
Female	12	57.1	52	61.9				
Marital Status					.27	1	.61	Ns
Married/Defacto	13	61.9	57	67.9				
Single/div/sep/wid	8	38.1	27	32.1				
Education					.24	1	.63	Ns
≥Year 12	11	52.4	39	46.4				
<Year 12	10	47.6	45	53.6				
Employment Status					.97	2	.62	Ns
Full-time	8	38.1	24	28.6				
P-t/vol/unemp°°°	7	33.3	37	44.0				
Retired/home-duties	6	28.6	23	27.4				
Drug use					.80	2	.67	Ns
Prescription (incl. nsaids°)	10	47.6	46	54.8				
Otc°° (incl. nsaids)	7	33.3	28	33.3				
No drugs	4	19.0	10	11.9				
Back operations					.02	1	.90	Ns
No	17	81.0	67	79.8				
Yes	4	19.0	17	20.2				
	Mean	SD	Mean	SD	t	df	p<	sig
Age (years)	56.05	9.37	42.80	12.26	-4.62	103	.001	***

*** p<0.000; °non-steroidal anti-inflammatory drugs; °°over-the-counter; °°°part-time, volunteer, unemployed.

Table 5.8. Comparisons between long-term (25 years or longer) and short-term chronic back pain participants: Means, standard deviations and t-tests for between-group differences.

Variables	Long-term group (≥25 years) (n=21)		Short-term group (<25 years) (n=84)			
	Mean	SD	Mean	SD	t	d
Pain severity	3.22	1.54	3.13	1.42	-0.27	.06
Interference	2.91	1.70	3.48	1.43	1.57	.38
Control	3.56	0.89	3.53	0.88	-0.17	.03
Social support	3.22	1.94	3.26	1.80	0.09	.02
Punishing Responses	1.12	1.33	1.67	1.6	1.45	.35
Solicitous Responses	2.79	2.05	2.71	1.6	-.17	.04
Distracting Responses	1.30	1.32	1.71	1.4	1.22	.29
Activity	2.83	0.99	2.91	0.80	0.44	.09
Pain self-efficacy	40.43	14.01	38.51	13.08	-0.59	.14
Internal Health locus of control	25.71	4.55	25.35	5.23	-0.29	.07
General self-efficacy	91.71	13.62	85.44	15.87	-1.66	.41
State Anger	11.71	2.37	11.92	4.48	0.20	.05
Trait Anger	15.00	3.79	18.44	6.13	2.45*	.60
Suppressed Anger	15.95	4.39	16.38	5.25	0.35	.08
Expressed Anger	14.05	2.73	13.98	4.44	-0.07	.02
Depression	13.10	11.07	16.48	10.01	1.35	.33

*p<0.05.

Unexpectedly, the 'ancient' group reported significantly less trait anger (t=2.45, df=103, p<0.05), with a medium to large ES of .60, than the controls (Table 5.8). This was the only significant difference between the two groups for cognitive variables. This is further evidence that duration of chronic pain does not necessarily predict depression. It is acknowledged, however, that the size of the 'ancient' group was only 21 participants which means that power could be low and the chance of making a Type II error, i.e. there is no difference when there really is, is increased. However, power analysis, as indicated by reporting of the effect sizes, mitigates this problem.

Overall, these findings indicate that there is little support for Hypothesis 3.

Hypothesis 4: The depressed people with chronic back pain will demonstrate higher levels of perceived interference and state anger and lower levels of internal health locus of control, pain self-efficacy and general self-efficacy, compared to the non-depressed.

To test Hypothesis 4, Independent samples t-tests were carried out to determine the extent of pain-related cognitive differences between the two groups. Refer to Table 5.9 for details.

Table 5.9. Comparison between depressed and non-depressed chronic back pain participants: Means, standard deviations and t-tests for between-group differences.

Variables	Depressed group (n=36)		Non-depressed group (n=69)			
	Mean	SD	Mean	SD	t	d
Pain severity	4.08	1.11	2.66	1.35	-5.80***	1.11
Interference	4.20	1.19	2.94	1.46	-4.46***	.92
Control	3.20	.95	3.71	0.79	2.93**	.60
Social support	3.33	1.98	3.22	1.76	-0.32	.06
Punishing Responses	2.16	1.88	1.25	1.28	-2.62*	.60
Solicitous Responses	2.71	1.88	2.74	1.66	0.09	.02
Distracting Responses	1.76	1.46	1.55	1.35	-.73	.15
Activity	2.70	.86	2.99	.81	1.73	.35
Pain self-efficacy	31.44	13.46	42.78	11.39	4.55***	.93
Internal Health locus of control	23.75	4.98	26.29	4.95	2.49*	.51
General self-efficacy	79.56	18.46	90.42	12.46	3.58**	.73
State Anger	13.44	6.37	11.06	1.81	-2.91**	.59
Trait Anger	18.94	7.76	17.13	4.58	-1.51	.31
Suppressed Anger	17.61	6.31	15.61	4.17	-1.95	.40
Expressed Anger	14.72	5.92	13.61	2.79	-1.31	.27
Pain duration	168.89	119.36	183.94	148.14	0.53	.11

*p<0.05; **p<0.01; ***p<0.001.

As can be seen from Table 5.9, depressed participants reported significantly more pain severity, interference, punishing responses and state anger and they reported significantly less life control, internal health locus of control, pain and general self-efficacy,

than the non-depressed participants. Power analysis showed that four of the ESs were large, while three were medium or medium to large. There was no significant difference between the groups in how supportive participants thought their social networks were or their activity levels. There were also no differences in levels of trait, expressed or suppressed anger. These findings do provide support for Hypothesis 4. Although it is not displayed, 56% of the participants scored greater than 39 on the PSEQ, indicating that a majority of them felt confident about dealing with their pain.

The following two hypotheses address the role of cognitive-behavioural variables in the relationship between chronic pain and depression. Initially, to test Hypothesis 5, data for the total group of 105 is examined to allow for comparison with previous research. Hypothesis 6 addresses gender differences in the cognitive-behavioural response to chronic pain.

Hypothesis 5: The direct correlation between chronic pain severity and depression will be positive but small and cognitive-behavioural variables will act as mediators between pain severity and depression. These cognitive-behavioural variables will include interference and pain self-efficacy.

In order to test Hypothesis 5, Pearson's product moment correlations were initially performed to determine the extent of inter-relationships. The results are presented in a correlation matrix, Table 5.11. Table 5.10 provides details of the abbreviations used in Table 5.11.

Table 5.10. Legend of abbreviations used for variables in Table 5.11.

Abbreviation	Variable
PSv	Pain Severity
Int	Interference
LC	Life Control
SS	Social Support
PR	Punishing Responses
Sol	Solicitous Responses
DR	Distracting Responses
Activ	Activity
PSE	Pain self-efficacy
SAng	State Anger
TAng	Trait Anger
AngIn	Suppressed Anger
AngOut	Expressed Anger
Iloc	Internal Health Locus of Control
GSE	General Self-efficacy
Depression	Depression
Dur	Pain duration

Table 5.11 Matrix of Pearson's product moment correlations among cognitive variables for chronic back pain participants (N=105).

	Int	LC	SS	PR	Sol	DR	Activ	PSE	SAng	TAng	AngIn	AngOut	ILoc	GSE	Dep	Dur
PSv	**.67*****	-.18	.14	.23	.01	.00	**-.23***	**-.52****	**.33*****	.15	.17	.07	**-.23***	-.18	**.57*****	.05
Int		-.09	**.30****	**.35****	.05	.15	**-.25****	**-.65*****	**.31****	.08	.13	-.02	**-.26****	**-.24***	**.49*****	-.12
LC			.02	-.03	-.03	.07	**.29****	**.39****	**-.30****	-.10	-.15	.01	.17	.11	**-.27****	.04
SS				.16	.73**	.50**	.10	**-.23***	.09	.05	.11	.08	-.10	-.05	.09	-.05
PR					.12	.22*	-.03	**-.29****	**.26****	.14	.09	.04	-.02	**-.33****	**.23***	-.07
Sol						.65**	.17	-.12	-.01	.03	.03	.09	-.09	.15	-.03	-.06
DR							.24*	-.04	.07	.05	-.01	.12	.05	-.07	.08	**-.22***
Activ								**.36****	.02	.13	.09	.09	**.22***	.18	-.19	.02
PSE									**-.25***	-.05	-.16	.10	**.35*****	**.24***	**-.42*****	-.07
SAng										**.39*****	**.49*****	**.345*****	**-.20***	-.13	**.45*****	-.04
TAng											**.41*****	**.62*****	-.15	**-.27****	**.22***	-.12
AngIn												**.25****	-.14	-.18	**.30****	.09
AngOut													-.14	-.18	.15	.02
ILoc														.08	**-.20***	.01
GSE															**-.39*****	.13
Dep																-.08

*** Correlation is significant at the 0.001 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Significant correlations are highlighted and as can be seen from Table 5.11, depression is significantly correlated with pain severity (0.57), unlike the non-significant correlation of 0.31 from Study 1. The respective Confidence Intervals were checked for these two correlations to determine if it was possible they were from the same population. They were for $r = 0.57$; CI [95] = [.43, .69] and for $r = 0.31$; CI [95] = [-0.06, 0.60]. The CI for the small sample does contain the point estimate for the large sample, however, if the first sample had been larger, the CI would have been narrower, the new CI would not contain 0.57 and it could be said that the groups were different. As it is, it is only just plausible that these two correlations are estimating the same population correlation. Depression is also positively and significantly correlated to interference, state anger, trait anger and suppressed anger, while it is negatively and significantly correlated to life control, pain self-efficacy, internal health locus of control and general self-efficacy. This suggests that cognitive-behavioural variables may play a role in the effect on depression. To test the hypothesis further, path analysis is used to determine whether mediating variables are operating and this will provide information about possible causal relationships.

As can be seen from Table 5.11, the two key elements of the proposed model, pain severity and depression are significantly correlated ($r = 0.57$, $p < 0.001$). Further inspection of the correlation matrix reveals the variables suitable for path analysis modelling. Only those variables that are significantly correlated with <u>both</u> pain severity and depression, are of interest here. Moreover, only those which reached a magnitude of 0.30, at $p<0.01$, are included, as correlations of less than this are considered weak given the sample size (Bryman & Cramer, 1997). It can therefore be seen that the variables suitable for inclusion in a model of chronic pain and depression for the 105 participants are interference, pain self-efficacy and state anger. Even though general self-efficacy is significantly correlated with depression it is not significantly correlated with pain severity so is not included in the path analysis.

To produce a path analysis model, four multiple regressions are performed. In the first three equations, each potential mediator is regressed onto the independent variable

(pain severity). It is recognized that these three beta values are the corresponding correlation values. In the fourth equation, the dependent variable (depression) is predicted by each potential mediator variable (interference, pain self-efficacy, state anger) and by the independent variable (pain severity). As previously described in Chapter 4, all four variables are entered into the equation. The regressions produced the values shown in Figure 5.1. Table 5.12 gives details of the main regression with all four predictor variables in the equation.

Table 5.12. Multiple regression analyses for path diagram for chronic back pain participants (N=105).

Predictor Variables	B	CI [95]	Beta	t	R^2	AdjR^2	F (df)
Equation 1 DV: Interference							
Pain severity	.70	.55, .85	.67	9.17***	.45	.44	84.22*** (1, 103)
Equation 2 DV: Pain self-efficacy							
Pain severity	-4.78	-6.32, -3.24	-.52	-6.15***	.26	.26	37.84*** (1, 103)
Equation 3 DV: State Anger							
Pain severity	.96	.43, 1.49	.33	3.58**	.11	.10	12.85** (1, 103)
Equation 4 DV: Depression							
Pain severity	2.57	1.08, 4.07	.36	3.42***	.42	.40	18.41*** (4, 100)
Interference	.67	-.92, 2.27	.10	.84			
Pain self-efficacy	-.01	-.24, .08	-.10	-1.01			
State Anger	.69	.29, 1.09	.28	3.46***			

**p< 0.01; *** p< 0.001.

The regression output which determined the path coefficients for depression showed that the four independent variables, pain severity, interference, pain self-efficacy and state anger together accounted for 42%[11] of the variance (40% Adjusted; F(4, 100) = 18.41, p<0.001). According to Cohen (1992), the effect size for this test (f^2) is equal to .72, for the fourth equation, which is a large effect size.

The three indirect effects of pain severity via the three tested mediators are each small in this case: via interference, 0.07 (i.e. 0.67*0.10); via pain self-efficacy, 0.05 (i.e. –0.52*-0.10) and via state anger, 0.09 (i.e. 0.33*0.28). These indirect effects of pain severity on depression are each low, though they do combine to have some influence. The

[11] (see also Footnote 7, Chapter 4, page 132) With a larger sample size and therefore more degrees of freedom then it is less likely that there will be much difference between R^2 and Adjusted R^2 and usually R^2 is reported.

largest effect in this case, however, is the direct effect (after indirect effects have been partialled out), of 0.36, as shown in Figure 5.1.

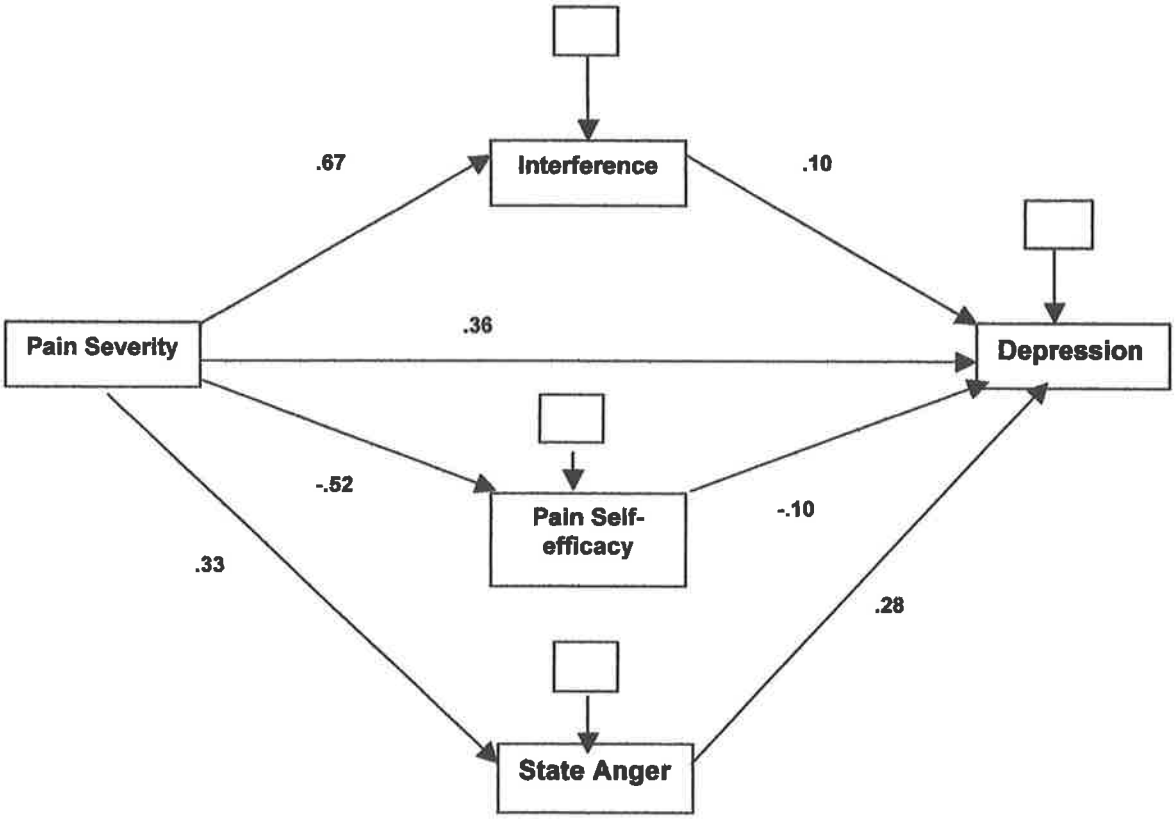

Figure 5.1. Cognitive-behavioural mediation model of chronic pain and depression for chronic back pain participants (N=105).

The findings indicate that Hypothesis 5 has been partially supported. That is, chronic pain and depression are directly related and there also is some indirect effect on depression from chronic pain via the mediating variables, interference, pain self-efficacy and state anger.

It is acknowledged that gender differences may cloud this overall picture. This possibility is investigated in the following section. In order to test the following hypothesis, path analysis models are developed for the male and female chronic back pain participants.

This hypothesis is based on previous research, by Haley et al. (1985), and Timmermans and Sternbach (1976), which suggested that males were more affected than females, by the interference of pain, in terms of developing depression. In contrast, females were more likely to develop depression as a direct result of reported pain severity (Haley et al., 1985).

Hypothesis 6: There will be gender differences in the relationship between chronic pain severity and depression. For males, chronic pain and depression are more likely to be indirectly related and mediated by cognitive-behavioural variables, particularly interference. For females, the relationship is more likely to be direct such that chronic pain severity and depression will be positively and significantly correlated.

To test this hypothesis, comparisons between genders were undertaken with Independent sample t-tests and Pearson's product moment correlations were performed separately for males and females. Refer to Tables 5.13, 5.14 and 5.15 for details. As mentioned previously, although some gender differences were expected, it was considered appropriate to consider the total sample in initial analyses above, as most previous research has not considered gender differences. Conducting analyses on the total sample was necessary to allow comparison with such research. It is acknowledged that be dividing the data into male and females groups, the groups are small and that power may be reduced. However, power analysis will be conducted to determine whether sample sizes are adequate.

Table 5.13. Comparisons between male and female chronic back pain participants: Means, standard deviations and t-tests for between-group differences.

Variables	Male chronic back pain group (n=41)		Female chronic back pain group (n=64)			
	Mean	SD	Mean	SD	t	d
Pain severity	2.89	1.40	3.31	1.44	-1.50	.29
Interference	3.51	1.52	3.28	1.48	0.77	.15
Life Control	3.65	.90	3.46	.86	1.10	.21
Social support	3.51	1.62	3.09	1.94	1.15	.23
Punishing Rs	1.92	1.78	1.32	1.37	1.84	.39
Solicitous Rs	2.85	1.65	2.65	1.78	0.58	.11
Distracting Rs	2.03	1.38	1.36	1.33	2.44*	.50
Activity	2.88	0.95	2.9	0.75	-.12	.02
Pain self-efficacy	36.32	14.22	40.55	12.38	-1.61	.32
Internal Health locus of control	27.15	4.36	24.31	5.23	2.89**	.58
General self-efficacy	83.93	17.62	88.47	13.99	-1.46	.29
State Anger	11.98	4.29	11.81	4.07	.19	.04
Trait Anger	16.8	6.21	18.36	5.64	-1.32	.26
Suppressed Anger	16.34	5.74	16.27	4.63	0.07	.01
Expressed Anger	13.93	4.2	14.03	4.13	1.12	.02
Depression	14.73	9.93	16.48	10.49	-.85	.17
Pain duration	184.3	146.7	175.2	134.1	0.33	.06

*p<0.05; **p<0.01.

Table 5.14 Matrix of Pearson's product moment correlations among cognitive variables for male chronic back pain participants (n=41).

	Int	LC	SS	PR	Sol	DR	Activ	PSE	SAng	TAng	AngIn	AngOut	ILoc	GSE	Dep	Dur
PSv	.78**	-.03	.04	.33*	-.23	.02	-.38*	-.53**	.17	.16	.02	.15	-.25	-.45**	.52**	-.02
Int		-.01	.20	.42**	-.23	.06	-.33*	-.69**	.25	.09	.02	.03	-.47**	-.55**	.61**	-.02
LC			.14	-.11	.05	.22	.39*	.46**	-.24	-.18	-.09	.15	.30	.24	-.06	-.05
SS				.18	.70**	.47**	.24	.02	-.25	-.11	-.04	-.07	.03	.04	-.04	.11
PR					.11	.28	-.16	-.33*	.19	.12	.06	-.01	-.29	-.40**	.34*	-.10
Sol						.59**	.28	.24	-.38*	-.13	-.00	-.07	.15	.26	-.18	-.05
DR							.35*	.20	-.12	-.05	.08	.07	.05	-.02	.16	-.15
Activ								.44**	-.06	.10	.15	.13	.29	.37*	-.27	.22
PSE									-.21	-.07	.08	.09	.44**	.47**	-.44**	-.05
Sang										.42**	.69**	.35*	-.43**	-.23	.50**	.03
Tang											.48**	.52**	-.29	-.25	.04	-.09
AngIn												.59**	-.25	-.10	.20	.13
AngOut													-.30	-.09	.04	.07
Iloc														.24	-.31*	.16
GSE															-.57**	.09
Dep																-.15

**Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).

Table 5.15 Matrix of Pearson's product moment correlations among cognitive variables for female chronic back pain participants (n=64).

	Int	LC	SS	PR	Sol	DR	Activ	PSE	Sang	Tang	AngIn	AngOut	Iloc	GSE	Dep	Dur
PSv	**.63****	**-.26***	.23	.23	.16	.05	-.12	**-.57****	**.45****	.12	**.29***	.02	-.18	-.01	**.59****	.11
Int		-.17	**.35****	**.28***	.21	.18	-.19	**-.62****	**.34****	.09	.22	-.06	-.20	.02	**.43****	-.19
LC			-.07	.01	-.09	-.08	.21	**.37****	**-.35****	-.02	-.20	-.08	.06	.02	**-.39****	.09
SS				.12	**.75****	**.51****	.02	**-.35****	**.28***	.16	.21	.16	-.21	-.09	.15	-.15
PR					.09	.11	.11	-.21	**.32***	.22	.12	.08	.06	-.22	.19	-.06
Sol						**.70****	.09	**-.35****	.21	.15	.05	.19	-.23	.08	.06	-.06
DR							.16	-.17	.20	.18	-.08	.17	-.05	-.05	.06	**-.29***
Activ								**.28***	.09	.17	.02	.06	.19	.00	-.14	-.17
PSE									**-.28***	-.07	**-.37****	.10	**.39****	-.01	**-.44****	-.07
SAng										**.37****	**.34****	**.35****	-.09	-.05	**.43****	-.09
TAng											**.36****	**.69****	-.03	**-.33****	**.34****	-.12
AngIn												-.01	-.08	**-.26***	**.38****	.05
AngOut													-.06	**-.25***	.22	-.02
ILoc														.06	-.11	-.09
GSE															**-.29***	.17
Dep																-.04

**Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

As can be seen from Table 5.13, males reported that their spouses or partners tried to distract them from their pain significantly more than females reported their partners as engaging in distracting responses (medium ES). Males also reported significantly greater internal health locus of control (medium to large ES). There were no significant differences between males and females on any other scale.

Tables 5.14 and 5.15 show correlations for males and females, respectively. The meanings for variable abbreviations are shown in Table 5.11. For both genders, pain severity and depression were significantly and positively correlated. As can be seen from Table 5.14 for males, the correlation between pain severity (predictor variable) and depression (outcome variable) was statistically significant ($r = 0.52$, $p < .01$). The correlation matrix shows the cognitive variables suitable for the path analysis modelling for the male participants. The same criteria were used to select these as with the basic model for all participants: that is, the variables must be significantly correlated with both the predictor and outcome variables. Therefore, for this sample of males, the suitable potentially mediating variables are: interference, pain self-efficacy and general self-efficacy. These three will be tested as mediators in a proposed model.

As with the general model, the male-specific path model assumes that there may be direct and indirect effects. It is therefore assumed that chronic pain may affect depression directly and indirectly via interference, via pain self-efficacy and via general self-efficacy. Each of the path values is a standard partial regression coefficient (Beta). The same procedure of multiple regression is carried out as was carried out for the general model. The regression analysis produced the values shown in Table 5.16. The complete model is shown in Figure 5.2.

Table 5.16. Multiple regression analyses for path diagram for males (n=41).

Predictor Variables	B	CI [95]	Beta	t	R^2	AdjR^2	F (df)
Equation 1 DV: Interference							
Pain severity	.85	.63, 1.07	.78	7.92***	.62	.61	62.69*** (1, 39)
Equation 2 DV: General self-efficacy							
Pain severity	-5.66	-9.31, -2.01	-.45	-3.14**	.20	.18	9.86** (1, 39)
Equation 3 DV: Pain self-efficacy							
Pain severity	-5.43	-8.22, -2.65	-.53	-3.95***	.29	.27	15.58*** (1, 39)
Equation 4 DV: Depression							
Pain severity Interference General self-efficacy Pain self-efficacy	.58 2.51 -.19 .002	-2.28, 3.44 -.65, 5.69 -.36, -.02 -.22, .27	.08 .38 -.34 .04	.41 1.61 -2.31* .21	.46	.40	7.62*** (4, 36)

**p< 0.01; *** p< 0.001

The multiple regression equation which determined the path coefficients for depression showed that the four independent variables, pain severity, interference, general self-efficacy and pain self-efficacy together accounted for 46% of the variance (F (4, 36) = 7.62, p<0.001). According to Cohen (1992), the effect size for this test (f^2) is equal to .85, for the fourth equation (i.e. total model), which is a large effect size. This shows that there are adequate cases in this sample to show a significant effect when one is present.

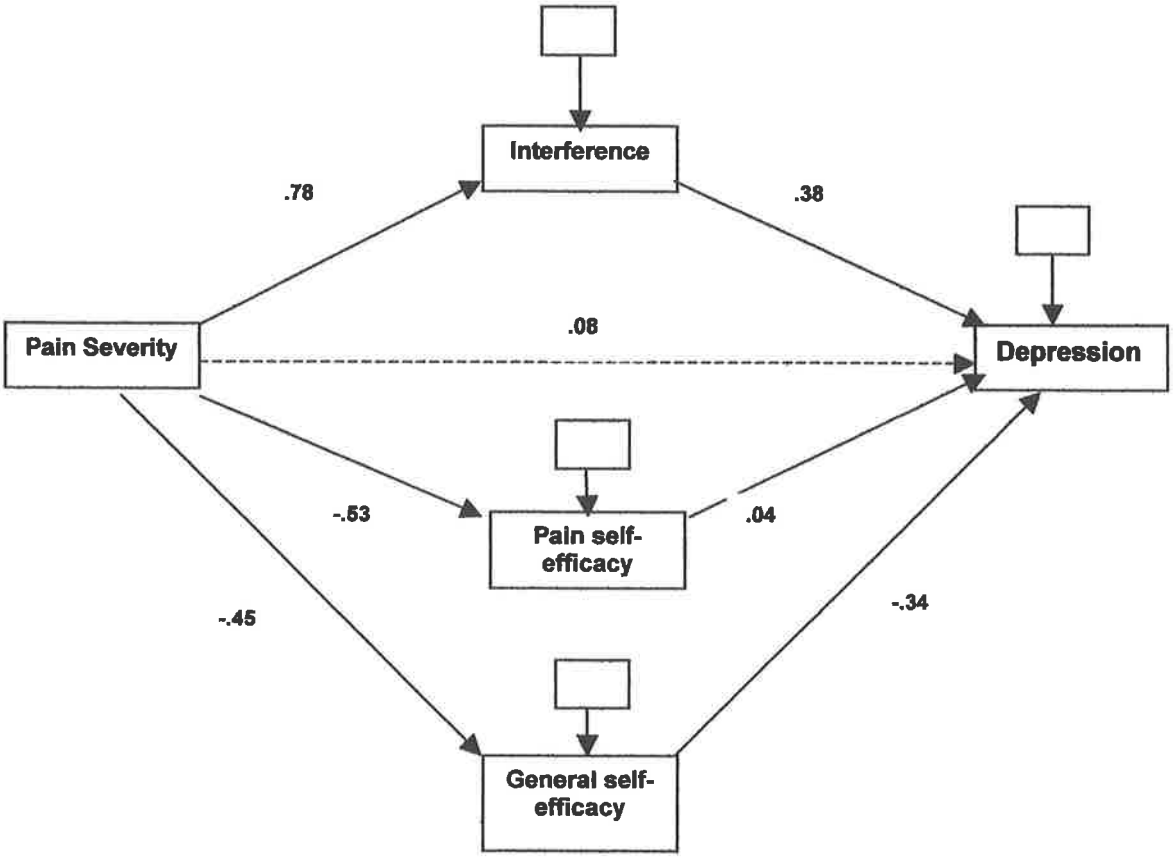

Figure 5.2. Cognitive-behavioural mediation model of chronic pain and depression for male chronic back pain participants (n=41).

As can be seen the direct effect of pain severity on depression is only 0.08 for males. For males, it is clear from Figure 5.2 that the relationship between pain severity and depression is mainly indirect, via the mediating effects of interference and general self-efficacy. This size of these effects is 0.30 (i.e. 0.78*0.38) and 0.15 (i.e. –0.45*-0.34), respectively. Pain self-efficacy had a negligible effect in the model of 0.02 (i.e. –0.53*0.04). Thus most of the effect derives from pain severity indirectly influencing depression through the two mediators, interference and lower general self-efficacy.

It may be considered odd that pain self-efficacy had a negligible mediating effect in the model, whereas the variable general self-efficacy had a marked effect, reflecting the different beta values (see Table 5.16). Such a result can occur with multiple regression,

since these two variables are fairly similar (r=0.47). Therefore, one variable (general self-efficacy) accounts for predicted covariance of this kind, leaving little effect for the similar predictor (pain self-efficacy).

It is acknowledged in presenting these regression models that all included variables are inter-correlated to a moderate extent. There may therefore be strong changes in beta values based on quite minor sampling variations in relative correlation values (e.g. the problem of multicollinearity as mentioned in Chapter 4 (Bryman & Cramer, 1997; Lewis-Beck, 1980). Whether the models prove reliable in their detail will depend on further replication with other samples.

The third model investigated the relationships between variables for the female participants only. There were 64 female chronic back pain participants. Investigating Pearson's product moment correlations between the variables of interest for the present sample is the first step in examining this third model. As can be seen from Table 5.15, the correlation between pain severity (predictor variable) and depression (outcome variable) for females was high and statistically significant ($r = 0.59$, $p < .01$). As with the general model, the female-specific path model assumes that there may be direct and indirect effects. It is therefore assumed that chronic pain may affect depression directly, and indirectly via interference, via life control, via pain self-efficacy, via state anger and, via suppressed anger. Each of the path values is a standard partial regression coefficient (Beta). The same procedure of multiple regression was carried out as for the general and male-specific models. The six regression analyses produced the values shown in Table 5.17. The complete model is shown in Figure 5.3.

Table 5.17. Multiple regression analyses for path analysis for females (n=64).

Predictor Variables	B	CI [95]	Beta	t	R^2	AdjR^2	F (df)
Equation 1 (DV: Interference)							
Pain severity	.65	.45, .85	.63	6.43***	.40	.39	41.34*** (1, 62)
Equation 2 (DV: Life control)							
Pain severity	-.15	-.30, -.01	-.26	-2.10*	.06	.05	4.39* (1, 62)
Equation 3 (DV: Pain self-efficacy)							
Pain severity	-4.91	-6.69, -3.12	-.57	-5.48***	.33	.32	30.07*** (1, 62)
Equation 4 (DV: State anger)							
Pain severity	1.27	.63, 1.91	.45	3.97***	.20	.19	15.77*** (1, 62)
Equation 5 (DV: Suppressed Anger)							
Pain severity	.93	.15, 1.72	.29	2.39*	.08	.07	5.72* (1, 62)
Equation 6 (DV: Depression)							
Pain severity Interference Life control Pain self-efficacy State Anger Suppressed Anger	2.91 .49 -2.59 .005 2.34 .39	.91, 4.93 -1.48, 2.46 -5.28, .09 -.24, .23 -.37, .84 -.09, .89	.40 .07 -.21 -.01 .09 .18	2.90** .50 -1.93 -.05 .77 1.62	.46	.41	8.21*** (6, 57)

*p<0.05; **p<0.01; ***p<0.001

As can be seen the direct effect of pain severity on depression is 0.40, while the indirect effects of pain severity on depression are each negligible. The six effect coefficients, calculated as before, for interference, life control, pain self-efficacy, state anger and suppressed anger are 0.04, 0.05, 0.006, 0.04, and 0.05, respectively. These data imply that most of the effect on depression derives directly from pain severity.

The final multiple regression equation showed that the six independent variables, pain severity, interference, life control, pain self-efficacy, state anger and suppressed anger together accounted for 46% of the variance ($F(6, 57) = 8.21$, $p<0.001$). Only the beta value (0.40) for pain severity was statistically significant, for females. The beta value for life control did approach significance ($p = 0.58$) and may have been significant with more cases. However, according to Cohen (1992), the effect size for this test (f^2) is equal to .85, for the sixth equation, which is a large effect size, indicating that the sample size is adequate to show a significant effect.

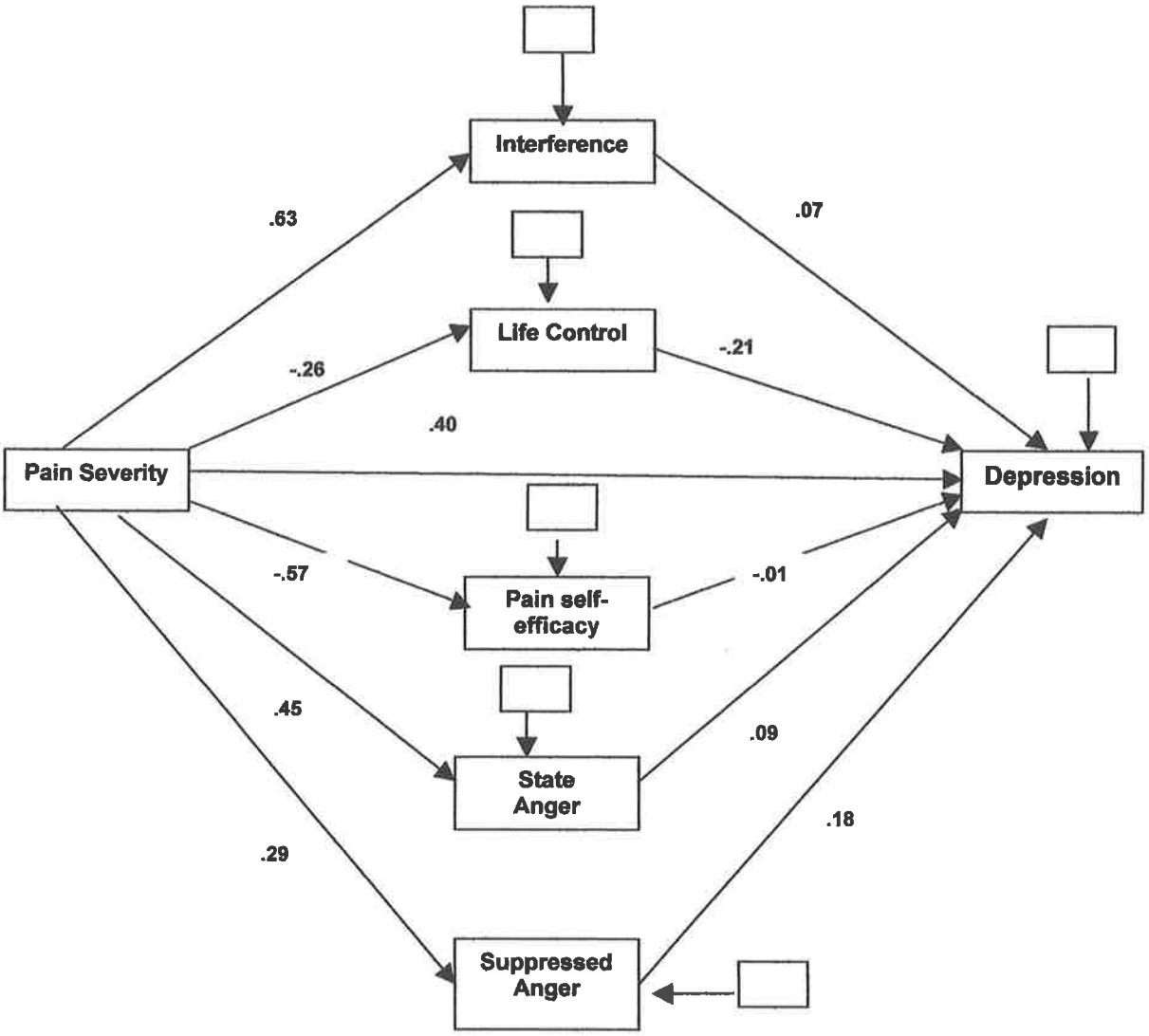

Figure 5.3. Cognitive-behavioural mediation model of chronic pain and depression for female chronic back pain participants (n=64).

The main effect on depression is originating from pain severity and there is only minimal effect from the mediating variables. Examining the path model shows that the indirect effects do not translate to substantial effects on depression, with the direct effect partialled out. The beta values for paths from interference, pain self-efficacy and state

anger to depression are very small indicating that their final impact on depression is minimal.

For the males in this study, depression was more likely to result when pain was perceived to be an interference and when general self-efficacy or confidence in dealing with life was low. In contrast, depression was more likely to be experienced by females as a direct result of the severity of chronic pain with some minor influence from poor control and increased levels of suppressed anger. These results therefore support Hypothesis 8. It would seem therefore that there are some gender differences in the response to chronic pain in terms of depression.

5.5 Discussion

The following discussion briefly covers the findings of Study 2 in the context of the specific hypotheses tested and will be referred to in the more comprehensive discussion presented in Chapter 7 which will incorporate findings from all studies.

5.5.1 Representativeness of the Sample

There was some concern about how representative the sample was in terms of its 'non-clinical' nature. Every effort was made to acquire such a sample, defining it in terms of 'not having attended or being referred to a pain clinic'. It is not clear from the literature how else a non-clinical sample may be defined, as it is accepted that only those most severely disabled by pain are referred to pain clinics by general practitioners (Elliott et al., 1999; Jensen et al., 1991). According to information provided by the ABS on the 2001 National Health Survey, taking medication, especially for pain relief, is one of the most common health related behaviours of Australians. For example, in 1995, nearly 60% of the population was reported to have taken recent medication and that it was commonly for pain relief. In the present sample, about 53% of the people with chronic back pain were taking prescription medication for pain relief.

Other information provided by the ABS showed that about a quarter of the Australian public had routinely visited a general practitioner or specialist in the previous two weeks. In the present research, 85% of the sample had visited a professional health care worker about their pain but information about when that had occurred was not available. It is not possible to determine whether this sample was making more or less visits to health care workers or taking more or less medication than average Australians. It is interesting to note that the Australian Health Survey revealed that women are two to three times more likely to visit an allied health professional than are men. In the present research, the majority of the participants were women and they demonstrated a preference for health professionals such as chiropractors and acupuncturists, which is somewhat in keeping with the national preference. These inadequacies could be overcome in future research by being more specific in the questions about health behaviours, similar to the type of information that is gathered by such organizations as the ABS in national health surveys.

From the limited amount of normative data that was available, it was shown that the present sample appeared to be less dysfunctional than other clinical pain groups and more depressed than one non-pain group. The latter would be expected given that those in chronic pain are likely to be more depressed than those not in pain (Banks & Kerns, 1996; Magni et al., 1993). Their general self-efficacy was significantly better than a non-pain group and their internal health locus of control was about the same. They scored on the anger measures about the same as a non-pain group, except for exhibiting less trait anger.

In terms of the chronic pain profile classification, they were much less able, as a group, to be classified into the three categories that clinical groups have routinely been classified into. Compared to a control group specific to this research, they were more depressed but their anger scores, internal health locus of control, and general self-efficacy were not different to the control group. None of this sample was considered by their general practitioners to be experiencing chronic pain so badly that they needed to be referred to a pain clinic. Given all these disparities, the present sample is not considered to be a clinical sample. To that end, it is considered to be a community or 'non-clinical' chronic pain group.

5.5.2 Limitations of the research

Acquiring participants for this study was as difficult as for the pilot study. Although the literature reports that most adults suffer with chronic back pain at some point in their lives and anecdotally, complaints about such pain appear to be high among the general public, people were mostly reluctant to take part in this type of research. It was found that while people with chronic pain were eager to share their experiences in the telephone interviews, they were not so interested in completing questionnaires. This was even more apparent when they discovered that there was no actual treatment for chronic pain being provided. The advertisements for volunteers did not indicate that treatment would be provided, but most people who made initial contact appeared to expect it.

A related problem was the participants were self-selected and therefore, non-random. Resource restraints and strict criteria for participation also reduced the number of eligible participants. Wide-spread advertising was conducted to ensure as representative, community sample as possible. Even though the sample sizes were smaller when subdivided, similar studies are routinely published in the literature, suggesting that acquiring suitable participants for this type of research is an on-going problem.

All questionnaires were self-report and despite the advantages of their use (see Literature Review, section 2.2.1), there are obvious disadvantages, such as problems with bias and inaccuracies. For example, the Anger inventory especially caused problems with participants. This author received both written and verbal (telephone) complaints about the difficulty of completing this questionnaire. In another example, the section in the PIQ about times that a professional had been seen, duration of visits etc. was very poorly completed. In addition, some of the questionnaires relied on the respondents' memory and it is debateable how accurate these responses were in some cases. Memory is also mood-dependent, so if pain affects mood, it is likely to also impact on memory (Eich, Rachman, & Lopatha, 1990).

It has been suggested that more objective measures of adjustment, such as observation by spouses and trained observers (Jensen et al., 1999), might overcome some

of the problems with self-report questionnaires. Such tools were not available in the present research, although other objective measures such as rest, medication and exercise were used. However, even these were not necessarily more illuminating and again, relied on self-report. It is not clear how such problems could be over-come in this type of research apart from adopting extensive observational methods which would need to be tested and this raises the question of resource suitability, availability and cost. Jensen et al. (1999) found that even using such measures does not always fully explain relationships between patient beliefs and functioning.

While it was considered useful to compare the people with chronic pain with a control group of people without pain, there is some concern about the composition of the group and whether there were enough for meaningful comparisons. The control group was matched on age, gender and marital status but was found to be significantly more educated. This relates to the availability of the sample. Most were acquired by word of mouth from the author and colleagues, as the media campaign was largely unsuccessful. Of the 99 people who volunteered to act as controls, only 66 could be matched to the people with pain. How this might be addressed in future research is discussed below. As it happens, in this study, education was found not to affect depression scores.

Finally, this study was cross-sectional. Although the analyses conducted were informative, they did not allow for causality to be addressed. Cross-sectional analyses are unable to inform about the relationships between pain severity, beliefs and long-term adjustment or lack of it. It is hoped that the longitudinal follow-up study to be presented in Chapter 6 will address some of the limitations of cross-sectional research.

5.5.3 The chronic pain profile classification system

The first hypothesis was not supported as only 59% of the participants could be classified into one of the three patient subgroups identified by Turk and Rudy (1988). While two of the groups, 'interpersonally distressed' and 'adaptive copers' were classified in similar proportions to previous research, the notable discrepancy was the percentage of

people who could be clearly classified as 'dysfunctional'. In the present study only about 9% of them were in that category. Turk and Rudy (1988), by comparison, found proportionally more clinical patients (about 43%) could be classified as such and several other studies by Turk and colleagues (Rudy et al., 1989; Talo, 1992; Turk et al., 1995; Turk & Rudy, 1990) have replicated these findings. The fact that only 9% were classed as dysfunctional suggests that this group was more non-clinical than clinical, which counters the previous comments about their medication useage and visits to health professionals, which suggested that they were more 'clinical' than expected.

The fact that 41% of the present sample could not be readily classified into any of the three major sub-groups typical of clinical research participants was further support for the conclusion that this sample was non-clinical. The previous work by Rudy and colleagues has reported few, if any, clinical patients who could be classified into the unanalysable, hybrid or anomalous categories.

The percentage of unanalysable in the present sample could be a reflection of lack of 'significant others', as two-thirds of them were living alone. The unanalysable category results from too much missing data on two of the nine scales used in the classification system. Those who lack a significant other are not able to complete section II of the MPI, which subsequently results in a high proportion of missing data. However, they may be receiving valuable social support from others with whom they do not cohabit. This is not clearly testable, as the scales of the MPI were calculated by a separate computer program. This suggests that the MPI may need updating and this is discussed further in Chapter 7.

5.5.4 Cognitive functioning in people with chronic pain versus non-pain controls

Hypothesis 2 was partially supported and is consistent with previous studies. About 34% of those with chronic pain were depressed, compared to about 17% of those without pain. Although limited research has compared depression between pain and non-pain persons, Magni et al. (1993) found that among depressed persons in the wider community, there were more that were in pain than not. Banks and Kerns (1996) and Fishbain et al.

(1997) also concluded that depression is more common in people who suffer persistent pain than those who do not. Blyth et al. (2003) reported about 22% of Australians with chronic pain were depressed, although this does not clearly inform about sub-groups of people with chronic pain, such as those with chronic back pain. In addition, the national depression initiative, beyondblue, reports that about 21% of the general public is depressed.

However, the hypothesis could not be fully supported. While the pain group recorded a slightly lower level of internal health locus of control than the non-pain control group, it was not significantly so. In addition, both groups recorded high scores on the internal health locus of control scale. This suggests that either the pain group had adapted to their condition and could cognitively protect themselves against personal failure (Skevington, 1983), or again it is another piece of evidence that the pain group does not represent the clinical patients that much of the previous research has targeted.

Likewise, there was no significant difference in general self-efficacy scores for the two groups. Although the highest possible score for this measure is 119, there is no indication in the literature of what is considered 'high'. In the present research, the pain group and non-pain group averaged around 87 and 89 respectively. This seems to reflect average to high general self-efficacy, but it is not possible to make more precise conclusions. In any event, chronic pain did not affect confidence in general functioning. In future research, a closer inspection of the scale items may provide further clues.

The finding that the chronic pain group did not differ to the non-pain group on any of the measures of anger is not consistent with previous research. Several previous studies have suggested that trait anger is higher in people with chronic pain (Beutler et al., 1986) and that suppressed anger is also more common in such people (Pilowsky & Spence, 1976). Achterberg-Lawlis (1982) also reported that anger is dominant in the personality profiles of people with chronic pain. Likewise, Hatch et al. (1991) reported that people in pain suppressed anger to a greater degree than did controls but in terms of expressed anger there was no difference. Again, this supports the notion that the particular sample under investigation is different from those people who have participated in most clinical

research. This adds further weight to the evidence that this sample was non-clinical and to idea that generalising from clinical to other groups is problematic.

Although the people with back pain as a whole in this study did not have elevated anger scores, those with more severe pain may have become angry. This is noted in the Conclusion section of this chapter. Another interpretation of this result could be that people who are in pain and prone to anger do not necessarily respond to advertisements for research volunteers. If this was the case, it reflects a bias in the recruitment strategy and not necessarily a true state of affairs with regard to anger among people with pain. Although not conclusive, the author encountered many people with chronic back pain who expressed anger during telephone interviews. Whether they then took part in the research is not clear. How this might be addressed in future research is also unclear. It is also noted that when the responders to the follow-up study were compared to those who did not respond, the latter group were found to have reported greater trait anger.

5.5.5 Depression versus non-depression among those with chronic back pain

Among the 105 people with chronic pain who took part in this study, approximately 34% scored as 'depressed' on the CES-D. This is high given that a higher cut-off point was used than is normally used for diagnosing clinical depression (Radloff, 1977). According to Turk and Okifuji (1994), this higher cut-off point reduces the risk of over-diagnosis of depression in medical conditions where there is an overlap of somatic/vegetative symptoms. These might include insomnia, somatic preoccupation, weight changes, anorexia, fatigue and psychomotor retardation (Cavenaugh, Clark, & Gibbons, 1983; Schwab, Bialow, Brown, & Holzer, 1967). The percentage of depressed in the present study is consistent with considerable previous research findings (Brown, 1990; Haythornthwaite et al., 1991). It is considerably higher than the 16% reported by Magni et al. (1993) in one of the few surveys that has been undertaken in a non-clinical population, however it is consistent with the work of Banks and Kerns (1996). This finding is important, given that the weight of evidence suggested the sample was non-clinical. It implies that the extent of depression in the

community may not be truly known and is an indication that more research into depression associated with chronic illness, is warranted.

Contrary to Hypothesis 3, there were no significant demographic differences between the depressed and non-depressed groups, except for age. The depressed group was, on average, significantly younger than the other group. This is consistent with findings by Haythornthwaite et al. (1991) who concluded that those who develop chronic pain as young people are more likely to develop depression in the long-term. Being restricted physically at a younger age may certainly be more challenging to emotional well-being when expectations may be higher.

The current finding is consistent with most previous research that has reported no gender differences in depressive symptoms among pain patients (Haley et al., 1985; Haythornthwaite et al., 1991; Marbach et al., 1983). This is despite research findings that women seek treatment more, and are more prone to developing depression, while in pain (Bush et al., 1993; White & Harth, 1999). Jensen et al. (1994) found that Swedish women are more vulnerable to the long-term effects of sickness and disability associated with chronic pain. Results of the present research do not support this. Novy et al. (1996) also did not find gender differences in total depression scores, but reported differences at the specific item level when using the BDI. They found that women were significantly more disturbed by body shape changes resulting from long-term pain, as well as levels of fatigue. It is possible that further investigation at the specific item level in the present group may yield gender differences, although the CES-D does not clearly measure beliefs about body image, per se. Further investigation of this point is beyond the scope of the present research.

Failure to find other demographic differences such as in years of education and employment status is consistent with previous research by Haythornthwaite et al. (1991). However, Magni et al. (1994) reported that low education and unemployment were both associated with depression resulting from persistent pain, and at follow-up only unemployment continued to predict depression. In the present study, the majority of

participants were middle-aged married females while approximately a third of all participants responded to employment status with 'retired' or 'home duties'. As such they may not be as vulnerable to lack of education and employment opportunities as some other members of the community may be. This finding suggests that further investigation with more younger and more male participants may be needed before firm conclusions about gender differences in community people with chronic pain can be made.

There has been limited research on the relationship between duration of chronic pain and depression. It was expected that the longer a person had suffered pain, the more likely it would be that they suffered depression (Swanson et al., 1986). In the present research, the variable 'pain duration' did not correlate significantly with any variables for the group with chronic back pain, except for a small, negative correlation with distracting responses. In accord with the gender ratio of the participants this correlation was repeated in the female data, but not the male data. This lack of relationship between pain duration and depression was confirmed with the later analysis on 'ancient' pain. The average pain duration for the control group was greater than reported by Swanson et al.'s (1986) control group (6.6 years). As expected, the 'ancient' pain group in the present study was significantly older than the control group, but there were no significant differences in terms of other demographics.

There appeared to be only one minor difference between the two groups. Those in the 'ancient' group reported significantly less trait anger than did the control group. Swanson et al. (1986) did not specifically measure trait anger but reported that generally elevated MMPI scores were reflective of "an overall increase in psychopathology" (p. 386). The finding about anger is therefore not consistent with the previous research. Unlike Swanson et al. (1986), the present study found that those with the prolonged chronic condition did not report significantly greater depression or drug dependency. Given that there is little previous research on how chronicity affects psychological functioning, this result shows again that assumptions based on previous clinical research are not necessarily relevant to other groups.

Hypothesis 4 was partially supported. As predicted by previous research, those reporting depression also reported low internal health locus of control, control and pain self-efficacy, and increased interference and anger (Blalock et al., 1989; Burns et al., 1998; Rudy et al., 1988; Skevington, 1983). The finding that their general self-efficacy was also low is expected given that pain self-efficacy and general self-efficacy are likely to affect each other and in Study 2 are correlated (0.24) (Shelton, 1990). With limited or no previous research on general self-efficacy, this study is one of the first to clearly show that chronic pain affects one's general level of confidence in daily functioning.

The finding that social support was not perceived to be significantly different in the two groups did not support previous research (Revenson, 1993). In this sample the depressed group perceived that they had slightly more social support. According to the social support model, more support should result in less depression, while according to the operant model the opposite may be true (Turk et al., 1995). Furthermore, from a cognitive-behavioural view, it depends whether the support is considered by the person in pain to be positive or negative. In line with this, the depressed group did perceive that they received significantly more punishing or negative responses than the non-depressed group which is consistent with Turk et al.'s (1995) study, in which negative responses were more related to an increase in emotional distress. There were no significant differences between the groups in terms of solicitous or distracting responses. This result may have been affected by the gender ratio of the sample. Traditionally, females are more likely to be called upon to provide support to males, and as Schiaffino and Revenson (1995) found, with a predominantly female sample, this situation may require role reversal and considerable frustration on the part of the male spouse. However, the present findings are inconclusive and more research is warranted.

As predicted, the depressed participants reported significantly more state anger than did the non-depressed participants. However, there were no significant differences on trait anger, suppressed or expressed anger between the two groups. In the present group, depressed persons reported greater proneness to anger, but not significantly so. This does

not support previous work by Beutler et al. (1986) who reported that people with chronic pain tend to be more prone to anger. However, that research was based on psychiatric patients unlike the present research, so the studies may not be directly comparable.

The research that has been done on anger management styles has reached conflicting conclusions with regard to how male and females with chronic pain deal with anger and depression. In the present group, to divide participants into depressed and non-depressed groups and then to divide them by gender would make the groups too small for valid comparisons and conclusions. From the research that has been done previously, suppressed anger is considered to be more of a predictor of depression in chronic pain than is expressed anger (Tschannen et al., 1992). While correlations indicated this for the total chronic pain group, suppressed anger between depressed and non-depressed was not significantly different. Burns et al. (1998) found that there was no relationship between depression and anger management style among female patients, while male patients who were depressed reported more expressed anger. Having more males in the present study may have allowed differences to be more clearly demonstrated.

Depressed people with chronic pain were less active than their non-depressed counterparts, which is consistent with most previous research (Rudy et al., 1989; Turk et al., 1995; Turk & Rudy, 1990), but the difference was not significant. Previous research has reported that chronic pain and less activity is more related to depression in males than they are in females (Haley et al., 1985; Timmermans & Sternbach, 1976). More male participants might allow a clearer investigation of this point.

5.5.6. The role of cognitive appraisal in the chronic pain-depression relationship

Consistent with previous research, increased depression was related to greater perceived interference and reduced feelings of control, general self-efficacy and pain self-efficacy (Nicholas, 1994; Rudy et al., 1988; Shelton, 1990). Finding that depression was significantly related to perceived pain severity is consistent with some previous research (Fishbain et al., 1997; Magni et al., 1993), although the correlation in Study 1 did not

achieve statistical significance. Although the Study 1 sample was originally thought to be too small for firm conclusions, power analysis showed that it was enough to provide a significant result. The disparate findings relating to pain severity demonstrate that while there may be a direct relationship between pain severity and depression in some groups, the relationship may be indirect in others.

Depression was not significantly correlated with social support, as noted above with the depressed/non-depressed comparison. This is not consistent with some previous research. A review by Rodin, Craven and Littlefield (1991) and work by Trief et al. (1995) have found that people with chronic pain who receive less social support are more likely to develop depression. In contrast, Schiaffino and Revenson (1995) found with RA patients that social support had no effect on the depression experienced by the patients. A possible explanation for this was provided. Those who initially perceived their condition as a great challenge, but who received positive support, suffered more depression in the long term. This could be due to the possibility that if initial optimism eventually becomes unrealistic, spousal support may not make much of a difference.

There may, however, be another explanation. In the present study, 34% of the sample was single or divorced, separated or widowed. The questionnaire used to measure social support (the MPI: Kerns et al., 1985) is designed to measure support from significant others who live with the individual; it does not account for social support which may be provided by friends and others who do not necessarily live with the person. Previous work by Turner et al. (1987) has indicated that those who seek social support are more likely to be experiencing pain for the first time. The present group had been experiencing chronic pain for about 15 years so this issue may not be as much of a concern as with those who have been more recently chronically impaired by pain. There is also the question of gender. Traditionally, women are the care-givers and most of this sample was female, so it is plausible to question whether women in distress are likely to seek support as much as males in distress.

Of the three 'responses from significant others' variables, only punishing responses showed a significant relationship to any of the key variables. Punishing responses include being ignored or when a significant other shows irritation, frustration or anger with the person with chronic pain. It appeared that those who perceived that punishing responses were high were also more likely to report depression, though the correlation (0.23) was quite low. None of the 'responses from significant others' were significantly correlated to pain severity. The correlation matrix also indicated that more punishing responses were related to higher state anger in the person with chronic pain and less reported pain self-efficacy and general self-efficacy. This gives tentative support to the notion that those living in conflict-ridden environments fare less well in terms of overall adjustment to pain (Schwartz & Kraft, 1999; Trief et al., 1995). However, these results need to be treated with caution, as the significant correlations were low.

Depression was not significantly and inversely related to activity, which is not consistent with previous research (Maxwell et al., 1998; Rudy et al., 1988; Turk et al., 1995), although, again it may be due to the composition of the sample. Sixty-one percent of the chronic back pain group was female and previous research has shown that a relationship between pain and activity is less likely to be demonstrated with females (Haley et al., 1985).

(i) Gender differences in the relationship between chronic pain and depression

For both males and females, depression was significantly related to high pain severity, interference and state anger and to low general self-efficacy and pain self-efficacy, but there were some specific gender differences. This is important given that there has been a dearth of studies that clearly address the issue of gender differences in psychological dysfunction associated with chronic pain (Novy et al., 1996). Explanations for gender differences have been discussed in the previous sections but some more specific details are presented here. For males in the present research, depression was also indicative of less activity while for females, depression related more to reduced control and greater suppressed anger.

Further analysis found that depression in males with chronic pain was mostly predicted by the degree of interference that they perceived pain to make in their lives, while for females it was mostly their reported pain that influenced their depression directly. These findings are consistent with Haley et al. (1985) and Timmermans and Sternbach (1976). This suggests that males are more affected by disruption to their daily lives while women are more affected by the pain itself. This is consistent with the negative and significant correlation between pain severity and activity for males only (-0.38), indicating that as pain becomes more severe, activity is reduced. Gender differences in response to chronic pain need to be acknowledged in terms of education, assessment and treatment.

5.5.7 Conclusion

From the path diagram for all participants, Figure 5.1, it can be seen that pain severity impacts on interference, pain self-efficacy and state anger in the expected directions. That is, with increased perceived pain severity, interference is seen to be high, confidence in dealing with the pain is reduced and state anger elevated. Also in total, high pain severity, high interference, reduced pain self-efficacy and high state anger account for about 42% of the variance in depression. This supports the idea that the chronic pain–depression relationship is multi-dimensional. However, none of the variables has a particularly strong impact on depression. The initial impact of pain severity on interference and negatively on pain self-efficacy is quite strong but this impact does not translate to a particularly strong effect on depression. From the model, it appears that people with chronic back pain in general are as affected by the severity of their pain as they are by the disruption it causes in their lives. However, the effects are somewhat different when looking at males and females separately.

As can be seen Figures 5.2 and 5.3, males respond differently to chronic pain than do females. In particular, the direct path between pain severity and depression is reduced to almost zero for males, suggesting that pain severity itself does not directly impact on depression levels. Figure 5.2 suggests that there is quite a strong mediating effect from

perceived interference, in particular. The correlations (Table 5.14) suggest that reduced actual activity levels may also have a small mediation effect. Males, in particular, are affected by perceived disruption to daily life that persistent pain can cause. While pain self-efficacy is affected by pain severity in the expected direction, i.e. is reduced, this effect does not influence depression levels much.

A different picture emerges when investigating the path diagram based on female data, Figure 5.3. While there are small mediating effects, pain severity appears to be having a mainly direct effect on women's depression levels. In conjunction with the actual severity of pain, women appear to experience a loss of control, which does affect their feelings of depression to a slight degree. Unlike the males, while pain is seen as interfering, this interference does not necessarily have an effect on depression, after partialling out the direct effect of pain severity. The effect on pain self-efficacy is similar to that experienced by men. While feelings of confidence are affected in the expected direction and to quite a degree, this does not translate to an impact on depression, over that produced by the pain severity itself directly. Likewise, while women in pain may feel generally angry inside, this does not necessarily influence how depressed they may feel about their condition. Moreover, like males, if they feel angry when actually experiencing pain, which is a dynamic process, this may have a small added effect on levels of depression.

While this major study has revealed some interesting findings, it cannot provide information about the longitudinal nature of chronic pain, given the cross-sectional design. In order to examine the long-term effects of chronic pain on cognitive functioning, with a specific interest in depression, a five-year follow-up study was designed and conducted. A 5-year follow-up study with 44 of the original participants is presented in Chapter 6.

CHAPTER SIX

STUDY 3: COGNITIVE-BEHAVIOURAL FUNCTIONING OF CHRONIC BACK PAIN PARTICIPANTS FIVE YEARS LATER

6.1 Introduction

This chapter provides details of the investigation into the extent of cognitive-behavioural dysfunction in chronic back pain participants at follow-up, five years after the first studies were conducted. As previously mentioned, one of the puzzles of chronic pain continues to be the temporal relationship between chronic pain and depression - does one precede the other and if so, which occurs first? The 'consequence' hypothesis argues that chronic pain precedes depression while the 'antecedent' hypothesis contends the reverse. One of the ways of clarifying this is to conduct longitudinal research. However, one of the criticisms aimed at chronic back pain research is that most studies are cross-sectional. In addition, previous research has so far been somewhat inconclusive about the relationship between chronic pain and depression in the community over the long term.

A cognitive-behavioural model of chronic pain and depression is an example of a model that explains the relationship in terms of the 'consequence' hypothesis (Fishbain et al., 1997). As most of the research from this perspective has been cross-sectional, it is often difficult to clearly determine the temporal relationship and often the implication of causality is retrospective (Magni et al., 1994). Moreover, most of the studies endorsing a cognitive-behavioural mediation model, have been cross-sectional (Kerns et al., 1985; Maxwell et al., 1998; Rudy et al., 1988).

There has been little published research that clearly addresses this issue. To briefly reiterate, Brown (1990) conducted a longitudinal study with RA patients that involved data collection over a 3 year time period. Brown (1990) determined that the only model which explained his data, albeit tentatively, was the 'pain leads to depression' model. Another

unique study, by Magni et al. (1994), was a longitudinal community survey conducted over 8 years. That research tested two hypotheses, 1) that chronic pain leads to depression and 2) that depression leads to chronic pain. The conclusion was that the two hypotheses are not mutually exclusive and that one may apply to some people while the other may be relevant for others. Moreover, for some with long-term pain, a cyclic, mutually causative process may occur. In keeping with these previous studies, the present research presented a rare opportunity to test these hypotheses with a group of people with chronic pain from the community.

Findings, from Studies 1 and 2 of the present research, have demonstrated a relationship between chronic pain and depression in community samples of chronic back pain participants. These were cross-sectional analyses, however, and do not necessarily provide information about causality, despite the inference of the cognitive-behavioural model. If chronic pain leads to depression as the chronic pain literature widely suggests, then it would be expected that this could be demonstrated in the present sample of chronic back pain participants who have been assessed at two different times, 5 years apart. In addition, it is clear from the earlier results that there are other variables that may mediate the chronic pain-depression relationship thus improving the explanation of the interactions. This study also provides a unique opportunity to determine whether a cognitive-behavioural mediation model is stable over time. That is, is such a model able to consistently explain variance in depression over time? This third study therefore seeks to overcome the limitations of cross-sectional research by analysing data longitudinally.

Initially, the responders to this follow-up study are compared to the non-responders on demographic characteristics, pain-related and cognitive-behavioural variables. Secondly, data acquired from Time 1 for the responders are compared to their responses at Time 2 to determine whether there had been any significant changes in their demographic characteristics and whether their cognitive-behavioural responses differed on the two occasions. Next, the test-retest reliability of the measures used in both Study 2 and 3 are reported, given that this longitudinal study represented an opportunity to do so. Testing of

hypotheses follows, including path analysis techniques. The first hypothesis explores the possibility of mediating variables in the relationship between chronic pain and depression at Time 2. In the second hypothesis, a longitudinal analysis seeks to determine whether chronic pain can be suggested as a precursor to depression in this group. Subsequently, gender differences are examined, although the final size of the male and female groups means that caution needs to be applied in terms of findings. Based on the previous studies of this research and reported findings in the literature, two hypotheses are suggested:

Hypothesis 1: At Time 2, chronic pain severity and depression will be directly linked, with some minor mediating contribution from other variables including perceived interference, life control and pain self-efficacy.

In addition, based on the previous research by Brown (1990) and Magni et al. (1994), a second 'cross-lagged correlations' hypothesis is suggested:

Hypothesis 2: If depression is a consequence of chronic pain, then pain severity (Time 1) should predict depression (Time 2) better than depression (Time 1) predicts pain severity (Time 2).

6.2 Method

As in the previous studies, comparisons between means, correlations, multiple regression and path analyses are utilized.

(i) Participants

The participants included 44 people who had taken part in the second study. They consisted of 16 males and 28 females, aged at Time 2 from 25 to 79 years with a mean age of 50 years and 8 months (SD=14 years). Details of their recruitment are given under Procedure.

(ii) Measures

The variables investigated in this study were assessed by several questionnaires, which were stapled together to form one document, as in the previous studies. The first part of the questionnaire document was the Patient Information Questionnaire as used in Study 2. However, fewer questionnaires were used in this third study than in Study 2. The questionnaires were reduced because it was clear from feedback from Study 2 that many of the participants felt there were too many questionnaires to complete. For example, several questionnaires had been returned uncompleted with notes attached from participants indicating that they felt there was "too much paper work" and they were not prepared to complete it. Under these circumstances it was decided to keep the number of questionnaires to a minimum. The key questionnaires, the MPI and the CES-D were included because they were fundamental to the research. In addition, the PSEQ was included as pain self-efficacy had been implicated in all cognitive-behavioural mediation models from Study 2 and it is a short questionnaire. The anger measures were omitted because there was also considerable negative feedback and complaints about interpretation and completion of the anger inventory. The internal health locus of control and general self-efficacy measures were also omitted, as they did not play a prominent role in the previous studies. Only the following questionnaires were chosen as being the most relevant, based on previous research and the findings from the earlier studies of the present thesis.

1) *Perceived pain severity* as assessed by the pain severity scale from the MPI (Kerns et al., 1985).
2) *Perceived life interference* as assessed with the composite Interference scale from the MPI (Kerns et al., 1985). This consists of the, a) social, b) work, and c) family interference scales.
3) *Perceived life control* as measured by the life control scale from the MPI (Kerns et al., 1985).

4) *Perceived social support* as measured by the Support scale from the MPI (Kerns et al., 1985).

5) *Punishing, solicitous and distracting responses* are also assessed with three scales from the MPI (Kerns et al., 1985).

6) *Activity level* as measured by the General Activity scale from the MPI (Kerns et al., 1985).

7) *Pain self-efficacy* as measured by the Pain Self-Efficacy Questionnaire (Nicholas, 1988).

8) *Depression* as measured by The Center for Epidemiologic Studies Depression Scale (Radloff, 1977).

(iii) Procedure

At the time Study 2 was conducted, more than 95% of the participants had indicated that they would be interested in taking part in future research. Out of the original 105 participants, contact details for 94 were available, i.e. address and/or telephone number. A follow-up letter (Appendix I), to re-establish contact, was sent out to these people, along with an information sheet (Appendix J), the relevant questionnaires and a reply-paid envelope. After 2 weeks a reminder letter was sent to those who had not responded to the first round. Two of the original 105 persons had died during the previous five years. A note received from a relative advised that two people had moved overseas and were not available to take part. Another two people advised that they were too ill to participate. Both had contracted cancer and were undergoing chemotherapy treatment. Five people returned the questionnaires uncompleted, explaining that they preferred not to take part. The rest were returned to the author unopened with a 'return-to-sender' stamp and mostly no reason was given. Despite extensive searching for telephone numbers etc. it was not possible to contact these people any other way. In total, 44 people responded. Therefore, the response rate represented 42% of the original sample from Study 2. The failure-to-return

rate is therefore 58%, which is not quite as good as the 52% reported in the literature for most studies using mailed surveys (see Yu & Cooper, 1983, cited in Brown, 1990).

6.3 Results

Initially, chi-square tests for demographics and pain related variables and an independent samples t-test for age were performed to compare the 44 respondents to the 61 non-respondents in order to determine whether the final follow-up group of respondents was representative of the original 105 participants in terms of demographics and pain-related characteristics. Table 6.1 shows the results.

This is in accord with suggestions by Feinstein (1977) and Cicchetti and Nelson (1994), that conclusions in follow-up research can only be generalized to the original sample, if it is established that the 'retained' cohort (i.e. those who respond to follow-up) does not differ on relevant variables from the 'lost' cohort (i.e. those who do not respond to follow-up). These tests were performed on the reported results from Time 1, i.e. Study 2, for responders and non-responders to Study 3.

As can be seen from Table 6.1, the 44 responders did not differ significantly from the non-responders on age, gender, marital status, education, employment status, drug use or back operations. To determine whether the responders differed significantly from the non-responders on the cognitive-behavioural variables at Time 1, independent t-tests were performed. These are reported in Table 6.2.

The respondent sample is small, implying an increased risk of reporting chance results as significant. While post-hoc correction testing may reduce this, it can be argued that this may not be appropriate (Feise, 2002). Bonferroni testing, for example, may not inform about individual differences. Correction testing may reduce Type I errors but increase the risk of Type II errors. Also, correction testing may imply that statistical significance supersedes research quality. Multivariate testing can also be problematic given that interpretation of results may still necessitate univariate testing. Therefore, correction testing was not performed on the data. It is recognized that these are debatable points.

Table 6.1. Demographic and pain-related characteristics of responders (n=44) compared to non-responders (n=61) at Time 1: Distributions and tests of significance (χ^2, t) of between-group differences.

Demographic Characteristics	Responders (n=44)		Non-responders (n=61)					
	n	%	n	%	χ^2	df	p	Sig
Gender					.23	1	.63	Ns
Male	16	36.4	25	41.0				
Female	28	63.6	36	59.0				
Marital Status					.37	2	.83	Ns
Marr/Defacto	29	65.9	41	67.2				
Single	6	13.6	10	16.4				
Div/Wid/Sep	9	20.5	10	16.4				
Education					1.71	1	.19	Ns
≥Year 12	28	63.6	31	50.8				
<Year 12	16	36.4	30	49.2				
Employment Status					1.97	3	.58	Ns
Full-time	15	34.0	17	27.9				
Part-time/vol	12	27.3	14	22.9				
Unemployed	8	18.2	10	16.4				
Ret/home-duties	9	20.5	20	32.8				
Drug use					1.52	2	.54	Ns
Pres. (inc.nsaids)	24	54.5	32	52.4				
Otc (inc. nsaids)	16	36.4	19	31.1				
No drugs	4	9.0	10	16.4				
Back operations					3.53	1	.06	Ns
No	39	88.6	45	73.8				
Yes	5	11.4	16	26.2				
	Mean	**SD**	**Mean**	**SD**	**t**	**df**	**p**	**sig**
Age (years)	45.6	14.00	45.2	12.2	-.20	103	.84	Ns

Table 6.2. Comparison between responders (n=44) and non-responders (n=61) at Time 1: Means, standard deviations and t-tests for between-group differences.

Variables	Responders (n=44)		Non-responders (n=61)			
	Mean	SD	Mean	SD	t	d
Pain severity	3.21	1.38	3.10	1.48	-.39	.07
Interference	3.38	1.37	3.36	1.58	-.08	.01
Control	3.63	.76	3.47	.95	-.93	.18
Social support	3.41	1.85	3.14	1.82	-.73	.15
Punishing Rs	1.43	1.42	1.65	1.67	.74	.14
Solicitous Rs	2.55	1.68	2.86	1.77	.90	.18
Distracting Rs	1.56	1.46	1.67	1.34	.39	.08
Activity	2.94	.78	2.85	.88	-.55	.11
Pain self-efficacy	39.45	11.84	38.49	14.23	-.37	.07
Internal Health locus of control	26.05	4.93	24.97	5.18	-1.10	.21
General self-efficacy	88.45	14.52	85.43	16.32	-.98	.19
State Anger	10.82	2.69	12.64	4.80	2.27*	.44
Trait Anger	16.07	5.74	18.97	5.74	2.55*	.50
Anger-in	15.66	5.21	16.75	4.96	1.09	.22
Anger-out	13.05	4.33	14.67	3.89	2.02*	.39
Depression	15.20	9.13	16.25	11.06	.52	.10
Pain duration	188.0	160.2	172.11	121.5	-.58	.11

*p<0.05 (2-tailed)

As can be seen from Table 6.2, there were no significant differences on variables except for three of the anger measures (two small ESs, one medium ES). The responders reported significantly less state and trait anger and also significantly less expressed anger. In terms of demographic information, the responders appear to be representative of the entire 105 participants. Since Study 3 did not included anger measures, it is reasonable to regard follow-up analyses as unbiased with respect to the entire original sample of 105 people, although the anger issue will be addressed more fully in the discussion. Table 6.3 shows demographic and pain related variable responses of the responders (N=44) at Time 1, compared to relevant responses at Time 2.

Table 6.3. Demographic and pain-related characteristics of responders at Times 1 and 2 (N=44).

Demographic Characteristics	Time 1		Time 2	
	N	%	N	%
Marital Status				
Married/Defacto	29	65.9	27	61.4
Single/div/sep/wid	15	34.1	17	38.7
Employment Status				
Full-time	15	34	14	31.8
Part-time/volunteer	12	27.3	14	31.8
Unemployed	8	18.2	10	22.7
Retired/home-duties	9	20.5	6	13.6
Drug use				
Presc (inc. nsaids)	24	54.5	14	31.8
Otc (inc. nsaids)	16	36.4	17	38.6
No drugs	4	9.1	13	29.5
Back operations				
No	39	88.6	36	76.7
Yes	5	11.4	8	23.3

As can be seen from Table 6.3, there was little change in demographic characteristics for the responders from Time 1 to Time 2. Two people became single in the 5 years. Three more responders had received back operations in the interim. A rather lower proportion (31.8%) was taking prescribed drugs, and fewer (30%) were doing full or part-time work.

Related sample t-tests were conducted on means of the relevant cognitive-behavioural variables for Times 1 and 2 and are presented in Table 6.4.

Table 6.4. Comparison between responders (N=44) at Time 1 and Time 2: Means, standard deviations and t-tests for between-group differences.

Variables	Participants Responders (N=44)					
	Time 1		Time 2			
	Mean	SD	Mean	SD	t	d
Pain severity	3.21	1.38	2.90	1.46	1.49	.21
Interference	3.38	1.37	3.18	1.22	1.25	.15
Control	3.63	.76	3.98	1.11	-2.20*	.36
Social support	3.41	1.85	3.67	1.51	-.13	.15
Punishing Rs	1.43	1.41	.92	1.23	2.34*	.38
Solicitous Rs	2.55	1.68	2.02	1.78	2.23*	.31
Distracting Rs	1.56	1.46	1.31	1.37	1.19	.18
Activity	2.94	.78	2.93	.90	.09	.01
Pain self-efficacy	39.45	11.84	39.39	12.01	.04	.01
Depression	15.19	9.13	16.68	12.52	-.94	.13

As can be seen from Table 6.4, the responders reported significantly greater control at Time 2 compared to Time 1 and also significantly fewer punishing and solicitous responses. Effect sizes are small to medium (Cohen, 1992). They did not differ significantly on any of the cognitive-behavioural variables. Although not presented in the Table, 32% of the responders indicated a score of 19 or more on the CES-D at Time 2, indicating high-risk levels of depressive symptomatology. This compares to 27% of the same people who had been depressed at Time 1. When this data was checked further, it was found that 50% of those depressed at Time1 still scored as depressed at Time 2. Of the 32% depressed at Time 2, 50% had not scored as depressed at Time 1. As this group amounted to only 14 persons at Time 2, further analyses were not conducted due to small sample size.

Correlations between Time 1 and Time 2 for the 44 responders were also compared and are presented in Table 6.5. As this is also an opportunity to confirm the test-retest stability of the measures used in the research, correlations for all the relevant variables are

presented in bold type[12]. Test-retest stability refers to the long-term consistency of a measure.

Table 6.5. Matrix of Pearson's product moment correlations among cognitive variables for responders at Times 1 and 2 (N=44), with a 5-year interval.

	PSv2	Dep1	Dep2	Int1	Int2	LC1	LC2	SS1	SS2	Activ1	Activ2	PSE1	PSE2
PSv1	**.52****	.53**	.38*	.57**	.43**	-.27	-.15	.13	.35*	-.32*	-.33*	-.41**	-.30
PSv2		.39**	.32*	.39**	.73**	-.23	-.43**	.11	.37*	-.40**	-.41**	-.41**	-.71**
Dep1			**.55****	.45**	.37*	-.38*	-.39**	.08	.01	-.52**	.33*	-.40**	-.34*
Dep2				.22	.24	-.49**	-.65**	-.18	-.15	-.23	-.23	-.25	-.27
Int1					**.66****	-.15	-.17	.42**	.40*	-.30*	-.01	-.61**	-.34*
Int2						-.24	-.51**	.30*	.40*	-.36*	-.09	-.44**	-.72**
LC1							**.40****	.14	-.07	.25	.33*	.32*	.22
LC2								.06	.09	.36*	.17	.17	.55**
SS1									**.54****	.05	-.06	-.17	-.07
SS2										.01	-.19	-.18	-.29
Activ1											**.53****	.30*	.47**
Activ2												.14	.29
PSE1													**.51****

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 6.6. Legend of abbreviations used for variables in Table 6.5 and 6.7.

Abbreviation (1=Time 1, 2=Time 2)	Variable
PSv	Pain Severity
Dep	Depression
Int	Interference
LC	Life Control
SS	Social Support
Activ	Activity
Pse	Pain self-efficacy

[12] Only the variables that have been consistently recognized as potential mediators are included in the matrix. Punishing, solicitous and distracting responses, for example, are not presented as none of them correlated significantly with any of the key variables.

It should be noted that some of the correlations for the variable, social support (Time 2) are significant only to 0.05 level, due to missing data. There are several points to note about how the Time 2 correlations agree with or differ from Time 1 correlations. All the Confidence Intervals for correlations were investigated to determine if CIs for Time 2 correlations contained the original Time 1 correlations, which would indicate that they were not different. This is not a straightforward investigation because the correlations are not independent, i.e. the sample participants are the same. For example, concentrating on pain and depression respectively, it can be seen that the correlations with pain severity are generally similar at both times, i.e. all four correlations with depression are significant and positive. However, some correlations with pain severity are stronger at Time 2, notable inversely with control ($r=-0.43$, $p< 0.01$; CI [95] = [-0.65, -0.15]), and inversely with pain self-efficacy ($r=-0.71$, $p< 0.01$; CI [95] = [-0.83, -0.52]). Although, with reference to the matrix, it can be seen that the correlation for pain severity and control at Time 1 (-0.27) falls within the confidence intervals for the correlation at Time 2, therefore they are not reliably different. On the other hand, the pain severity-pain self-efficacy correlation at Time 1 (-0.41) does not fall within the confidence intervals for the correlation at Time 2, so it is possible that they are different. With regard to depression, some correlations are stronger at Time 2, while others are weaker. The correlation with control at Time 2 is stronger ($r=-0.65$, $p < 0.01$; CI [95] = [-0.79, -0.44]) and the Time 1 correlation (-0.38) does not fall within the confidence intervals of the correlation for Time 2. The correlations with activity and pain self-efficacy fall to non-significant values at Time 2. When checking the confidence intervals for Time 2 for these variables, [-0.49, 0.07] and [-0.53, 0.03] respectively, it can be seen that the original correlation for activity (-0.52) does not fall within the confidence intervals of the Time 2 correlation, while the original correlation for pain self-efficacy (-0.40) does. All other relevant correlations were checked and it was found that there were no other significant differences. These findings suggest that pain severity was more strongly related to pain self-efficacy and depression was more related to control and less associated with activity at Time 2. As can also be seen from Table 6.5, all measures at Time 1 are significantly

correlated at the 0.01 level, with their respective counterparts at Time 2, which would be expected if they had good test-retest reliability. This is one of the main ways of checking the degree of stability of a measured trait.

6.3.1 Hypothesis Testing

Given that the respondents were assessed a second time, it is important to determine the relationships between key variables, especially as the correlation analyses showed that some of the relationships have changed. The first hypothesis addresses the cross-sectional nature of the Time 2 data, and is based on the previous findings.

Hypothesis 1: At Time 2, chronic pain severity and depression will be directly linked, with some minor mediating contribution from other variables including perceived interference, life control and pain self-efficacy.

The next step in analysing the data is to perform correlations, multiple regressions and path analyses, where relevant. Correlations for the relevant cognitive-behavioural responses for all participants are displayed in Tables 6.7, those that are significant in bold type, with the multiple regression results displayed in Table 6.8 and the path analysis presented in Figure 6.1.

Table 6.7. Matrix of Pearson's product moment correlations among cognitive variables for responders at Time 2 (N=44).

	Dep	Int	LC	SS	Activ	PSE
PSv	**.32***	**.73****	**-.43****	**.37***	**-.41****	**-.72****
Dep		.24	**-.65****	-.15	-.23	-.27
Int			**-.51****	**.40***	-.09	**-.72****
LC				.09	.17	**.55****
SS					-.19	-.29
Activ						.29

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

To determine which variables were mainly predicting depression in the responders at Time 2, a multiple regression was performed with depression as the dependent variable and the significant variables as independent variables. Based on the significant correlations with depression, only pain severity and control were entered into the multiple regression as independent variables.

Table 6.8. Multiple regression analysis: Predictors of depression in responders at Time 2 (N=44).

Predictor Variables	**B**	**Beta**	**CI [95]**	**t**	**R^2**	**AdjR^2**	**F (df)**
Equation (DV: Depression)							
Control	-7.03	-.63	-10.02, 4.04	-4.74***	.42	.39	14.83***
Pain severity	.41	.05	-1.86, 2.69	.37			

Only life control, inversely, depicted depression significantly in this analysis, with a beta of -.63. With life control partialled out, pain severity had a negligible direct effect on depression, beta of .05. Life control therefore accounted for 42% (39% adjusted; $F(2, 41) = 14.83$, $p<0.000$). Given that the sample consisted of 44 participants, there might be concern that the sample size was inadequate or that a Type I error may have been made. However, power analysis showed that the ES of this analysis (f^2) is equal to .72, which is a large ES. In fact, according to Cohen (1992), in a multiple regression with two independent variables, a large effect can be achieved with just 30 cases. The relationships between pain severity, depression and control can be depicted in a path diagram, as shown in Figure 6.1.

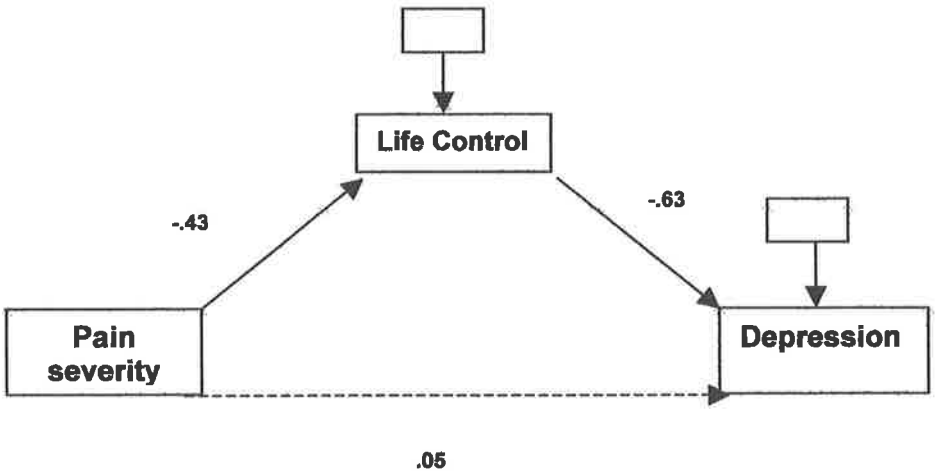

Figure 6.1. Cognitive-behavioural mediation model: Responder chronic back pain participants (N=44).

The path diagram supports the notion that in this group of responders at Time 2, the link between pain severity and depression is mediated by life control (Baron & Kenny, 1986). In the path diagram, it can be seen that the direct path from pain severity to depression is only 0.05, while the indirect path via control equals (-.43)*(-.63): 0.27. So at Time 2, for all participants, depression was mainly predicted by perceived control with only a minor contribution directly from pain severity. The hypothesis is therefore not supported in its postulated form. The effect of pain severity on depression in this case is mainly via its influence in decreasing perceived life control. Therefore, at Time 2, pain severity and depression are mediated by control over pain.

Hypothesis 2: If depression is a consequence of chronic pain, then pain severity (Time 1) should predict depression (Time 2) better than depression (Time 1) predicts pain severity (Time 2).

To test the second hypothesis, the relevant correlations were extracted from Table 6.7, and entered in a cross-lagged panel. This is shown in Figure 6.2.

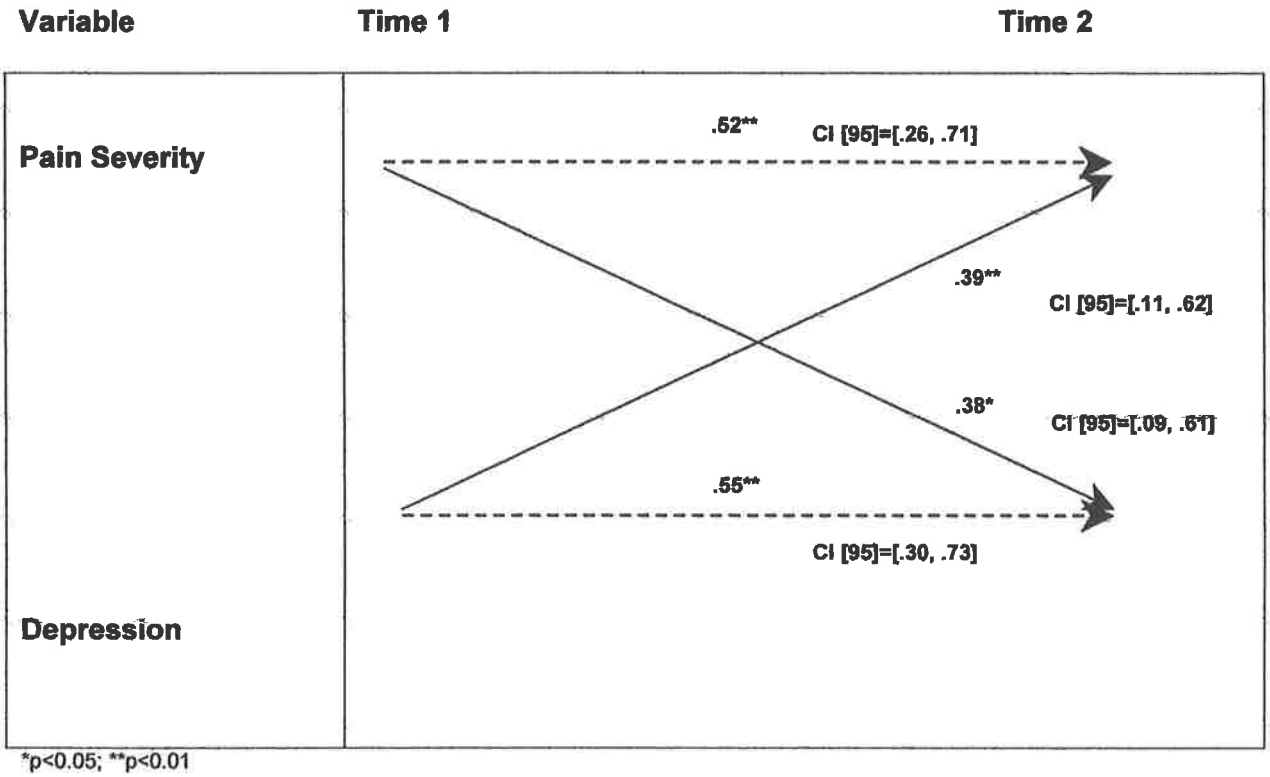

Figure 6.2. Cross-lagged panel for the correlations between pain severity and depression in Study 3 (N=44).

As shown in Figure 6.2, the stability values for pain severity and depression across the 5-year interval are about the same (0.52 and 0.55, respectively). Similarly, the relationships predicting depression (Time 2) from pain severity (Time 1), and predicting pain severity (Time 2) from depression (Time 1) are about the same (0.38 and 0.39, respectively). This was confirmed by checking the Confidence Intervals.

To further investigate this outcome, a series of hierarchical multiple regression analyses were conducted (see Table 1 & 2, Appendix K). The first of these with Depression Time 2 as the dependent variable, controlled for Depression at Time 1 and then Pain severity, Time 1 was entered on the next step, followed by the next associated variable,

Control 1. In Table 1 (Appendix K), it can be seen that Depression at Time 1 does not add significantly to the prediction of Pain severity at Time 2, with Pain severity at Time 1, controlled for. None of the other correlated variables entered into this regression adds significantly to the prediction of Pain severity at Time 2 even when Pain severity at Time 1 is taken account of. Similarly, in Table 2 (Appendix K), it can be seen that Pain Severity at Time 1 does not add significantly to the prediction of Depression at Time 2, once Depression at Time 1 was controlled for. Consistent with the correlations, Control at Time 1 does add significantly to the prediction of Depression at Time 2, even when Depression at Time 1 is controlled for. This is the only variable that contributes to the prediction of Depression at Time 2. There are no significant predictors from the relevant variables for Pain Severity at Time 2.

There is thus no evidence in favour of Hypothesis 2, that is, there is no evidence to suggest that either pain severity or depression has priority as a temporal, causal effect on the other. Instead, it seems likely that the two variables mutually influence each other, only and if at all, through the mediating effects of other cognitive-behavioural variables, as already suggested.

In the final section of this Chapter, gender differences among male and female participants, who took part in the follow-up study, are explored. In general, the male and female sub-samples (n = 16; n = 28, respectively), are not sufficient to allow separate analysis. For example, there are enough female participants to allow for large ES correlations with power of .8 and alpha set at 0.05 but, strictly speaking, the male group is too small. However, one difference between male and female correlations is so large as to be noteworthy. Correlations for males and females are shown in Tables 6.9 and 6.10, respectively.

Table 6.9. Matrix of Pearson's product moment correlations among cognitive variables for male responders at Time 2 (n=16).

	Dep	Int	LC	SS	Activ	PSE
PSv	.62*	.77**	-.25	.35	-.55*	-.74**
Dep		.48	-.40	.07	-.39	-.79**
Int			-.24	.38	-.25	-.54*
LC				.13	.30	.39
SS					.16	-.11
Activ						.11

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 6.10. Matrix of Pearson's product moment correlations among cognitive variables for female responders at Time 2 (n=28).

	Dep	Int	LC	SS	Activ	PSE
PSv	.23	.70**	-.49**	.41	-.33	-.70**
Dep		.17	-.66**	-.12	-.16	-.07
Int			-.62**	.45*	-.01	-.82**
LC				.01	.09	.58**
SS					-.42*	-.44*
Activ						.11

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

For males, the inverse correlation between pain self-efficacy and depression was –0.79 (CI [95] [.64, .88]) at Time 2 ($p<0.01$), whereas for females that correlation value was

only –0.07 (CI [95] [-0.83, -0.51]). From inspection of the relevant CIs, it can be seen that these correlations are reliably different[13]. This was not the case at Time 1 when pain self-efficacy was significantly correlated with pain severity for both males and females, at r= -0.53 and r= -0.57, respectively. This suggests that confidence in dealing with pain is important for alleviating depression in males but not necessarily for females, at least not in the long term. For females only, life control is significantly related to depression, negatively (-0.66, $p<0.01$), but not to pain severity.

6.4 Discussion

There are limitations with this study. Some are the same as for Study 2 and will be discussed more fully in Chapter 7, but there are those that specifically relate to the longitudinal nature of this study. For example, according to the pervading medical opinion, once a person has developed a chronic pain condition, it is generally acknowledged that in most cases the experience is a continuous and often indefinite condition, with times when pain is worse and others where it may not be so bad. Do these times represent discrete episodes of pain or a continuum? The literature lacks consensus on this. It is clearer on the issue of depression and depressive episodes. A person may suffer a level of depression most of the time or be prone to depression and only suffer at specific times or under certain situations. Depression can therefore be a discrete condition that occurs episodically, with each episode unrelated to preceding or later episodes. Therefore, in this research it cannot necessarily be assumed that depression at Time 2 is "a continuation of the former illness [or that there is] an unbroken causal connection [between the two] " (Magni et al., 1994, p. 294). At the same time, it is likely that the person who demonstrates depressive symptomatology on two occasions may be more prone to depression than someone who does not (Magni et al., 1994). In this study, about half of those depressed at Time 1 were still depressed at Time 2, but the others were not. In addition, 50% of those who reported depression at Time 2, had not reported depression at Time 1. As these

[13] A z test confirmed this ($z=2.93$, $p<0.01$).

numbers were quite small, further analyses were not conducted, however, consistent with Magni et al. (1994), those who were depressed on both occasions would be more at risk of further depression bouts than some-one who was not.

The results of this study are discussed briefly here, and more comprehensively within the context of the entire project in Chapter 7. Overall, analysis showed that the responders differed little from the non-responders in terms of demographic characteristics or cognitive-behavioural responses at Time 1. However, there were significant differences in three of the anger measures. The non-responders had originally reported more state anger, more proneness to anger and greater expressed anger, although the effect sizes of the differences in state and expressed anger were small to medium. According to Bezeau and Graves (2001), it is reasonable to aim for 'medium' effect sizes, however, this implies that with such ESs small samples may have low power. With larger ESs, smaller samples may be adequate. In clinical research, a medium effect would allow about a 60% correct diagnostic classification rate which is scarcely better than chance (i.e. 50-50). The ES for trait anger was of medium magnitude as well, so does this significant difference between the responders and non-responders represent a real difference? If so then this is consistent with previous research that has found that those in chronic pain tend to demonstrate a greater proneness to anger (Achterberg-Lawlis; 1982; Beutler et al., 1986), although the evidence is limited. In any event, overall the responders were more like the non-responders than not, and were therefore considered to be representative of the whole group.

Also, the responders appear to have changed little in the 5 years since they first participated in the research in terms of demographic characteristics and most of the cognitive-behavioural responses to their pain. However, they did report greater perceived life control and fewer punishing or solicitous responses from others. Given the overall lack of change, it is reasonable to regard the Study 3 sample as fairly representative of the larger group.

Hypothesis 1 was not supported, as depression was not mainly predicted by the direct effect of pain severity. Even though the follow-up group was small, power analysis

revealed that it was adequate to demonstrate certain significant differences. The path analysis demonstrated that most of the effect on depression from pain severity was indirect, via life control. It appeared from the path diagram that with increased pain severity, life control was reduced and this was related to increased depression. The direct effect, with life control partialled out, was very small (0.05). These results support the idea that chronic pain is dynamic and that the relationship between chronic pain and depression changes over time. For this sample, it appears that after a period of 5 years, the relationship between chronic pain and depression has changed for the 'retained' cohort, despite the fact that the t-tests did not show a significant difference in cognitive-behavioural responses. At Time 1, depression was almost equally affected by the direct, as well as the indirect, effects of pain severity via mediating variables. However, 5 years later at Time 2, depression appeared to be related more to how much life control people perceived they had lost over their lives as a result of chronic pain, with little direct effect from pain severity.

This result is partially consistent with previous research, which had found that control was one of the mediators between chronic pain and depression (Rudy et al., 1988; Turk et al., 1995; Turk & Rudy, 1990). Those who feel more in control of their lives, despite persistent pain, are likely to be more involved with their own care and are more likely to report that their pain is less severe (Philips, 1987a). In keeping with this, the responder group did report at Time 2 that perceived pain severity was less than at Time 1, although this was not a significant difference. In addition, they reported significantly more control on average at Time 2 compared to Time 1. The previous research also reported that interference was a mediator as well, which was not found in Study 3, although it was in Study 2. Perhaps after 5 years, some individuals had adapted to the interference that chronic pain represents by developing strategies to cope more positively with the condition. They reported that pain was less of an interference at Time 2, but again this was not significant. Although interference still appeared to be related to pain severity, this did not translate to an effect on depression in Study 3.

Hypothesis 2 was not supported as the cross-lagged correlations and multiple regression analyses could not support the notion that pain severity causally precedes depression, but rather control was the only predictor of depression. Instead, the findings are consistent with conclusions by Magni et al. (1994) that "depression promotes pain and pain promotes depression" (p. 289). They argued that one relationship may be relevant for one group of people with chronic pain, while the other may fit equally as well for a different group. It is possible that the time interval to follow-up in Study 3 was too long, as it has been shown in previous research that the chance of developing depression may be higher in the early months and years post-onset of the chronic pain condition (Love, 1987). Brown (1990) reported that, even with waves of data collection in a longitudinal study, support for the hypothesis that "depression results from pain [.....is only] tentative" (p.132). Brown's (1990) 3-year study found that the prediction of depression by pain severity improved with time. That conclusion is not supported by the present findings. Hurwitz, Morgenstern and Yu (2003) also found that 'psychological distress', a composite measure, including depression and anxiety, and pain could both "be causes and consequences of each other" (p. 463), although the associations were small, and that longitudinal relationships were not as strong as cross-sectional associations. These findings highlight the difficulties of conducting longitudinal research and the fact that there are likely to be many different variables that confound any relationship.

Gender differences were examined but unfortunately due to the small sizes of the male and female sub-samples, only limited analysis could be performed. However, some useful information was noted. Pain self-efficacy was related to both chronic pain severity and depression for males at Time 2 ($r=-.74$ and $-.79$, respectively, both $p<0.01$). This is consistent with previous research (Arnstein et al., 1999). Men who are confident that they can deal with daily life, despite their pain, are more likely to cope with their pain and are less likely to feel depressed. They are less likely to be affected by poor performance when trying to exercise, which in others could result in loss of confidence. For example, a man with high self-efficacy will interpret pain experienced while exercising as a normal part of the healing

process, whereas someone with low self-efficacy may interpret this as personal inadequacy or failure (Bandura, 1977).

For females at Time 2, life control played a key role as the mediator of chronic pain and depression and it was the only significant correlate of depression (r= -.66). This is consistent with the previous work by Turk and Rudy and others (Maxwell et al., 1998; Rudy et al., 1988; Turk et al., 1995; Turk & Rudy, 1990) who found that control was a key mediator between pain and depression. Those with a greater sense of control are more likely to be actively involved in their treatment and less likely to engage in avoidance behaviour (Philips, 1987). This is crucial in terms of taking opportunities to test one's physical and mental health and being encouraged by successes, rather than being deterred by failures.

These results will be discussed further within the context of the entire 5-year research project and presented in Chapter 7.

CHAPTER SEVEN

GENERAL DISCUSSION

7.1 Introduction

This chapter commences with a discussion of the rationale for the present research which has sought to address one of the most puzzling aspects of chronic pain: why do some people adjust to chronic pain and live relatively normal lives while others do not adjust and become depressed? The empirical work presented in this thesis has addressed this question by exploring several key objectives, and the major findings associated with these are presented and discussed in this chapter. Methodological issues and limitations of the research are subsequently considered. This is followed by a discussion of implications of the research with respect to cognitive-behavioural theory as well as for community education and future research. Finally, the chapter briefly concludes with an evaluation of the contribution that this research has made to the chronic pain knowledge base.

7.2 Rationale for the research

In order to explain the rationale for this research, several key points about the state of knowledge about chronic pain as detailed in the literature review are reviewed. Chronic pain is recognized as one of the most debilitating and prevalent medical conditions within the community, while being one of the least understood (Elliott et al., 1999). When it occurs with depression, it has the potential to become a severely disabling and a potentially deadly condition (Gallagher & Verma, 1999). It is therefore important to understand the influence of depression within the context of pain. It appears, however, that most knowledge about the condition is based on a small percentage of severely psychologically impaired people, often male, referred to medical pain centres or clinics (Crombie & Davies, 1998; Ruda, 1993). Despite a considerable literature base, the intricacies of the psychological effects of chronic pain in the wider community, especially among women, are far from clear.

The chronic pain literature has been criticized for lack of theoretically based research and methodological rigour (Bonica, 1979; Fishbain et al., 1997; Gamsa, 1994a; Sullivan et al., 1992). Mostly, those suffering chronic pain subscribe to a traditional medical model that promotes the idea that degree of pain perception reflects degree of tissue damage (Philips, 1989). Moreover, in a majority of chronic cases, medical tests have failed to reveal or identify damage. Pain is mainly a subjective experience and there is currently no well accepted technological method of objectifying the severity of chronic pain (Von Korff et al., 1992). In addition, there is still considerable debate about why some people adjust to their condition when others fail to adjust and develop depression (Spence, 1993).

There have been many theories promoted but cognitive-behavioural theory has the strongest evidence base, in terms of explaining the relationship between chronic pain and depression. Moreover, some say it has endorsed a 'reciprocal deterministic' approach (Novy et al., 1995). Although there have been considerable, recent theoretical advances in terms of cognitive-behavioural theories, the literature has not necessarily provided clear ways of testing such theories. A unique and innovative study by Rudy et al. (1988) claimed to be ".....the first direct empirical demonstration that psychological mediators may be involved in depressive symptomatology among chronic pain patients" (p. 136), and that depression is a consequence of chronic pain. Subsequently, several other groups of researchers have supported this approach to the study of chronic pain and depression (Devins et al., 1993; Haythornthwaite et al., 1991; Kleinke, 1991; Maxwell et al., 1998; Turk et al., 1995). In addition, recent research by Hurwitz et al. (2003), investigating psychological distress and pain, report the efficacy of a cognitive-behavioural mediation approach. Other research has shown the aptness of applying cognitive mediation models to a range of psychological issues in other areas of interest, besides chronic pain (Chang et al., 2003; Papworth & James, 2003).

The basis for the studies reported here was essentially a partial replication of the Rudy et al. (1988) model. There were several important differences as follows. Firstly, as indicated, the present sample was non-clinical because most conclusions about pain and

depression are based on clinical patients who do not represent the majority of people in the wider community and therefore, little is known about the latter (Novy et al., 1995). Secondly, measures used in this research differed somewhat to those used in the Rudy study. Some, such as the MPI, were the same while others, such as the CES-D, were not. The MPI was used as the standard chronic pain assessment tool, because it was uniquely "designed to assess chronic pain from a cognitive-behavioural perspective" (Keefe et al., 1992, p. 531) and therefore, was fundamental to the project. Instead of using the BDI to assess depression, the CES-D was used because it was specifically designed for community survey research (Plutchik & Conte, 1989), to assess depressed mood in non-clinical groups, rather than as a clinical diagnosis tool like the BDI. It is also "the most generally useful self-report test" (Turk & Okifuji, 1994, p. 11) compared to other tests including the BDI, the Zung (Zung, 1965) and the MMPI Depression Scale (Plutchik & Conte, 1989). Furthermore, the CES-D has long been used to assess depressed mood in chronic pain patients (Fishbain et al., 1997) and its validity is not seriously threatened by physical impairment, typical of chronic pain (Berkman et al., 1986). Also, as there were other variables of interest besides those used in the Rudy study, the most valid and reliable instruments were chosen to measure these, e.g. the STAXI for anger and the PSEQ for pain self-efficacy.

Thirdly, the structural equation modelling techniques used in the Rudy study, were found to be inappropriate for testing a cognitive-behavioural mediation model of chronic pain and depression in this research, for two reasons. One was the size of the available sample. Rudy et al. (1988) used SEM with 100 participants but since then, critics have warned against misusing SEM because for meaningful results, at least 200 participants are needed (Hoyle, 1995; Loehlin, 1998; Mueller, 1996) and it is not suitable for all data (Pedhazur & Schmelkin, 1991; pers. com. Willson, 1999). The second reason was that other studies about mediation based on Rudy's research have used correlations, regressions and/or path analytic techniques to legitimately support a cognitive-behavioural perspective (Maxwell et al., 1998; Turk et al., 1995). Given the difficulties in acquiring participants for the present

thesis and that the final group numbered just 105 persons, path analysis rather than SEM was therefore considered more appropriate.

A fourth difference between the Rudy et al. (1988) research and this thesis was that the former relied on cross-sectional analysis and did not address gender differences. In the present research, cross-sectional data is augmented by a longitudinal analysis and gender differences have been investigated. In terms of variables of interest, the pain literature suggested that there were several other cognitive-behavioural factors worthy of investigation as correlates of chronic pain and depression, besides those used by Rudy et al. (1988). The present research was undertaken to determine the merit of such speculation.

Finally, it must be remembered that the cognitive-behavioural mediation model testing was only part of a more comprehensive study into characteristics of people with chronic pain from the community.

The main issues addressed by the empirical work were to:

- determine the general characteristics of non-clinical chronic back pain participants in terms of demographic and pain-related characteristics and cognitive-behavioural responses to pain;
- determine the relevance of 'profiling' to people with chronic back pain;
- compare chronic back pain participants to non-pain participants on several specific cognitive measures to determine the extent to which pain affects cognitive functioning;
- compare sub-groups within the sample of people with chronic back pain, i.e. specifically compare those with depression to those without depression on demographic and cognitive variables;
- consider the role of cognitive appraisal in the chronic pain-depression relationship by testing cognitive-behavioural mediation models, especially gender-specific models;
- investigate the longitudinal relationship between chronic pain and depression to determine whether a cognitive-behavioural mediation model is stable over time;

- discuss the implications of this research for cognitive-behavioural theory;
- examine the implications of this research in terms of community education including the possibility of raising community awareness about the dangers of long-term pain conditions to mental and physical health, social relationships and subsequent economic costs to society.

7.3 Major findings

Major findings related to those objectives will be discussed in the following sections.

7.3.1 The general characteristics of chronic back pain participants

With regards to the demographic characteristics, participants were generally a restricted group in terms of nationality and occupation. Most were Australian or British born and there was little representation of other nationalities. The majority of participants felt that their pain was made worse by bending and lifting and this had been elaborated at many of the interviews to mean that it was the type of work they did, rather than a single incident. That is, 'bending' and 'lifting' were cited as being integral to their occupations and thought to be largely responsible for their ongoing problems. This is consistent with the fact that most occupations were of a manual type, including nursing, which involves considerable physical effort. There were few who worked in white-collar type professions. Most no longer worked full-time and the majority of those who did reported that their work was restricted by their pain. Even with conservative estimates, most of them knew the origin of their pain condition which is not consistent with the literature report that most people with chronic back pain have no clear idea how their pain began (Birbaumer et al., 1995).

Pain relief was mainly provided by resting, medication, massages, hot showers, stretching and walking. The health professionals who were reported to provide most pain relief were physiotherapists, chiropractors, general practitioners and acupuncturists, in that order. The sample was also found to be divided into those who were taking mostly prescription medication for pain (n=56) versus those who were not (n=49). They were not

different in terms of demographic characteristics, except that the script-takers had significantly more back operations. This is an interesting point – if back surgery is promoted as a key method of dealing with back pain, why were those who had had more back surgery taking stronger medication? This finding could add weight to the argument that medical interventions do not consistently relieve chronic pain. In keeping with this, those who took more prescription medication were also more inclined to visit their general practitioners, while the rest appeared to prefer less orthodox health care workers, such as chiropractors.

Less than half the participants reported that exercise provided pain relief. This is a concern when it is widely promoted in the media by health experts that exercise is integral to the maintenance of good health. This could imply that many people with chronic pain do not see this as a major option. Is this because they perceive any pain produced by exercise as a punishment or negative reinforcement and so fail to test their abilities or is it that they are not exposed to enough information about how to cope with back pain? This suggests that more education to the public about how to manage unremitting back pain may be useful.

Were the participants in Studies 1 and 2 representative of non-clinical or community groups? The weight of evidence suggested they were. That is, it could not be concluded that they were any more likely to visit a health professional or take more pain medication than the general public, they appeared to be less dysfunctional than clinical samples and no more dysfunctional than non-pain samples and they were far less likely to fit a chronic pain 'profile' than clinical patients.

It has been shown that they responded significantly differently to clinical patients on several key responses. Furthermore, their responses were all in the expected directions, if it is assumed that they are less psychologically affected by chronic pain, as the literature suggests (Crombie & Davies, 1998; Elliott et al., 1999; Jensen et al., 1991; Linton, 1994; Ruda, 1993). However, the normative data available with which to compare their responses was quite limited so while these results are interesting, it is not possible to make any firmer conclusion except to say that they appeared to be less psychologically disturbed than reported clinical pain samples and more depressed than a community non-pain sample.

Perhaps more importantly, they did demonstrate significant depression, which supports the idea that chronic pain has severe psychological consequences, which may be 'hidden' in the community.

The bias toward female participants in the present group was not unusual. Croft et al. (1993) found that more females were not only registered in selected general practice areas, but also that women were more likely to complete questionnaires for research purposes. However, this is inconsistent with most previous chronic pain research, which has been conducted on small samples of males (Ruda, 1993). That more women volunteered to take part, as well as complete the questionnaires, is consistent with the notion that women are more aware of health issues and have a greater "willingness to admit health problems" (Verbrugge, 1980, p. 327). Women are also reported to use health care facilities more than men (Bush et al., 1993). Consistent with this, although not conclusive, significantly more males than females failed to complete the questionnaire and/or withdrew from the research in Study 2. This carried through to the follow-up study where the gender ratio was similar to the other studies.

Are women more willing to seek health care early when in pain, compared to males? If so, this may partly explain the seeming over-representation of males in clinical research. Do males leave their physical and mental discomfort so long that when they do seek help, they find themselves having to seek specialist care? And does this reflect the social tendency to care more about paid work by men and their capacity to continue in paid employment? Although there has been limited research into gender differences in terms of developing depression when in pain, those who have investigated this issue have reported that females are more at risk (Turk & Okifuji, 1999; White & Harth, 1999). Even though females were more represented in this research, it could not be shown that they were more depressed than the male participants. Furthermore, this result is consistent with work by Haythornthwaite et al. (1991) who reported no gender, educational or marital status differences, between depressed and non-depressed clinical patients.

The present studies could not demonstrate that there were any significant differences in terms of other demographic characteristics that were assessed, i.e. marital status, education or employment status. Although White and Harth (1999) found that single females were more at risk from chronic pain in terms of depression, that result was not replicated here. It might be expected that the single person would have less support than the married person in pain and therefore be more likely to suffer psychologically. Turk et al. (1992a) have shown that it is the type of support, i.e. negative or positive which is more important than social support, as such. This suggests that if individuals perceive that they have adequate, positive support, they should be less prone to depression. However, in the present research, the depressed group indicated they received more negative support, in the form of punishing responses. Perhaps the single participants received more support from their wider social networks, while the married participants relied more on spouses, giving a similar outcome. Kraaimaat et al. (1995) reported that chronic pain had a debilitating effect on spousal relationships while significant others may inadvertently confirm the negative perception that people with chronic pain have about their prognosis, impeding adjustment (Schiaffino & Revenson, 1995). Both those scenarios may explain why the presence of significant others may not necessarily indicate that support is positive. These results indicate that the social support issue is clearly complicated and further research is warranted.

7.3.2 Chronic pain profiles

The present research also presented a rare opportunity to evaluate a groups of people with chronic back pain from the wider community, in terms of 'profile'. There is little published research about profiles of community groups of people with chronic pain, to date (Gatchel, Noe, Pulliam, Robbins, Deschner, Gajraj et al., 2002). The profile classification system, developed by Kerns et al. (1985), identified three chronic pain subgroups, dysfunctional, interpersonally distressed and adaptive copers. A substantial body of clinical research by Turk, colleagues and others (Rudy et al., 1989; Talo, 1992; Turk et al., 1995;

Turk & Rudy, 1986, 1987a; Turk & Rudy, 1988; Turk & Rudy, 1990) has also shown that the system is useful in terms of devising relevant treatment protocols for chronic pain patients. This classification system is clearly useful with pain clinic patients because it identifies those most at risk. In the present research, a high proportion of the participants could not be classified into any of the three groups. It was therefore not necessarily informative when used with this community chronic pain sample.

Only about 24% of the present sample could be classed as 'adaptive copers', the group considered to be least at risk and most likely to cope with the ongoing stress of chronic pain. Of even more concern is the fact that about 40% were classed as 'hybrid', 'anomalous' or 'unanalysable'. These categories have also been defined by Rudy (1989), but do not appear to account for any participants in clinical studies. The number of anomalous was especially alarming. An anomalous profile represents MPI scores that "make no sense according to established [cognitive-behavioural] theory" (Rudy et al., 1989, p. 40) and could be due to random responses, reading problems and other unknown difficulties. Does this mean that some participants truly misunderstood the questions, were ambivalent about their answers or is it further evidence that non-clinical groups are different to clinical patients and cannot be categorized in the same way? It could also represent further support for the idea that all people with chronic pain do not belong to some homogenous group and they do not fit the 'uniformity' myth (Gamsa, 1994b).

The 'unanalysable' category refers to participants who did not fill in all the responses. This appears to relate mainly to those without a 'significant other' in their lives so people who were single were unable to complete these responses. This challenges the assumption that such people can be assessed in terms of risk, which appears to be a clear limitation of the MPI. Since this research was conducted, other researchers have also reported that the MPI can be problematic with respect to classifying patients into subgroups (Bernstein, Jaremko, & Hinkley, 1995; Okifuji, Turk, & Eveleigh, 1999b; Riley, Zawacki, Robinson, & Geisser, 1999). Allowance was not made for this in the MPI used in the present research and the

result is that many participants who did not complete the 'significant other' section, may have been classified as 'unanalysable' due to missing data.

There is an increasing trend toward 'living alone' in contemporary society, which means the 'significant other' may no longer be someone who cohabits with a person who has chronic pain, and yet will still provide valuable social support. These results indicate that the questionnaire may need updating to allow for changing social trends. Although some have reported that changing the instrument would be premature (Riley et al., 1999), Okifuji et al. (1999b) have since addressed the issue and report that a modified MPI has improved classification, which augurs well for future research involving the MPI.

7.3.3 *Cognitive functioning in the chronic back pain participants compared to non-pain controls*

The non-pain control group were matched on age, gender and marital status to the people with chronic pain. They had significantly more formal education than those with pain but did not differ on any other demographic characteristics. They were significantly less depressed than those with pain but did not differ significantly on any other relevant cognitive-behaviour variable. This result regarding depression validates the suggestion of the higher cut-off point of 19 for the CES-D by Turk and Okifuji (1994). This also tends to support the premise that depression is more common in those with chronic back pain than in the non-pain public (Fishbain et al., 1997; Sullivan et al., 1992), at least with this group.

It was expected that controls would report significantly greater internal health locus of control than the pain participants but this was not found. It has been reported that people with a high internal health locus of control generally have a greater sense of responsibility for their own health, tend to engage in more self-help type behaviours (Wallston & Wallston, 1978) and cope better with pain (Crisson & Keefe, 1988; Skevington, 1983). However, an internal orientation to health may not be sufficient for people to engage in healthy behaviour (Wallston, 1992). The health locus of control measure tends to focus on how much health is

valued and does not necessarily take into account the expectations people have about outcomes of certain behaviours.

Health locus of control may only account for a small proportion of the variance in health behaviours. Perceived control over health takes this aspect into account, i.e. it is not just how much health is valued but also what people *perceive* or believe will be the outcome of certain behaviours, in terms of their health (Wallston, 1992). The present finding supports this idea but also highlights that health locus of control probably needs further investigation, in both chronic pain and non-pain groups.

This research also offered an opportunity to evaluate general self-efficacy. There has been little published work on the general self-efficacy construct, except with students and alcoholics (Lennings, 1994; Sherer & Adams, 1983; Sherer et al., 1982; Tipton & Worthington, 1984; Woodruff & Cashman, 1993), so there is little data for comparison. It was predicted that the non-pain control group would report significantly more general self-efficacy but this was not the case, although scores were in the expected direction. General self-efficacy was implicated in the relationship between pain and depression for males with chronic pain only. This suggests that as there were more females in the total group, the influence of general self-efficacy for the whole group was weakened. The construct of general self-efficacy is considered to be more of a global construct and should influence specific self-efficacy such as pain self-efficacy according to Shelton (1990). In the present research, it was significantly correlated to pain self-efficacy, although the correlation was low. These results suggest that more research into the construct of general self-efficacy is warranted, particularly as it relates to chronic pain and depression.

The finding that trait anger was not significantly greater in the pain group is not consistent with previous research by Beutler et al. (1986), who speculated that people with chronic pain should be more prone to anger. This view is based in a psychodynamic approach to the study of pain and the idea that some people have personalities more susceptible to pain and its attendant psychological dysfunction. This idea has found little support across a number of studies (Dworkin et al., 1992; Gamsa & Vikis-Freibergs, 1991;

Romano & Turner, 1985; Turk & Salovey, 1984; Von Korff et al., 1993). While Achterberg-Lawlis (1982) demonstrated that anger appeared to be a dominant personality characteristic among people with rheumatoid arthritis, there is limited evidence from research with other pain groups. Recent work by Burns et al. (1998) has shown that anger management style may be more important than proneness to anger in the person with chronic pain. Again this finding may highlight a difference between clinical patients and people from the community with chronic pain and that evidence based on one group does not meaningfully generalize to another.

7.3.4 *Depression versus non-depression*

Overall, 34% of participants reported depressive symptomatology. This finding is cause for concern (Turk & Okifuji, 1994; Turk et al., 1995), as it is much higher than the 16% reported in other community research (Magni et al., 1993). Research in the 1980s and early 1990s reported that depression among people with chronic pain varied between 8% and 87% (Fishbain et al., 1986; Haythornthwaite et al., 1991; Kramlinger et al., 1983; Large, 1986). One could argue that such a wide range is almost inclusive. In addition, most of these studies used traditional, medical and/or psychiatric based assessments such as the RDC (Spitzer et al., 1978) and the DSM (APA, 1994) unlike the Magni et al. work, which also used the CES-D. More recently, this estimate has been refined i.e. 30 to 54% according to Banks and Kerns (1996), so the present finding is more consistent with, as well as adding support, to that research.

In the present research, the cut-off point of ≥19 on the CES-D was chosen because the intent was to 'flag' those who were more at risk from depression, rather than making a formal clinical diagnosis (Turk & Okifuji, 1994). Much of the previous research has used a cut-off point of 16 (Magni et al., 1994; Murrell et al., 1983). Even accounting for possible inflation due to the self-selection of participants, the present finding suggests that depression among people with chronic back pain in the community may be high and if so, this warrants further investigation. It should also be noted that in the Magni study chronic pain was

defined as that persisting for more than 1 month, which is not consistent with the more orthodox guideline of 6 months (American Psychiatric Association (APA, 1994). This makes the statistic of the present study of even more concern, given that the definition for chronic pain used here was 6 months.

Contrary to expectations, those reporting depression did not differ significantly from those who did not in terms of demographic characteristics, except that they were significantly younger. This is not consistent with previous work by White and Harth (1999) who found that people with chronic pain who were depressed were more likely to be older. However, Averill et al. (1996) have also reported that young female pain clinic patients were more depressed than older female patients. This is consistent with the notion proposed by Haythornthwaite et al. (1991) that those who develop chronic pain early in life are more likely to suffer depression than those who develop chronic pain at a later age. However, the depressed and non-depressed in the present study did not differ significantly on the time they had been in pain so there was no suggestion that their pain had begun earlier in life than the non-depressed participants. There were no other significant demographic differences, in terms of gender, marital status, education and employment status.

The groups did differ significantly on relevant cognitive-behavioural variables, as predicted. Those who were depressed reported more pain, interference and state anger and less perceived control, internal health locus of control, pain self-efficacy and general self-efficacy. These findings are consistent with the previous research, which has been detailed. The association between pain severity and interference and depression is well documented. Similarly, having less control and confidence about dealing with life in general, despite suffering persistent pain is acknowledged. These results also offer further support for the idea that interactions between chronic pain and depression are complicated and interactive.

According to Roy (1986) many researchers fail to report duration of pain. Duration on its own is a poor indicator of response to pain. There has been limited research into how duration of pain relates to the degree of suffering or how people cope with protracted pain over decades. The research that has been conducted tends to be contradictory. For

example, longer duration of pain and illness have been found to be associated with increased risk of depression and disability (Averill et al., 1996; Sullivan et al., 1992) and the elderly unwell are more likely to be depressed than younger people (Parmelee, Katz, & Lawton, 1991; Turner & Noh, 1988). This may be likely if the elderly person in pain has fewer social support contacts (Turner, Noh, & Levin, 1985). Other research has found the opposite, for example, that older persons in pain experience less severe pain (Herr et al., 1993) and fewer psychological effects (Riley et al., 2000).

Despite the limited previous research, it was expected that the longer a person had suffered pain, the more likely it would be that they suffered depression. This was not found for either gender or for the depressed group. This suggests a certain level of adaptation to the condition so that some people accept their situation and adjust their lives accordingly. Given that the depressed group also reported that their pain severity was significantly greater, it appears that it is not the length of time that someone has experienced pain that is contributing to depression but the degree of pain that is experienced and how it impacts on relevant cognitions and gender roles. This is consistent with the major findings.

The present research also provided an opportunity to investigate ancient pain or pain of more than 25 years duration. Previous research has shown that such pain is related to greater depression and greater drug dependency (Swanson et al., 1986). In the present sample, there were no significant differences in drug use or cognitive-behavioural responses except that the short-term group reported significantly more trait anger than did the long-term group. In addition, and conversely, the long-term group reported somewhat less depression than the other group, although it was not statistically significant. This is consistent with previous work by Turk et al. (1995) who found that although there was not a significant difference between younger and older people with chronic pain with respect to depression, the young scored more depressive symptomatology than did the old. These results are more supportive of the suggestion that the likelihood of developing depression may be greatest in the early years post-onset of the chronic pain condition (Love, 1987). While the prevalence of physical disability, including chronic pain, increases with age (Wood

& Turner, 1985), older people may also be more tolerant of increased disability and suffer less psychologically. Further research into this issue is warranted given that chronic pain is so common among elderly people, in particular.

7.3.5 The role of cognitive appraisal in the chronic pain-depression relationship

In Study 1, the positive but non-significant correlation between pain severity and depression was of concern. Was the lack of significance purely due to the small size of the group? Power analysis showed that this was not a problem as the effect sizes were large. Given that pain severity and depression were each significantly related to most of the other cognitive-behavioural variables, it seemed fair to speculate about the possibility that they were being mediated by these other variables. Analysis did support the notion of testing mediation with a larger group.

The relationship between pain severity and depression was significant in Study 2 with depression being mainly predicted by pain severity. This was consistent with the other research that has demonstrated that depression is most commonly associated with chronic pain (Fishbain et al., 1997; Jensen et al., 1991; Pearce & McDonald, 1998; Romano et al., 1988; Turner, J., 1982). It was not consistent with the result from Study 1, however, when the Confidence Intervals for the respective correlations were checked it could be concluded that they might have been from the same population, but not with a high degree of confidence. If the Study 1 sample had been larger, the Confidence Intervals would have been even narrower, with even less chance that they contained the Study 2 correlation. These results also add weight to the argument that research findings are plagued with "...considerable inconsistency and controversy" (Sullivan et al., 1992, p. 5) and that participants are generally quite heterogenous in terms of pain conditions and ages. It is further confirmation that it may not be helpful to treat people with chronic pain as an homogenous group or expect that individuals will conform to some 'typical' pain type, "....with predictable personal history and personality characteristics" (Gamsa, 1994b, p. 22).

For Study 2 participants, the analyses supported the claim that the pain severity and depression relationship is complicated. The original significant correlation between pain severity and depression was reduced when further analysis was performed to examine the relationship. According to Baron and Kenny (1986), this suggested that there were mediators operating. While most of the impact on depression appeared to be directly from pain severity, a percentage was experienced via the mediating variables, although, it is clear from the error terms that there were other unknown variables or "noise" contributing to the variance in depression, although it is not possible to be more specific about this.

(i) Gender differences in the relationship between chronic pain and depression

Multiple regression analyses was useful for demonstrating the predictors of depression for all participants and male and females with chronic pain, specifically. However, the more advanced analyses using path analysis techniques gave a clearer picture of the complexity of the chronic pain-depression relationship and gender differences in the response to pain. While it was feared that the groups would be too small for analysis, again, power analysis showed this not to be the case.

While the path model for the total sample and also for female participants showed that pain severity was more directly than indirectly related to depression, the model for the male participants showed a different picture. As predicted for males, pain severity and depression were indirectly related and mostly mediated by the perceived interference of pain and general self-efficacy. This is consistent with previous research that suggests that depression is likely when usually reinforcing activities are disrupted (Fordyce, 1976; Lewinsohn, 1974). As Haley et al. (1985) also found, males may be more affected by the disruptive nature of pain that prevents them from receiving the rewards of reinforcing activities. In terms of general self-efficacy, this finding is important given that there has been no previous published research detailing such a result. It also highlights the impact that chronic pain can have on all aspects of daily living.

This is in keeping with identity research conducted by Girard (1993) who argued that males are more likely to be psychologically affected by chronic illness than are females, because males are more likely to be judged on 'work' performance while women, if homemakers, are not. If a female with chronic pain is unable to carry out her normal household duties, this is less likely to cause problems than the chronically ill male who cannot work. Such a male is likely to feel that his identity is threatened because his 'bread-winning' capacity is eroded and to suffer accordingly.

Depression in females may be more related to pain severity compared to males, because both pain and depression represent suffering and females may be more susceptible to the overall suffering of chronic pain (Haley et al., 1985). In most Western cultures, it is socially acceptable for females to express suffering overtly, whereas males are more likely to have been socially conditioned to hide suffering or deny it (Lamberg, 1998).

However, beyond that, these results show that gender differences need to be taken into account when trying to better understand the chronic pain experience because it appears to be much more complex in females than in males. Previous studies have mainly concentrated on males (Ruda, 1993) or have not addressed gender differences in the expression of depression (Novy et al., 1996), so this would appear to be a valuable finding. This finding suggests that the female with chronic pain may need different considerations in terms of pain treatment. Again, this information is likely to be useful to those health professionals working in the community with persons with chronic back pain.

7.3.6 Longitudinal evaluation of chronic back pain

Chronic pain research has been widely criticized for excessive dependence on cross-sectional research, so a 5-year follow-up study was conducted to complete the present research. Based on previous research it was expected that a causal relationship between chronic pain and depression might be demonstrated. While most research has been cross-sectional there is none-the-less considerable support in the literature for the hypothesis that depression is more likely to result from chronic pain than the alternative (Breslau et al.,

1994; Brown, 1990; Holroyd et al., 1993; Kubinski et al., 1991; Magni et al., 1994; Rains & Lohr, 1993). Theoretically, if pain severity leads to depression rather than the reverse, then pain severity at time 1 should predict depression at time 2 better than the alternative. Forty-four of the original 105 participants of Study 2 agreed to take part in the 5 year follow-up study.

The responders were compared to the non-responders on demographic characteristics reported at the time of the second study. As there was found to be no significant demographic differences, they were considered to be generally representative of the whole group. There were some differences in anger between the responders and non-responders, although the statistical differences reported were of low magnitude. Overall, the responders could be said to be more like the non-responders than the alternative. Previous research has indicated that under these circumstances, it is reasonable to regard the follow-up sample as generally representative of the entire original sample (Cicchetti & Nelson, 1994). The responders completed fewer questionnaires than they were required to do in Study 2. This was intentional as it was felt that it was important to assess the constructs of importance without overloading participants, as there had been considerable criticism in Study 2 from participants that the entire questionnaire was too long. For this reason, they were asked to complete only the PIQ, the MPI, the CES-D and the pain self-efficacy inventory.

Analyses failed to provide support for the hypothesis that pain severity precedes depression. This may be explained by referring to a similar finding by Magni et al. (1994) in a 10-year follow-up study with 2,324 community participants. That study concluded that "depression promotes pain and pain promotes depression" (p. 289), although the latter was marginally more powerful than the former. This implies that the two predictions are not necessarily mutually exclusive but might equally apply to different pain groups. Magni et al. (1994) suggested that the follow-up of 10 years was too long, noting that the perception of depression at both times does not necessarily infer "an unbroken causal connection" (p. 294).

Hurwitz et al. (2003) also conducted longitudinal research with 681 chronic back pain patients, who were assessed at 6 weeks, 6 months, 12 and 18 months. They concluded that psychological distress and pain could both "be causes and consequences of each other" (p. 463). Furthermore, the associations were small and longitudinal relationships were weaker than cross-sectional relationships. These studies provide evidence of longitudinal findings with a variety of time-spans, from short to lengthy, none of which appeared to be particularly conclusive.

In the present research, the follow-up study occurred at 5 years but as with both these previous works, it cannot be assumed that depression measured at both times is causally related. Love (1987) maintains that depression is a more likely consequence in the early years post-onset of the chronic pain condition, rather than in the later years. It is reasonable to speculate that there were probably many other factors likely to confound a relationship between chronic pain and depression during that time. This finding is further confirmation that pain groups are heterogenous, especially in terms of maintaining factors. Also, this finding adds weight to the argument that generalizing findings about chronic pain from clinical samples to non-clinical groups is problematic. This also has implications for education of the general public by health professionals about the possible consequences of poorly managed chronic back pain. In addition, the findings are further indication that conducting longitudinal research is difficult and needs to be well controlled. How this might be better achieved with non-clinical samples is unclear.

Unfortunately, due to small sub-samples sizes, extensive analysis of gender differences was not possible in the longitudinal study. Suffice to say, that correlations suggested that for males with chronic pain, confidence in dealing with pain specifically was important in the short and the long term. This was not the case for females with chronic pain. Rather, in the long term, females were much more affected by feelings of control over pain. These findings are discussed in more detail below in the context of implications for community education.

7.4 Summary of major findings

The main aim of this thesis was to explore the relationship between chronic pain and depression in a sample of people from the community with chronic back pain. It has been well documented in the chronic pain literature that depression is a major consequence of chronic pain, but some theorists argue that the relationship is indirect and that chronic pain and depression are mediated by cognitive-behavioural variables. However, most of the research has been based on studies conducted with people who attend pain or hospital clinics for treatment. The key questions guiding this research were 1) are chronic pain and depression associated and if so, is this relationship direct or indirect, and 2) does chronic pain precede depression or vice versa?

The first question was initially investigated in Chapter 4 and according to the evidence, for the total sample, although chronic pain and depression were not correlated, path analysis showed that pain severity, interference and pain self-efficacy were together affecting depression, implying that there were direct and indirect effects, although in this case, these were small. This confirmed that pain severity and depression were associated but also indirectly related by cognitive-behavioural variables.

In the larger sample, it was found that while pain severity directly accounted for most of the variance in depression, there were also indirect effects of pain severity via interference, pain self-efficacy and state anger. More specifically, gender differences were demonstrated. For males, there was little direct effect of pain severity but considerable indirect effect, mostly via perceived interference and general self-efficacy. This suggests that it is not only how much pain interferes in the life of a male with chronic pain, but also the effect that such pain has on the confidence to deal with life, generally.

In contrast, the model for females with chronic pain demonstrated a much more complicated view of the chronic pain experience. Most of the variance in depression for the female appeared to be accounted for by pain severity with a smaller percentage accounted for by a combination of cognitive-behavioural factors. For women then it seems that the primary predictor of depression is the pain severity itself, with some mediating effects from

control and suppressed anger. These results support the view that the relationship between chronic pain and depression is multi-dimensional and interrelated. Depression appears to be associated both directly and indirectly to the severity of chronic pain, depending upon gender.

The second question was tested in a longitudinal study and it was found that pain severity predicted depression slightly less well than the alternative and that depression was actually better predicted by control. This finding supported the idea of cognitive-behavioural mediation between pain severity and depression. It also supports previous research by Magni et al. (1994) and Hurwitz et al. (2003) that the scenarios, chronic pain leads to depression and depression leads to chronic pain, are not mutually exclusive and that they may both represent the reality for different groups of people with chronic pain.

This research has indicated that a community sample does not readily compare to the clinically based research, which confirms the premise that clinical samples are not necessarily representative of people with chronic pain from the wider community. This research has also demonstrated the importance of considering gender differences in the response to unremitting pain. It has also demonstrated the value of longitudinal research and that generalizing from cross-sectional research can be problematic.

7.5 Methodological issues

Data analysis for this research depended on the responses to questionnaires. While self-report has been criticized, all of the questionnaires used in this research have been shown to be reliable and valid. Some of the results raise questions about the usefulness of such instruments, what they measure and how participants responded to them. For example, in the first study, state anxiety was found to be predicting a large percentage of the variance in depression to the exclusion of other possible predictors. This finding raises the question of whether the relevant scales were measuring some 'general negative mood state' associated with the chronic pain condition, rather than two distinct constructs. This is consistent with the findings of Feldman (1993) who has also suggested that if this is the

case, perhaps they are caused by "some unitary underlying vulnerability" (p. 636). Rouillon (1999) reports that while anxiety and depression are considerably prevalent in the community, it is often difficult to discriminate between them. Not only can they occur co-morbidly, but one may predispose a person to the other or they both may represent external manifestations of an underlying cause, for example, in this case, chronic pain. The issue of discriminating between anxiety and depression remains contentious.

A similar criticism can be aimed at the other tests used. For example, the anger inventory was particularly unpopular with participants who found that it was annoying to complete. The statements and places to score are not on the same page or pages. Also, it was felt by many that the statements did not cover the extent of emotion or were not specific enough.

The MPI, undoubtedly the most comprehensive and useful test for assessing chronic pain, can be problematic. For example, the solicitous and distracting responses did not feature at all in any of the analyses, causing speculation about their relevance. Bernstein et al. (1995) have concluded that these particular scales were separately redundant and reported the same for 'activities away from home' and 'social activities'. In other research, Riley et al. (2000) found that life control and negative affect loaded on a single factor, concluding that this suggested instability of the scale.

In the present work, there appeared to be a problem with the scales used to assess 'general activity level' in the MPI. Close inspection of the actual measures and the responses revealed that most respondents were keeping up with household chores but not outside chores. An explanation for this may be that most of the sample was female and middle-aged, and traditionally it could be expected that the inner house environment is more likely to be the domain of the female of the house. It is possible that a different level of activity is required for outside household chores than inner house chores, which also may be more easily taken over by other people. Furthermore, the sub-scales which make up the general activity scale do not allow for the reporting of specific activities such as 'walking the dog' or 'going for a daily walk' which means that some participants may not have been able

to report their actual activity levels or indicate the degree of actual 'activity' involved in specific social activities.

In addition, the previously mentioned problem with profiling of chronic pain cases when there is ambiguity about 'significant others' is another area of controversy with the MPI (Okifuji et al., 1999b). While the instrument is clearly a sound measure of the cognitive-behavioural components of pain, these discrepancies indicate that further research into its psychometric qualities is warranted, especially with respect to factors such as gender. Continued updating and strict testing of questionnaires appears to be warranted in terms of relevance to contemporary subject groups assessed for research purposes.

Variables that were related to both chronic pain and depression in previous research did not demonstrate the same relationships in the present research. For example, life control was only related to depression but not to pain severity while interference was related to both. Also, pain self-efficacy was found to play a greater role in inter-relationships of variables than control. This raises the question of how different these two constructs are. While some researchers insist that self-efficacy and control are different constructs (Litt, 1988b), there is not complete consensus. Lefcourt and Davidson-Katz (1991), for example, argue that because they both imply a sense of control, they are similar.

There have been considerable arguments mounted against the use of trait measures such as those referred to above. For example, many behavioural theorists argue that traits are only of value when they are set in a context and that an understanding of the context is more important than the traits that may be involved. Currently the research on pain, as evidenced by the literature review, could be described as having a high degree of heterogeneity with little agreement in way of method or substance. What is needed to unify research is what has been referred to by Kuhn (1970) as a 'paradigm shift' with some agreement reached on methods and core concepts. Until that time longitudinal research on unselected subjects, such as that reported here may continue to provide information to assist in forming that paradigm. It was not intended that the methodology used in this thesis demonstrate the 'reciprocal determinism' that is claimed by some to be fundamental to

cognitive-behavioural theory. Besides, the findings showed, in a rudimentary way, that the theory that all elements of a chronic pain experience are 'reciprocally determined' is problematic in terms of demonstration. The methodology did reveal that the chronic pain-depression relationship is certainly multi-dimensional and interactive. No published research to date, has been able to definitively show the 'reciprocal determinism' of chronic pain. However, this present work has been informative and has taken a unique perspective of chronic pain and the problems associated with examination of this puzzling and serious condition.

7.6 Limitations of the research

Participants for this research proved to be extremely difficult to recruit. Although the first study was planned as a pilot and opportunity to test instruments and determine hypotheses, the final number of participants (N=30) was not as great as expected. The interviews necessary to develop rapport were lengthy and required significant resources influencing the number recruited. These difficulties were reflected in the difficulty of acquiring participants for Study 2 and also retaining them for the follow-up study.

A serious criticism of this type of research is that the participants are self-selected. While randomised research is considered preferable for scientific study, lack of resources usually precludes satisfactory randomisation. Also the participants were required to meet a fairly stringent set of criteria, which necessarily reduced the number of eligible participants. Advertising widely in the metropolitan area was considered necessary to ensure that the sample was as representative as possible

All data were collected via the use of self-report measures, which have been criticized because they are not considered to be objective, although such measures are generally easy to obtain as well as being resource effective. According to Jensen et al. (1999), the self-report is generally acceptable in research and continues to be a valid measure of beliefs, providing evidence for a cognitive-behavioural model. Even the more objective measures of adjustment used in this research such as rest, medication and exercise were

not necessarily more illuminating and again these also relied on self-report. It is not clear how such problems could be over-come in this type of research apart from adopting extensive observational methods.

7.7 Implications of this research for cognitive-behavioural theory

Cognitive-behavioural theory has been of considerable importance in the quest to understand chronic pain. According to this perspective, beliefs and coping behaviours are central to adjustment to chronic pain. One way of determining whether adjustment to chronic pain has occurred is to test for the presence or absence of depression in the person experiencing chronic pain. There has been a considerable amount of empirical evidence, which has demonstrated significant relationships between beliefs and measures of dysfunction, such as depression, among those who have chronic pain (Jensen et al., 1991; Turk & Okifuji, 1999). The present research has confirmed that chronic pain and depression are not only directly related but may also be mediated by specific beliefs. This has been shown to be consistent longitudinally and between genders. Although pain severity was found to effect depression levels more in females, while for males beliefs were more important, this research has also highlighted the complicated relationships between pain severity, beliefs and adjustment. Despite the limitations noted, the results of this research highlight the importance of cognitive appraisal as it relates to depression in those who experience chronic pain. The differences in the association of pain and depression between genders is of particular importance and deserves further attention as it may have implications for treatment of chronic pain at a community level.

These findings, however, indicate that the hypothesized relationships between pain, depression and beliefs are far from straightforward as suggested by cognitive-behavioural theory and that more recent conceptualisations of chronic pain and depression suggested by researchers such as Pincus and Williams (1999) and Sharp (2001) may be more viable, in so far as future directions for research have been promoted. It also highlights the difficulties of this type of research, given the array of factors that could possibly affect response to

persistent pain. This present research only concentrated on cognitive factors but as Sharp (2001) has indicated there are so many other possible interactions. In particular, anxiety, which was not adequately examined in this research, and its relationship to fear and pain need to be more fully examined. Whether these future models can be adequately tested, given their inherent complexities, remains to be seen.

There has been considerable criticism of research that has depended too much on cross-sectional samples, however, the present research has shown that even when longitudinal research is conducted, it also can be fraught with inadequacies and limitations, confirming previous difficulties with longitudinal research by others such as Brown (1990), Magni et al. (1994) and Hurwitz et al. (2003).

This research lends some support to the original findings. For example, Rudy et al. (1988) found that pain was insufficient to account for variance in depression among those with chronic pain, but rather was a consequence of low life control and increased life interference related to pain. The present results show that the relationships demonstrated by Rudy and others are not as straightforward in non-clinical groups. While those particular cognitive appraisal variables were important, this varied between groups of participants and over time. While interference was most strongly associated with pain in cross-sectional analyses, control was most dominant in longitudinal analyses. In addition, pain self-efficacy was also found to be a key variable in the cross-sectional analyses. This research has highlighted that some beliefs appear to be more important than others in the relationship between chronic pain and depression, in particular, interference, general self-efficacy, pain self-efficacy and control. However, it is also acknowledged that beliefs are only part of an array of psychosocial factors, including social and environmental context, cultural background, personal pain attributions, attitudes and biological factors that are involved in the chronic pain experience (Turk & Okifuji, 1999). Further research into the complicated relationships between cognitive factors and pain continues to be warranted. This research has added to the considerable body of knowledge about the psychological aspects of chronic pain.

7.8 Implications of this research in terms of community education

Given that Jensen et al. (1999) have noted how crucial it is that chronic pain research continues to study those people in the community who do not actively seek treatment in pain clinics, this piece of research has provided important insights into this topic. The people with chronic pain investigated in this research showed significant levels of depression and this highlights the need for health professionals to be particularly aware of the psychological consequences of unremitting pain. These results indicate that there is a need for more education at a community level. Community health centres might play a greater role in transmitting important information about the current state of knowledge about how pain is perceived and the consequences of a long-term condition. The gender differences that have been demonstrated in this research also suggest that more attention needs to be paid to the different difficulties experienced by male and females with chronic pain.

The cross-sectional analyses revealed that for males it is most important to address the interference in daily living that results from chronic pain, whereas for females, pain relief is the primary objective in terms of reducing the risk of depression. In terms of education and assistance provided by health professionals, this may be useful information. Males may benefit from more education about coping strategies and how to reduce the interference that chronic pain presents to daily life, while females may benefit from more information about pain relief, not just pharmacological but including other so-called alternative measures, such as massage and heat treatment. However, the longitudinal analysis, despite its limitations, revealed that in the long term males are affected by the specific confidence needed to deal with pain whereas females were much more affected by feelings of control over pain. These issues could also be addressed by effective education and specific training in confidence and control building techniques provided by professional psychological care.

This research also revealed that people with chronic back pain were significantly more depressed than non-pain participants. This is consistent with previous research that has reported that depression is higher among pain groups than in the rest of the community. In addition, although the people with pain reported less internal health locus of control and

general self-efficacy, their responses were not significantly different. These findings indicate that more investigation into these constructs with other pain groups is warranted. Taking responsibility for one's own health and feeling confident to function generally well on a daily level would seem to be integral to adjusting to the demands of a chronic illness. This research has highlighted how important it is to raise community awareness about the dangers of long-term pain conditions in terms of mental and physical health, social relationships and subsequent economic costs to society.

7.9 Implications for future research

Research into people with chronic pain from the community might be improved by conducting more survey type assessments as described by Magni et al. (1994). However, unlike Magni and consistent with Brown (1990) and Hurwitz et al. (2003), it might be more useful to gather data in 'waves', that is, 6-monthly or annually for 3 years or longer, if possible. This type of longitudinal research is difficult and costly to implement and is not conducive to typical higher degree research in terms of time frames. Also, there needs to be more precise criteria in terms of defining chronic pain and depression. The Magni studies defined chronic pain as that which had been present for "....at least 1 month of the 12 months preceding the interview" (Magni et al., 1994, p. 290). This definition is not consistent with the medical requirement that pain has been experienced for most of the time, for at least the previous 6 months (IASP, 1994).

With respect to acquiring control groups for future research, it might be useful to acquire participants by asking each volunteer with pain to pass a non-pain questionnaire onto a friend who does not suffer with chronic pain. This might ensure that controls are not 'significant others' and are more likely to be from similar backgrounds in terms of demographic characteristics.

This research concentrated on people with chronic pain from the community who appear to be dissimilar to most of the clinical samples described in the majority of the literature. Future research might consider comparing non-clinical to both clinical patients

with chronic pain and people without chronic pain in a three-way design, which examines the relationship between chronic pain and depression, both cross-sectionally and longitudinally, although this would be very resource and time intensive.

7.10 Conclusion

With the limited knowledge about people with chronic pain from the community as a foundation, this thesis has explored the general characteristics of such people, as well as addressed several key issues. Of particular interest was the investigation of two major questions. Firstly, are chronic pain and depression associated and if so, is this relationship direct or indirect? Secondly, does chronic pain precede depression or vice versa?

Given that this research was primarily exploratory in nature, it is reasonable to promote caution in attempting to generalize findings. Broadly, results have shown that chronic pain and depression are significantly related but indirect, non-significant relationships have also been demonstrated. This is consistent with previous research (Maxwell et al., 1998; Turk et al., 1995). This research has shown that other cognitive-behavioural variables besides interference and control (Rudy et al., 1988) are involved in the association, most particularly pain self-efficacy. With regard to the second question, the longitudinal study cannot confirm that chronic pain is an antecedent of depression.

This thesis has contributed important information about the psychological impact of chronic pain in a sample of people with chronic pain from the community. Concern that the sample may have been more clinical than not, was shown to be doubtful as the evidence demonstrated that they were dissimilar to clinical patients in several key areas. The results of this research emphasize the value of educating the general public about the possible psychological consequences of chronic back pain. In particular, acknowledging that chronic pain may be influenced by other factors, such as gender, highlights the need for awareness among health professionals involved in pain treatment and management programs. This research has implications for the development and/or enhancement of public education programs with a multi-therapy focus. Teaching people to better manage their own pain

provides opportunities for people with chronic pain to gain increased confidence and abilities to deal with the impact that chronic pain has on daily living. This is integral to reducing the burden of chronic pain on health, economic and social systems.

This thesis has investigated chronic pain from a psychological perspective, which is in keeping with the holistic approach that acknowledges the multi-dimensional and, very subjective, nature of pain. Psychological factors have been shown to be integral to a better understanding of pain from the initial stimulus to the interpretation of the pain experience to the emotional and behavioural reactions to pain. The psychological approach therefore allows an increased understanding of the cognitive processes involved in the dynamic and reciprocal process of the pain experience. Psychological factors are also important in terms of understanding the contextual meaning of pain that people with chronic pain may attribute to their pain condition. In the present research, exploring psychological factors has also provided a greater understanding of how other factors, such as gender, may influence persistent pain. Finally, by providing techniques for the exploration of models of pain, psychology plays a fundamental and crucial role in the continued search for answers to the enigma that is chronic pain.

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APPENDIX A

STRUCTURED INTERVIEW GUIDE FOR CHRONIC PAIN PATIENTS
(based on Guide developed by M. K. Nicholas PhD
University of Sydney Pain Management and Research Centre
Royal North Shore Hospital, Sydney. 1994).

*1. Where is your pain? Specify.

*2. Can you tell me how it started? When, where it began as you remember it?

*3. What type of treatment did you get?

*4. What type/s of treatment/s have you received since that time? Medical, non-medical?

*5. Are you on any medication? How often? Type (ie. prn/otc and/or prescribed)

*6. Have you had any surgery related to pain?

*7. Any accidents or trauma of any type associated with pain?

*8. Is your pain always present? (if not, how often does it occur?)

*9. Does its intensity vary?

*10. Is there any pattern to its variation? In other words, is it often worse or better at particular times of the day?

*11. What makes your pain worse? What sorts of activities are likely to make your pain worse when you do them?

*12. How do you respond when you feel your pain is increasing? [actions] [if takes medication for pain, specify what medication, and what is the pattern of use -prn or time-based?] What sort of things run through your mind when your pain increases? (if not sure) imagine you were at home (or work) now, how might you react if the pain started to build up now?

*13. What has your pain problem stopped you doing? In other words, what sort of things don't you do because of the pain? (Try to be specific, give me some examples, eg. work, home duties, sport, social family).

*14. How have you been spending your days lately? (eg. at home/work, types of activity - perhaps you could describe a typical day for you lately).

*15. Do you have 'good' days and 'bad' days? (if so, what do you do on good days that is different to what you do on bad days? [evidence of overdoing or pacing?]

*16. Looking at the evenings, what time have you been going to bed lately?

*17. After you are in bed, lights out, how long does it take you to get to sleep (lately)? Have you been taking any medication in the evenings? (if so, what tablets? how many?) What medication do you take through the day? (amount?)

*18. Have you been waking during the night lately? If so, how often?

*19. Are you ever in pain when you wake during the night?

*20. What do you do when you wake and you are in pain?

*21. What time have you been waking finally in the mornings lately? And how have you been feeling at this time? (eg. still tired, rested, pain-free?)

*22. What time have you been getting up in the mornings lately?

*23. Do you often rest or nap during the day? (if so, for how long? and how much of that is sleep and how much reclining only?)

*24. Who lives at home with you now? (spouse, children). Their ages? Are they working/not working/at school? (type of work and full-time/part-time?) and how is their health? Do they have any problems (eg. at school/work)?

*25. What does your (husband/wife/partner) do for you now that they wouldn't normally do if you didn't have pain? [check if anyone else, eg. homecare, other relatives etc, is involved in providing assistance]

*26. What does your (husband/wife/partner) not do because of your pain- things s/he would otherwise do if you didn't have pain? (ie. what difference does your pain make to her/his activities/lifestyle?)

*27. How has your pain problem affected your relationship with your (husband/wife/partner/children)?

*28. Has your pain problem affected your sexual activity? [ask unless reason to believe there is no sexual activity. you are after two things: (a) is there a change in the frequency of sexual activity as a result of the pain problem, and (b) if there is, why?]

*29. Can your (husband/wife/people around you) tell when your pain is bad? If so, how? [push for specifics, not just "by my face"].

*30. When (they) know your pain is bad, what do they do? (let me put it like this: suppose I was looking into your lounge and you were there. Your (husband/wife etc) comes in and sees that your pain is bad. What would I see and hear him/her do or say?)

*31. Do you ever feel tense or cranky (irritable) lately?

*32. When you get tense or cranky, what do you notice about your pain? Is it better/worse/no different?

*33. When you get really relaxed, what do you notice about your pain? Is it worse/better/no different?

*34. Now I'd like to ask some questions about how you have been feeling lately. how would you describe your general mood in the last couple of weeks? (happy, sad, low, high). [check for symptoms of anxiety and depression].

*35. As you look back over the last year or so [less if relevant], would you say your pain problem has been getting better, worse, or not changing?

*36. As you look down the road 1-2 years, what do you expect will happen to your pain problem? Not what you'd like to happen but what you expect will happen. (will it gradually improve, get worse, or not really change?)

*37. If your pain could be relieved completely, what would you do that you are not doing now? (be realistic).

*38. Given all the information you have about your pain, what is your best guess as to why you have pain now? What do you think is wrong with your body that is still causing your pain now (this long after surgery/injury). Not what your doctors think but what you think? (and, do you ever think you might have something seriously wrong, like cancer, which the doctor hasn't picked up?

*39. Apart from your pain problem, do you have any other major stressors or problems or big changes in your life at present, or in the last year or so (e.g. loss of member of family, friend, job, change of residence, financial issues).

APPENDIX B

PATIENT INFORMATION QUESTIONNAIRE

(based on PIQ from Royal North Shore Hospital and University of Sydney Pain Management and Research Centre, 1994)

(please remember that this information is anonymous and confidential)

Date completed:_____

1. Male_____ Female_____

2. Age
   18-19____
   20-29____
   30-39____
   40-49____
   50-59____
   60-69____
   70 and over____

3. Your country of birth_____

4. What is your current marital status: (please tick one)

   married____ defacto_____
   divorced____ separated____
   single____ widowed____

5. If you have children, how many do you have?_____

6. What are their ages?_____

7. Do you live (tick one)

   alone____ with husband/wife and children____
   with child/children only___ with other relatives____
   with spouse/partner____ with friends/flatmates____

8. What is your highest level of education (please tick one)

   University___ CAE___
   TAFE___ Year 12___
   Year 11 (leaving)___ Year 10 (intermediate)___
   Less than year 10___
   Other (please specify)_____

9. For how long have you lived in Australia?

   all my life___
   more than 10 years___
   between 6 and 10 years___
   between 2 and 5 years___
   less than 2 years____

10. What was your main occupation before your pain/injury?

_____

11. What is your current work status?

full-time work___ part-time work___
voluntary work___ home duties___
retired___ student___
unemployed due to pain___ retraining___
unemployed due to other reasons___

The next 6 questions are to do with paid work. If they do not apply please tick (not applicable).

12. If working, is your work restricted due to pain? yes___no____ (not applicable)

13. If working, how much time (in months or weeks) have you taken off work due to pain?

In the last year?_____months (or)_____weeks (not applicable)

14. If not working (and you are not retired), state for how long since you last worked:

_____years_____months (not applicable)

15. If not working now, do you have a job to go back to? yes___no____ (not applicable)

16. If not working now, have you attempted to return to work? yes___no____ (not applicable)

If yes, how long did you last? (if you returned to work more than once, what was the longest period?

less than 1 week___
1-4 weeks___
5-12 weeks___
3-6 months___
7 months-1 year___
more than 1 year___

17. If not working now, but you would like to return to work, do you feel you are currently able to work at your regular job? (please tick ONE)

as much as I could prior to my pain/injury___ (not applicable)
only with reduced hours or a lot of help___
in my present condition I can't work at all___

PAIN HISTORY

1. Please describe your pain problem

.....
.....
.....

2. How did your pain begin: (tick ONE; if more than one applies, tick the one which applies BEST)

accident at work___
at work, but not involving an accident___
accident at home___
car accident___
after surgery___
after an illness___
pain just began, no clear reason___

other reasons (please describe)

_____

_____

3. Which statement best describes your pain?

always present, always the same intensity___
always present, intensity varies___
usually present, but have short periods without pain___
often present, but have pain-free periods lasting up to several hours___
often present, but am pain-free for much of the day___
occasionally present for brief periods, but not every day___
rarely present - have pain episodes every now and then, with days or weeks in between___

4. What makes your pain worse? (you may tick more than one)

sitting	household chores	cold weather	sex
standing	everything	hot weather	stress
lying-down	loud noise	wet weather	tension
lifting	working	weather changes	driving
bending	any movement	walking	going up/down stairs
no clear reason			

other (please describe).....

5. What makes your pain better? (you may tick more than one)

sitting	watching TV	cold weather	sex
standing	working	hot weather	alcohol
lying-down	warm/hot bath	pressure	rest
stretching	warm/hot shower	massage/rubbing	nothing
relaxing	tablets	walking	being with other people
reading	hot/cold packs	keeping busy	keeping my mind off pain
sleep			

other (please describe).....

6. Please rate the intensity of your pain by circling a number on the following scales.

For every question: 0='no pain at all' and 10='worst pain imaginable'

a) How intense is your pain at this moment?

0	1	2	3	4	5	6	7	8	9	10
No pain										Worst pain imaginable

b) What were the highest and lowest levels of your pain in the last week
(make 2 circles)

0	1	2	3	4	5	6	7	8	9	10
No pain										Worst pain imaginable

c) What was the usual level of your pain in the last week

0	1	2	3	4	5	6	7	8	9	10
No pain										Worst pain imaginable

MEDICATION

1. Do you think you need more medication, or stronger medication, than you are currently taking? To answer, circle one of the numbers on the scale below.

1	2	3	4	5
agree strongly	agree	unsure	disagree	disagree strongly

2. Please list all the medications you are taking **at present**:

Medication	Dose	how often?	side effects?	date started

3. Please list all the medications you have taken **in the past** for your pain?

Medication	Dose	how often?	side effects?	comments

4. Please list any allergies you may have?

.....

.....

.....

5. Since your pain began, which of the following people have you seen about it? (tick all those that apply)

Tick all those that apply	Number of times Seen in the past year (weekly, monthly etc)	Duration of visits	How helpful (not, some, very)
__Acupuncturist			
__Anaesthetist			
__Chiropractor			
__Homeopath			
__Hypnotherapist			
__Neurologist			
__Neurosurgeon			
__Occupational therapist			
__Orthopaedic surgeon			
__Physiotherapist			
__Psychologist			
__Psychiatrist			
__Rheumatologist			
__Pain clinic			
__General practitioner			
__Other _____			

6. Of these health care workers, who was the most helpful and why?

_____

_____

_____

7. Of these health care workers, who was the least helpful and why?

_____

_____

_____

8. Based on your pain experience, what do you realistically expect will happen to your pain in the coming months: (tick ONE)

it will get worse___
it will not change___
it will be reduced by 25%___
it will be reduced by 50%___
it will be reduced by 75%___
it will be completely relieved or cured___

6. If your pain could be reduced, but not completely, how much of a reduction would there need to be for you to feel that you could live with it?

My pain would need to be reduced by_____% for me to be able to live with it.

7. Do you think that your pain may be due to a serious disease which your doctors have not found or have not told you about? (tick one) yes___no___not sure

8. TIME LINE

List by year (starting at childhood) all illnesses and operations you have had since childhood.

YEAR	OPERATIONS (surgical: e.g. cervical fusion)	YEAR	ILLNESSES (medical: eg. measles, diabetes)

Thank you so much for completing this questionnaire.

It will help us to better understand your pain problem.

APPENDIX C

PAIN: S-E QUESTIONNAIRE
MKN 1988
Pain Management Centre
St. Thomas' Hospital, London.

Please rate how confident you are that you can do the following things at present, despite the pain. To indicate your answer circle one of the numbers on the scale under each item, where 0 = not at all confident and 6 = completely confident.
For example:

0	1	2	3	4	5	6
Not at all confident						Completely confident

Remember, this questionnaire is not asking whether or not you have been doing these things, but rather how confident you are that you can do them at present, despite the pain.

---

1). I can enjoy things, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

2). I can do most of the household chores (e.g. tidying-up, washing dishes, etc. despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

3). I can socialize with my friends or family members as often as I used to do, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

4). I can cope with my pain in most situations.

0	1	2	3	4	5	6
Not at all confident						Completely confident

PTO

5). I can do some form of work, despite the pain
('work' includes housework, paid and unpaid work).

0	1	2	3	4	5	6
Not at all confident						Completely confident

6). I can still do many of the things I enjoy doing, such as hobbies or leisure activities, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

7). I can cope with my pain without medication.

0	1	2	3	4	5	6
Not at all confident						Completely confident

8). I can still accomplish most of my goals in life, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

9). I can live a normal lifestyle, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

10). I can gradually become more active, despite the pain.

0	1	2	3	4	5	6
Not at all confident						Completely confident

ADELAIDE UNIVERSITY PSYCHOLOGY DEPARTMENT

BACK PAIN ?

A DISTRESSING PROBLEM

WE NEED VOLUNTEERS FOR RESEARCH!

If you are troubled by this difficult problem then we need details of your experience for our research programme.

We only require you to complete several questionnaires which will be mailed to you with a reply-paid envelope. Confidentiality and anonymity are assured. If you can help us, please contact Della Steen on 83033965

APPENDIX E

Chronic Pain Research

Information sheet for participants

Thank you for agreeing to take part in this research that is being conducted in the Department of Psychology, at the University of Adelaide. We are investigating the effects of chronic back pain, e.g. pain that has been present for at least 3 to 6 months or more. It appears that people deal with back pain in many different ways. Therefore, we are interested in the various factors that may be involved in your pain experience, such as how you cope with your pan and whether you get depressed or angry. The reason we wish to learn more about the effect of chronic pain is so that this knowledge can be used to improve treatment procedures for people with this condition.

To help with this investigation please complete the enclosed questionnaires. Before you complete them, please read and sign the enclosed consent form indicating that you have read this information sheet and have given your consent to take part in the research. Even though you must sign the consent form, **any information that you provide on the questionnaires will be completely confidential and anonymous**. There is no need for you to put your name on the questionnaires. People participating in this study will not be identified in any report of this research. We also assure you that we are conducting independent research and are not connected with any other organisation.

Completing the questionnaires should take 1 to 2 hours of your time. You do not have to do it all at one sitting but please try to complete them all on the same day if you are able. It is important to fill them out in the order that they are stapled together and to answer every question, if possible. Some of the questionnaires are double-sided so please be sure to check that you have completed them all. You may also think some questions are the same but still complete them anyway. In addition, **please be aware that there are no right or wrong answers**. We are only interested in **your** feelings and beliefs. We would like **your** answers to the questions and therefore would ask that you do them on your own, if possible.

When you have completed the questionnaires, please return them in the reply-paid envelope that has been provided. **If you decide not to complete the questionnaires, please return them to the University as failing to do so is a breach of copyright.**

If you have any questions, please contact Della at the University of Adelaide, Department of Psychology, on 83033965 or Dr. Don Pritchard, on 83033172.

Thank you very much for your help.

Ms. Della Steen,
PhD Candidate.

Dr. Don Pritchard (Supervisor),
Clinical Senior Lecturer, Department of Psychology.

APPENDIX F

GENERAL PERSONAL MASTERY
(from Sherer et al., 1982)

This questionnaire relates to your personal mastery of a variety of general experiences. You are asked to rate your agreement with each of the items below by writing the appropriate number in the blank space using the scale of 1 to 7 illustrated below.

1=strongly disagree
2=disagree
3=slightly disagree
4=neither agree nor disagree
5=slightly agree
6=agree
7=strongly agree

PLEASE BE SURE TO ANSWER EVERY STATEMENT

_____1. When I make plans, I am certain I can make them work.

_____2. One of my problems is that I cannot get down to work when I should.

_____3. If I can't do a job the first time, I keep trying until I can.

_____4. When I set important goals for myself, I rarely achieve them.

_____5. I give up on things before completing them.

_____6. I avoid facing difficulties.

_____7. If something looks too complicated, I will not even bother to try it.

_____8. When I have something unpleasant to do, I stick to it until I finish it.

_____9. When I decide to do something, I go right to work on it.

_____10. When trying to learn something new, I soon give up if I am not initially successful.

_____11. When unexpected problems occur, I don't handle them well.

_____12. I avoid trying to learn new things when they look too difficult for me.

_____13. Failure just makes me try harder.

_____14. I feel insecure about my ability to do things.

_____15. I am a self-reliant person.

_____16. I give up easily.

_____17. I do not seem capable of dealing with most problems that come up in life.

APPENDIX G

ADELAIDE UNIVERSITY PSYCHOLOGY DEPARTMENT

VOLUNTEERS NEEDED URGENTLY!!!

If you are aged over 18 years and do NOT suffer with any type of chronic pain, you may be able to help us.

We wish to compare a group of people who do not suffer chronic pain with the chronic pain sufferers who have already helped us with our research.

We only require you to complete several questionnaires which will be mailed to you with a reply-paid envelope. These questionnaires will take you about 30 minutes to complete. Confidentiality and anonymity are assured. If you can help us, please contact Della Steen on 83033965.

APPENDIX H

Chronic Pain Research

Information sheet for non-pain participants

Thank you for agreeing to take part in this research that is being conducted in the Department of Psychology, at the University of Adelaide. We are investigating the effects of chronic back pain, e.g. pain that has been present for at least 3 to 6 months or more. It appears that people deal with back pain in many different ways and we are trying to learn more about this. We also need to be able to compare pain sufferers with people who do not suffer with chronic pain, especially with regard to effects such as depression, anger and loss of self-confidence which are commonly experienced by people in constant pain. We hope that this knowledge can be used to improve treatment procedures for people with this condition.

To help with this investigation please complete the enclosed questionnaires. Before you complete them, please read and sign the enclosed consent form indicating that you have read this information sheet and have given your consent to take part in the research. Even though you must sign the consent form, **any information that you provide on the questionnaires will be completely confidential and anonymous**. There is no need for you to put your name on the questionnaires. People participating in this study will not be identified in any report of this research. We also assure you that we are conducting independent research and are not connected with any other organisation.

Completing the questionnaires should take about 30 minutes or so of your time. It is important to fill them out in the order that they are stapled together and to answer every question, if possible. Some of the questionnaires are double-sided so please be sure to check that you have completed them all. You may also think some questions are the same but still complete them anyway. In addition, **please be aware that there are no right or wrong answers**. We are only interested in **your** feelings and beliefs. We would like **your** answers to the questions and therefore would ask that you do them on your own, if possible.

When you have completed the questionnaires, please return them in the reply-paid envelope that has been provided. **If you decide not to complete the questionnaires, they must still be returned to the University as failing to do so is a breach of copyright.**

If you have any questions, please contact Della at the University of Adelaide, Department of Psychology, on 83033965 or Dr. Don Pritchard, on 83033172.

Thank you very much for your help.

Ms. Della Steen,
PhD Candidate.

Dr. Don Pritchard (Supervisor),
Clinical Senior Lecturer, Department of Psychology.

APPENDIX I

Date

Dear

About 5 years ago you kindly helped me with research on the effects of chronic back pain by filling out a survey questionnaire about how you dealt with pain. I am very grateful for your help. Your contribution, and that of others, helped explain how people are able to deal with a painful and ongoing condition. This information is extremely valuable to health care workers, employers and others who are often unaware of the difficulties faced by people who suffer with chronic pain.

You may remember that at our last contact, I said I hoped to do further research into the impact of chronic pain. I am therefore inviting you to take part in a final, follow-up survey about the *long-term effects* of back pain. I hope this survey will increase our understanding of chronic pain and the stress many people with back pain suffer on a daily basis. I believe that this final survey is important because back pain can change over time and very little is known about how people deal with such long term changes. I realize that your condition could have improved, worsened or remained the same since our last contact. The survey I am now doing deals with whether <u>your</u> experience of pain has changed at all and, if so, how.

To take part all you need to do is fill in the enclosed questionnaire and return it to me in the reply paid envelope. I hope you will take the time (about 20 minutes) to fill it in and return it to me within the next two weeks. If you do not wish to take part, could you please return the uncompleted questionnaire to me indicating your reason (space is available for this on the last page)? If I have not received your copy of the questionnaire within a couple of weeks, I will contact you to see how it's going. No matter what you decide to do, as before, any information you provide will be **completely confidential and anonymous**. I am very grateful for the help you have already provided and sincerely hope your condition has improved since you took part in the previous survey.

Regards,

Della Steen
Researcher
83035552

Dr. Helen Winefield
Senior Researcher
Psychology Department
University of Adelaide

APPENDIX J

Chronic Back Pain Research

Information sheet for participants in Follow-Up Survey

Thank you for your previous participation in this research being conducted in the Department of Psychology, at the University of Adelaide. In this final survey, we are interested in the *long-term* effects of chronic back pain. It appears that people deal with back pain in many different ways and we are trying to learn more about this. While we have some very valuable information from the earlier survey, little is known about how people deal with pain over a long period of time. This is important because we know that the experience of pain can change over time. We hope that this new information will improve the understanding of health care workers and the general public who may be unaware of the difficulties faced by people who suffer persistent pain.

To help with this research please complete the enclosed survey questionnaire. There is no need for you to put your name anywhere, as each set of questionnaires is identified by number only. **Any information that you provide will be completely confidential and anonymous**. People participating in this survey will not be identified in any report of this research. We also assure you that we are conducting independent research and are not connected with any other organisation.

Completing the questionnaire should take about 20 minutes of your time. It is important to fill it out in the order it is stapled together and to answer every question, if possible. Some of the pages may be double-sided so please be sure to check that you have completed them all. You may also think some questions are the same as others but still complete them anyway. In addition, **there are no right or wrong answers**. We are only interested in **your** feelings and beliefs. We would like **your** answers to the questions and therefore would ask that you do them on your own, if possible.

When you have completed the questionnaire, please return it in the reply-paid envelope that has been provided. **If you do not wish to take part in this survey, please return the questionnaire uncompleted. It would also be very helpful to us if you could tell us why you are unable to take part. There is a place for this on the last page of the questionnaire.**

If you have any questions, please contact Della on 83035552 or Dr. Helen Winefield (supervisor) on 83033172. If you have any ethical concerns and you wish to speak to someone not connected to the research, please contact Dr. Peter Delin on 83035007.

Once again, thank you very much for your help.

Della Steen,
Researcher

Dr. Helen Winefield
Senior Researcher
Psychology Department
University of Adelaide

APPENDIX K

Table 1. Multiple regression analysis: Predictors of depression for responders at Time 2 (N=44).

Predictor Variables	B	CI [95]	Beta	t	R^2	Adj R^2	R^2cha	F (df)
Dependent Variable Depression 2								
Model 1								
Depression 1	.75	.39, 1.11	.55	4.24***	.30	.28	.30***	17.99*** (1, 42)
Model 2								
Depression 1	.66	.23, 1.08	.48	3.13**	.31	.28	.01	9.27***
Pain Severity 1	1.15	-1.66, 3.96	.13	.83				(2, 41)
Model 3								
Depression 1	.52	.092, .93	.37	2.46*	.40	.35	.09*	8.84***
Pain severity 1	.87	-1.81, 3.54	.09	.65				(3, 40)
Control 1	-5.28	-9.71, -.856	-.32	-2.41*				

*p<0.05; **p<0.01; ***p<0.001

Table 2. Multiple regression analysis: Predictors of pain severity for responders at Time 2 (N=44).

Predictor Variables	B	CI [95]	Beta	t	R^2	Adj R^2	R^2cha	F (df)
Dependent Variable Pain severity 2								
Model 1								
Pain severity 1	.56	.28, .84	.53	4.03***	.28	.26	.28***	16.20*** (1, 42)
Model 2								
Pain Severity 1	.47	.14, .80	.44	2.84**	.30	.26	.02	8.66**
Depression 1	.03	-.02, .08	.16	1.04				(2, 41)
Model 3								
Pain severity 1	.41	.07, .75	.38	2.42*	.33	.28	.04	6.64**
Depression 1	.02	-.03, .07	.11	.69				(3, 40)
Pain self-efficacy 1	-.02	-.06, .01	-.21	-1.46				
Model 4								
Pain severity 1	.40	.06, .73	.38	2.4*	.37	.30	.04	5.66**
Depression 1	.00	-.05, .06	.01	.04				(4, 39)
Pain self-efficacy 1	.02	-.06, .01	-.19	-1.32				
Activity 1	-.41	-.98, .16	-.22	-1.47				
Model 5								
Pain severity 1	.40	.03, .77	.38	2.21*	.37	.28	.00	6.79**
Depression 1	.00	-.05, .06	.01	.05				(5, 38)
Pain self-efficacy 1	-.02	-.07, .02	-.19	-1.17				
Activity 1	-.41	-.99, .16	-.22	-1.45				
Interference 1	-.00	-.41, .39	-.01	-.05				

*p<0.05; **p<0.01; ***p<0.001

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