THE IMPACT OF NATURE OF ONSET OF PAIN AND POSTTRAUMATIC STRESS ON ADJUSTMENT TO CHRONIC PAIN AND TREATMENT OUTCOME

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- together, you are my everything.

Abstract

Despite the demonstrated efficacy of cognitive-behavioural therapy for chronic pain, recent research has attempted to identify predictors of treatment outcome in order to improve the effectiveness of such treatments. This research has indicated that variables such as the nature of the onset of the pain and psychopathology are associated with poor adjustment to chronic pain. Accordingly, these variables might also be predictive of poor response to treatment. Individuals who experience a sudden onset of pain following an injury or accident, particularly when the instigating event is experienced as psychologically traumatic, may present for treatment with high levels of distress, including symptoms consistent with a posttraumatic stress response. The impact of this type of onset of pain and posttraumatic stress symptoms on adjustment to chronic pain and treatment outcome is the focus of this thesis. Three studies were conducted to clarify and extend earlier research findings in this area.

Using 536 patients referred for treatment in a tertiary referral pain management centre, the first study examined the psychometric properties of a widely used self-report measure of posttraumatic stress symptoms (the PTSD Checklist, or PCL), modified for use in a chronic pain sample. This study provided preliminary support for the suitability of the PCL as a self-report measure of Posttraumatic Stress Disorder (PTSD) symptoms in chronic pain patients. However, the study also highlighted a number of issues with the use of self-report measures of posttraumatic stress symptoms in chronic pain patient samples. In particular, PCL items enquiring about symptoms which are a common aspect of the chronic pain experience (e.g. irritability, sleep problems) appeared to contribute to high mean scores on the PCL Avoidance and Arousal subscales. Furthermore, application of diagnostic cut-off scores and an algorithm recommended for the PCL in other trauma groups suggested that a much larger proportion of the sample was identified as potentially

meeting diagnostic criteria for PTSD than would have been expected from previous research.

The second study utilised the modified PCL to investigate the impact of different types of onset of pain (e.g. traumatic onset) and posttraumatic stress symptoms on adjustment to chronic pain in a sample of 196 chronic pain patients referred to the same centre. For patients who experienced the onset of pain related to a specific event, two independent experts in the field of PTSD determined whether these events satisfied the definition of a traumatic event according to DSM-IV diagnostic criteria. Adjustment was assessed through a number of validated measures of mood, disability, pain experience, and pain-related cognitions. Contrary to expectations, comparisons between patients who had experienced different types of onset of pain revealed few significant differences between them. That is, analyses comparing patients presenting with accident-related pain, or pain related to other specific events, to patients who had experienced spontaneous or insidious onset of pain revealed no significant differences between the groups on measures of pain severity, pain-related disability, and symptoms of affective distress after adjustment for age, pain duration, and compensation status. Similarly, comparisons between patients who had experienced a potentially traumatic onset of pain with those who had experienced a non-traumatic or spontaneous or insidious onset of pain also revealed no significant differences on the aforementioned variables. In contrast, compensation status, age, and a number of cognitive variables were significant predictors of pain severity, pain-related disability, and depression.

The final study investigated the impact of type of pain onset and posttraumatic stress symptoms on response to a multidisciplinary cognitive-behavioural pain management program. Unlike the previous study, this treatment outcome study revealed a number of differences between onset groups. Most notably, patients who had experienced

an insidious or spontaneous onset of pain reported greater improvements in pain severity and maintained these improvements more effectively over a one month period than patients who had experienced pain in the context of an accident or other specific incident. There was also limited evidence that improvements in depression favoured patients who had experienced an insidious or spontaneous and non-traumatic onset of pain. Consistent with this, posttraumatic stress symptoms were a significant predictor of treatment outcome, with higher levels of symptoms being associated with smaller improvements in pain-related disability and distress. Notably, this study also revealed that certain cognitive variables (i.e. catastrophising, self-efficacy, and fear-avoidance beliefs) were also significant predictors of treatment outcome, consistent with previous findings in the pain literature. This provided some perspective on the relative roles of both PTSD symptoms and cognitive variables in adjustment to persisting pain and treatment response. These findings were all consistent with expectations and with previous research. Implications for future research and for the assessment and treatment of chronic pain patients who present with posttraumatic stress symptoms are discussed.

TABLE OF CONTENTS

1. OVERVIEW OF CHRONIC PAIN 1

1.1. Chronic pain 1

1.1.1. The scope of the problem 2

1.1.2. Associated features and the costs of chronic pain 4

1.2. Conceptualisations of pain 7

1.3. Psychological models of chronic pain 10

1.3.1. Psychodynamic models 10

1.3.2. Behavioural models of pain 11

1.3.3. Cognitive-behavioural models 16

1.4. Treatment 22

1.4.1. Behavioural and cognitive-behavioural treatments for chronic pain 22

1.4.2. Treatment issues 24

1.5. Thesis overview 27

2. THE ROLE OF ONSET OF PAIN 29

2.1. Onset of pain 29

2.2. Onset of pain and adjustment to chronic pain 31

2.2.1. Onset of pain related to a specific incident 31

2.2.2. Onset of pain related to accidents 36

2.2.3. Summary 40

2.3. Potential explanations for the differences observed between onset groups 41

2.3.1. Onset of pain related to a specific incident 41

2.3.2. Onset of pain related to accidents 47

2.3.3. Summary 53

2.4. Conclusions and implications for research 54

3. OVERVIEW OF TRAUMATIC EVENTS AND PTSD 56
3.1. The definition of a traumatic event 56
3.2. Posttraumatic Stress Disorder 61
3.2.1. Prevalence of PTSD 62
3.2.2. Prognosis and associated features 65
3.2.3. Risk factors for the development of PTSD 67
3.3. PTSD following traumatic injury 68
3.4. Psychological models of PTSD 71
3.3.1. Learning theory model of PTSD 71
3.3.2. Ehlers and Clark's (2000) cognitive model of PTSD 74
3.5. Treatment of PTSD 78
3.6. Chapter summary 82
4. CHRONIC PAIN AND PTSD 83
4.1. Comorbidity of chronic pain and PTSD 83
4.1.1. Prevalence of PTSD in chronic pain 84
4.1.2. Prevalence of chronic pain in PTSD 87
4.2. Impact of the co-occurrence of chronic pain and PTSD on pain and adjustment-related variables 88
4.2.1. Pain severity 88
4.2.2. Pain-related disability and affective distress 89
4.2.3. Summary 92
4.3. Potential explanations of the relationship between chronic pain and PTSD 93
4.3.1. The aetiology of comorbid chronic pain and PTSD 93
4.3.2. The mutual maintenance model 102
4.3.3. Summary 111

4.4. Limitations of the research on chronic pain and PTSD 111
4.4.1. Self-report measures of posttraumatic stress symptoms 112
4.4.2. Association of symptoms to specific traumatic events 115
4.4.3. Evaluation of the nature of the events reported 117
4.4.4. Overlap between chronic pain and PTSD 118
4.5. Chapter summary 119
5. IMPLICATIONS FOR TREATMENT 120
5.1. Impact of type of onset of pain on treatment outcome 120
5.2. Comorbid chronic pain and PTSD – Impact on response to treatment 123
5.2.1. Impact of PTSD on treatment for chronic pain 123
5.2.2. Impact of chronic pain on treatment for PTSD 125
5.2.3. Research implications 129
5.3. Treatment implications 129
5.4. Chapter summary 131
6. STUDY 1 - PSYCHOMETRIC PROPERTIES OF A MODIFIED VERSION OF THE PTSD CHECKLIST (PCL) 132
6.1. Introduction 132
6.2. Method 136
6.2.1. Participants 136
6.2.2. Procedure 136
6.2.3. Measures 137
6.2.4. Aims 145
6.2.5. Data analyses 145
6.3. Results 151
6.3.1. Participant characteristics 151

6.3.2. Preliminary analyses 154
6.3.3. Main analyses 159
6.4. Discussion 168
6.4.1. The modified PCL 169
6.4.2. Comparisons with PCL studies in other populations 171
6.4.3. Psychometric properties of the PCL 174
6.4.4. Factor structure of the PCL 176
6.4.5. Application of the diagnostic algorithm and cut-off scores 177
6.5. Summary 179
7. STUDY 2 - IMPACT OF TYPE OF ONSET OF PAIN ON PAIN-RELATED ADJUSTMENT 181
7.1. Introduction 181
7.2. Method 183
7.2.1. Participants 183
7.2.2. Procedure 184
7.2.3. Classification of participants into onset groups 184
7.2.4. Measures 186
7.2.5. Aims and hypotheses 186
7.2.6. Data analyses 188
7.3. Results 191
7.3.1. Participant characteristics 191
7.3.2. Preliminary analyses 191
7.3.3. Main Analyses 195
7.4. Discussion 211
7.4.1. Impact of type of onset of pain 212

7.4.2. Construct validity of the PCL 221
7.4.3. Diagnostic utility of the PCL 223
7.5. Summary 227
8. STUDY 3 - IMPACT OF TYPE OF ONSET OF PAIN AND POSTTRAUMATIC STRESS SYMPTOMS ON TREATMENT OUTCOME 229
8.1. Introduction 229
8.2. Method 231
8.2.1. Participants 231
8.2.2. Procedure 232
8.2.3. Measures 233
8.2.4. Aims and hypotheses 236
8.2.5. Data analyses 238
8.3. Results 241
8.3.1. Participant characteristics 241
8.3.2. Preliminary analyses 241
8.3.3. Main analyses 243
8.4. Discussion 256
8.4.1. Impact of type of onset of pain on treatment outcome 257
8.4.2. Predictors of treatment outcome 258
8.4.3. Onset of pain and treatment history 260
8.5. Summary 260
9. DISCUSSION 262
9.1. Main findings 263
9.1.1. Study One 263
9.1.2. Study Two 265

9.1.3. Study Three 266
9.2. Limitations of the current studies 267
9.3. Strengths of the current studies 269
9.4. Theoretical implications 270
9.5. Clinical implications 272
9.6. Implications for future research 273
9.7. Conclusion 274
REFERENCES 276

LIST OF TABLES

Table 3.1. Examples of traumatic events according to DSM-IV Criterion A……59

Table 6.1: Participants' demographic characteristics compared with the normative sample (Nicholas et al., 2008)..... 152

Table 6.2: Participants' pain-related characteristics compared with the normative sample (Nicholas et al., 2008)..... 153

Table 6.3: Means and standard deviations for self-report measures in the current study and in the normative sample (Nicholas et al., 2008)..... 154

Table 6.4: Comparison of patients who consented and did not consent to participate in research..... 155

Table 6.5: Descriptive statistics for the PCL Scales..... 159

Table 6.6: PCL Item means, standard deviations and percentage of participants rating each item as "3" or above..... 161

Table 6.7: Corrected item-total correlations and Cronbach's alpha correlation coefficient if item is deleted..... 162

Table 6.8: Comparison of eigenvalues from PCA and PAF and the corresponding criterion values obtained from parallel analysis..... 164

Table 6.9: Pattern matrices (loadings)* and communalities (h2) for the rotated two-factor solution using PCA and PAF..... 166

Table 6.10: Corrected item-total factor correlations and Cronbach's alpha correlation coefficient for factors if item is deleted..... 167

Table 7.1: Participants' demographic and pain-related characteristics compared with the normative sample (Nicholas et al., 2008)..... 192

Table 7.2: Comparison of patients who consented and did not consent to participate in research..... 193

Table 7.3: Onset of pain data for the Accident onset comparisons 195
Table 7.4: Demographic and pain-related variables by group (Accident onset comparisons) 196
Table 7.5: Accident group comparison for pain severity and pain-related disability (variables not transformed) 197
Table 7.6: Accident group comparison for anxiety and stress symptoms (transformed variables) 198
Table 7.7: Accident group comparison for depression symptoms (non-parametric tests) 199
Table 7.8: Onset of pain data for the traumatic onset comparisons 200
Table 7.9: Demographic and pain-related variables by group (Traumatic onset comparisons) 201
Table 7.10: Traumatic group comparison for pain severity and pain-related disability (variables not transformed) 202
Table 7.11: Traumatic group comparison for depression and anxiety symptoms (non-parametric) 203
Table 7.12: Traumatic group comparison for stress symptoms (transformed) 203
Table 7.13: Descriptive statistics for the PCL scales 204
Table 7.14: Traumatic group comparison for PCL scales (transformed variables) 205
Table 7.15: Traumatic group comparison for the PCL Reexperiencing subscale (non-parametric tests) 206
Table 7.16: PCL scores and type of onset of pain of participants noted as reporting PTSD symptoms in their medical file 207
Table 7.17: The results of the diagnostic algorithm across traumatic onset groups 208
Table 7.18: Results of GLMs including cognitive variables 211

Table 8.1: Participants' demographic and clinical characteristics 242

Table 8.2: Onset of pain data for the Accident onset comparisons 243

Table 8.3: Onset of pain data for the traumatic onset comparisons 244

Table 8.4: Demographic and clinical variables by group (Accident/Specific Incident comparisons) 245

Table 8.5: Demographic and clinical variables by group (Traumatic onset comparisons) 245

Table 8.6; Unadjusted means (standard deviations) for the outcome variables for each onset group and the total sample at all time points 247

Table 8.7: Adjusted mean (LS mean)* for the outcome variables across all time points (Accident/Specific Incident onset comparison) 248

Table 8.8: Adjusted mean (LS mean)* differences overall and 95% CI (Accident/Specific Incident onset comparison) 248

Table 8.9: Adjusted mean (LS mean)* for the outcome variables across all time points (Traumatic onset comparison) 250

Table 8.10: Adjusted mean (LS mean)* differences overall and 95% CI (Traumatic onset comparison)E 250

Table 8.11: Summary of treatment outcome predictors* 255

LIST OF FIGURES

Figure 2.1: Different types of onset of pain reported in epidemiological and clinical studies 29

Figure 2.2: Types of onset of pain derived from previous research..... 55

Figure 8.1: Adjusted effect sizes – Accident/Specific Incident onset comparison 251

Figure 8.2: Adjusted effect sizes – Traumatic onset comparisons (Non-traumatic vs Insidious/Spontaneous)..... 252

Figure 8.3: Adjusted effect sizes – Traumatic onset comparisons (Traumatic vs Non-traumatic)..... 252

Figure 8.4: Adjusted effect sizes – Traumatic onset comparisons (Traumatic vs Insidious/Spontaneous)..... 253

1. OVERVIEW OF CHRONIC PAIN

1.1. Chronic pain

Pain is a ubiquitous human experience. Australian data from a range of sources indicates that pain is a common symptom, often prompting individuals to seek medical attention. In the 1995 National Health Survey, headaches were the most commonly reported individual illness condition, and 24% of the sample had used pain relievers in the two weeks prior to the survey (Australian Bureau of Statistics, 1995). National general practice activity statistics indicate that almost four million visits to general practitioners between 1998 and 2004 were related to abdominal pain or headaches (BEACH Program, 2004; Charles, Ng & Britt, 2005), and back pain was the sixth most common reason to visit a general practitioner in 1998-9 (Bolton & Mira, 2000). Consistent with this, in a population-based study of over 1900 adults randomly selected from the electoral roll, Walker, Muller and Grant (2004) reported that over 64% of respondents had experienced at least one episode of back pain in the previous six months.

Most experiences of pain can be classified as acute pain; that is, short-term pain that resolves spontaneously or is responsive to treatment (Bonica, 1980). In other words, although the experience of pain is common, most of these experiences are temporary. For example, while up to 80% of the general population experiences low back pain at some point in their lives, 90% of these episodes resolve within six weeks (Carragee & Hannibal, 2004).

However, some pain experiences do not resolve and develop into a chronic problem. Chronic pain is defined as pain that persists beyond the expected healing time of an injury or disease process (Bonica, 1990). Although in practice the time taken for this to occur varies, the International Association for the Study of Pain (IASP, 1986) defines

pain as chronic once it has persisted for longer than three months. Unlike acute pain, chronic pain does not serve the important biological function of being a warning signal of damage (Bonica, 1980), and tends to be refractory to treatment (Waddell, 2004).

1.1.1. The scope of the problem

Chronic pain has been referred to as a major public health problem (Crombie, 1997), and has been described as imposing a significant burden on the economy, society, and the individual sufferer (Smith, Macfarlane & Torrance, 2007). The results of epidemiological studies, both in Australia and internationally, have supported these observations, indicating that chronic pain is both a pervasive and costly problem.

Helme and Gibson (1997) surveyed elderly Australians selected randomly from electoral rolls and reported a prevalence of 51% in the 65-74 year age group, and 55% in those aged 85 years and over. In the 2001 National Health Survey, 21% of the sample surveyed reported having long-term back and disc problems, and 14% reported problems with arthritis (Australian Bureau of Statistics, 2001b).

Only one study, Blyth, March, Brnabic et al. (2001), has specifically examined the prevalence of chronic pain in the adult Australian population. In this study chronic pain was defined as pain experienced every day for three months in the six months prior to the survey, and over 17000 participants randomly selected across New South Wales were interviewed as part of the 1997 State Health Survey. Twenty percent of females and 17.1% of males reported chronic pain. The authors noted that more respondents reported chronic pain than other chronic conditions included in the survey, including diabetes, hypertension, asthma, and depression (Blyth et al., 2001).

Although there have only been a few studies of chronic pain prevalence conducted in Australia (Blyth et al., 2001), a large body of international research indicates that

chronic pain is a common problem in other countries. These studies report prevalence figures ranging between 2% (Kohlmann, 1991; cited in Verhaak, Kerssens, Dekker, Sorbi & Bensing, 1998) and 55.2% (Andersson, Ejlertsson, Leden & Rosenberg, 1993) in the general population. In a review of 15 prevalence studies, Verhaak et al. (1998) reported a median point prevalence of chronic pain of 15% in the population. In a more recent review, Harstall and Ospina (2003) reported that the weighted mean prevalence of chronic pain across studies using IASP criteria to define chronic pain was 35.5%. The variation in the prevalence figures reported in epidemiological studies is notable, and several authors have argued that this is due to the heterogeneity in methodologies across studies; for example, the application of different definitions of pain, the use of diverse samples, and a wide range of questions used to enquire about the experience of pain (Crombie, Davies & Macrae, 1994; Harstall & Ospina, 2003; Von Korff & Le Resche, 2005).

Despite the differences in methodology across epidemiological studies, a number of fairly consistent findings have been reported. The body sites most commonly associated with chronic pain appear to be the back, head, and lower extremities (Von Korff, Dworkin, Le Resche & Kruger, 1988; Catala, Reig, Artes, Aliaga, Lopez & Segu, 2002). Studies have also noted high rates of joint pain and headaches among individuals reporting chronic pain (Von Korff et al., 1988; Gureje, Von Korff, Simon & Gater, 1998). Chronic pain has often been identified as being more prevalent in women (e.g. Magni, Caldieron, Rigatti-Luchini & Merskey, 1990; Magni, Rossi, Rigatti-Luchini & Merskey, 1992; Croft, Rigby, Boswell, Schollum & Silman, 1993; Buskila, Abramov, Biton & Neumann, 2000; Blyth et al., 2001; Eriksen, Jensen, Sjogren, Ekholm & Rasmussen, 2003), and as varying in prevalence with age (e.g. Brattberg, Thorslund & Wikman,

1989; Andersson et al., 1993; Eriksen et al., 2003; Rustoen, Wahl, Hanestad, Lerdal, Paul & Miaskowski, 2005).

1.1.2. Associated features and the costs of chronic pain

Several studies (e.g. Magni et al., 1992; Magni, Marchetti, Moreschi, Merskey & Rigatti-Luchini, 1993; Elliott, Smith, Penny, Smith & Chambers, 1999; Blyth et al., 2001; Eriksen et al., 2003) have reported that chronic pain is associated with socioeconomic variables such as unemployment, and lower levels of income and education. Chronic pain has also been found to be associated with increased psychological distress, particularly depression (e.g. Von Korff et al., 1988; Magni et al., 1990; Croft et al., 1993; Gureje, Von Korff, Simon & Gater, 1998; Blyth et al., 2001; McWilliams, Cox & Enns, 2003), and anxiety (e.g. McWilliams et al., 2003; McWilliams, Goodwin & Cox, 2004; Von Korff, Crane, Lane, Miglioretti, Simon, Saunders, Stang, Brandenburg & Kessler, 2005).

The evidence also suggests that chronic pain is associated with significant utilisation of health care resources (Magni et al., 1990; Von Korff, Wagner, Dworkin & Saunders, 1991; Von Korff, Ormel, Keefe & Dworkin, 1992; Buskila et al., 2000; Blyth, March & Cousins, 2003a; Blyth, March, Brnabic & Cousins, 2004). For example, Blyth et al. (2004) reported that chronic pain with a high level of disability was associated with a 2-fold increase in general practice visits over the previous 12 months, and with a 5-fold increase in the numbers of emergency department visits over the previous 12 months, compared with no chronic pain, even after adjusting for known predictors of health care use such as age, gender, and comorbid medical conditions. Similarly, in a large Danish population survey, Eriksen et al. (2003) reported that participants with chronic pain reported twice as many contacts with health professionals than participants who did not report pain.

Finally, it has been well-established that chronic pain is often associated with significant levels of disability (Linton, 1987). For example, in the Australian chronic pain prevalence study already mentioned, Blyth et al. (2001) reported that 13.5% of females and 11% of males with chronic pain reported some degree of interference in daily activities due to pain. Von Korff, Dworkin and Le Resche (1990) reported that in a sample of over 1000 adults enrolled in a health service, 2.7% reported pain accompanied by seven or more days in the previous six months in which their activities had been limited by pain.

Similarly, chronic pain is associated with high rates of lost work productivity (Frymoyer, Pope, Clements, Wilder, MacPherson & Ashikaga, 1983; Magni et al., 1990). Blyth, March, Nicholas and Cousins (2003b) reported that while most of their participants with chronic pain reported that they were working full-time or part-time, when both lost work days and reduced-effectiveness work days were combined, an average of 16.4 lost work day equivalents occurred in a 6-month period. Magni et al. (1990) reported that, in a randomly selected sample representative of the US population, 8.5% of the subjects who reported chronic pain had lost more than 30 days of work due to pain in the previous 12 months, and 23.1% had changed jobs completely because of their pain. Similarly, Eriksen et al. (2003) reported that the odds of having changed jobs for health reasons were seven times higher among participants with chronic pain than among those not reporting pain.

These figures highlight the fact that although some individuals report being significantly disabled by their pain, others are able to maintain employment and other activities despite pain. Von Korff et al.'s (1990) population-based prevalence study illustrated that while 45% of respondents reported chronic pain, only 2.7% reported that their pain was associated with significant disability. Similarly, Elliott et al. (1999)

reported a prevalence of chronic pain of 46.5% in a randomly selected sample of general practice patients, but noted that only 15.8% reported being significantly disabled by their pain. Magni et al. (1990) pointed out that while 16% of subjects with chronic pain had lost some time from work due to pain, over 80% had not lost a single day.

However, it has been noted that the small proportion of individuals with chronic pain who are significantly disabled by their pain contribute disproportionately to the societal and economic costs associated with chronic pain (Spitzer, LeBlanc, Dupuis & al, 1987; Frymoyer & Cats-Baril, 1991; Dionne, 1999). For example, in the case of low back pain, approximately 75% of the costs of compensation payments and absence from work have been attributed to the 5-7.5% who become temporarily or permanently disabled by low back pain (Spitzer et al., 1987; Frymoyer & Cats-Baril, 1991).

It is worth noting that these costs are not insignificant. Costs include direct costs, such as health care expenditure, and indirect costs such as unemployment, loss of productivity, lost tax revenue, and disability compensation (Kerns, 1994; Turk & Melzack, 2001). A recent report by ACCESS Economics estimated the total cost of chronic pain in Australia in 2007 at AU$34.3 billion. Turk and Okifuji (1998) estimated the annual direct and indirect costs of chronic pain in the United States as being in excess of US$375 billion. Similarly enormous estimates of the costs of chronic pain have been reported for countries such as the Netherlands (van Tulder, Koes & Bouter, 1995) and Canada (Spitzer et al., 1987; Statistics Canada, 1999).

In summary, epidemiological studies have shown that chronic pain is a common and costly problem. Chronic pain is often associated with high levels of disability and psychological distress; however, it is also apparent that not all individuals with chronic pain become significantly disabled by their condition. Attempts to understand this

variability have ranged from attributing it entirely to physical factors to more recent approaches which take a more multidimensional perspective.

1.2. Conceptualisations of pain

The biomedical model, or unidimensional sensory model, of pain is based on the Cartesian concept that the experience of pain is a purely sensory event (Craig, 1994). The biomedical model proposes a linear relationship between tissue damage or pathology and the experience of pain (Haldeman, 1990). According to this perspective, pain can be understood and treated by identifying underlying pathology through physical examination or diagnostic testing, and variations in clinical presentation can be accounted for by differences in pathology or severity of pathology (Waddell, 1987). In other words, the biomedical model posits that the varying responses to pain (including variations in disability) can be accounted for by differences in physical pathology. The biomedical model considers functional impairment or emotional disturbance as being reactions to the disease, and proposes that these secondary factors will automatically be alleviated once the underlying pathology has been detected and treated (Craig, 1994; Turk & Monarch, 2002).

Although this unidimensional model has dominated theories of pain for centuries (Turk, 2001), there is now considerable evidence that there is no direct link between physical pathology and pain, particularly in the case of chronic pain (Turk & Monarch, 2002; Keefe, Abernethy & Campbell, 2005).

In the area of chronic back pain, studies investigating the relationship between pain and physical pathology identified by radiological investigations have indicated that physical findings are not reliable indicators of back pain. For example, among participants reporting back pain, spinal pathology identified by magnetic resonance

imaging (MRI) is often not significantly associated with reports of pain (e.g. Beattie, Meyers, Stratford, Millard & Hollenberg, 2000; Geisser, Haig, Tong, Yamakawa, Quint, Hoff, Miner & Phalke, 2007). Furthermore, a number of studies (e.g. Wiesel, Tsourmas, Feffer, Citrin & Patronas, 1984; Boden, Davis, Dina, Patronas & Wiesel, 1990; Jensen, Brant-Zawadzki, Obuchowski, Modic, Malkasian & Ross, 1994a) have discovered that substantial abnormalities on CT or MRI scans of the spine are frequently seen in individuals with no history of back pain. Other investigators have reported that there is little association between changes seen on MRI over a number of years and the development of low back pain (e.g. Elfering, Semmer, Birkhofer, Zanetti, Hodler & Boos, 2002; Carragee, Alamin, Cheng, Franklin, van den Haak & Hurwitz, 2006). In addition, a large proportion of back pain patients are not found to have any spinal pathology on investigation, and most cannot be given a clear diagnosis (Deyo, 1986b; Spitzer et al., 1987; Carragee & Hannibal, 2004).

Similarly, the evidence for a direct relationship between physical pathology and disability is also limited (Waddell, 1987; Hunter, 2001). For example, Waddell and Main (1984) reported that in a sample of low back pain patients less than half of the variance in disability was accounted for by physical factors. Cairns, Mooney and Crane (1984) found that tissue pathology was not predictive of changes in activity and return to work following multidisciplinary treatment for chronic low back pain. In two separate studies of rheumatoid arthritis patients, Hagglund, Haley, Reveille and Alarcon (1989) and Flor and Turk (1988) found that measures of disease activity and severity were not significantly related to pain intensity or measures of functional impairment. Similarly, Rudy, Turk, Zaki and Curtin (1989) reported that physical abnormalities were not useful for identifying differences in distress or dysfunction between temporomandibular joint pain patients. More recently, in a prospective study investigating predictors of low back

pain disability, Carragee, Alamin, Miller, Carragee (2005) reported that pathology detected by MRI and discography were only weakly associated with back pain episodes and had no association with pain-related disability or number of medical visits. In another recent longitudinal study, Haig, Tong, Yamakawa et al. (2006) also found that MRI did not predict future reports of pain or function in individuals with spinal stenosis.

The lack of a direct relationship between physical variables, reports of pain, and functional outcome, and the limited success of somatic treatments aimed solely at treating physical pathology (Fordyce, 1980) has led to increasing recognition that variations in physical factors are insufficient to account for individual differences in response to pain. The Gate Control Theory of pain proposed by Melzack and Wall (1965) signified a major shift away from the unidimensional view that pain was a purely physical phenomenon (Turk, 1996a). Melzack and colleagues (Melzack & Wall, 1965; Melzack & Casey, 1968) considered pain to be a multidimensional experience, involving the integration of cognitive-evaluative, affective-motivational, and sensory-discriminative dimensions. By postulating that pain perception could be modulated by both peripheral and central nervous system processes, Melzack and colleagues highlighted the role of psychological factors, such as mood and motivation (Turk, 2001). While proponents of a unidimensional perspective of pain had relegated psychological factors to secondary importance, the Gate Control Theory led to psychological variables being incorporated into pain research by emphasising that they were an integral aspect of the pain experience (Melzack, 1991).

In recent decades, a biopsychosocial perspective of pain has gained increasing acceptance (Turk & Okifuji, 2002). The biopsychosocial model postulates that complex and reciprocal relationships between biological, psychological, sociocultural, and environmental variables determine an individual's response to pain, thus accounting for

individual variations in adjustment to chronic pain (Turk & Flor, 1999). The advent of the Gate Control Theory of pain and the growth of a biopsychosocial approach to chronic pain has contributed to the development of numerous psychological models of chronic pain. The following sections will provide an overview of the most prominent of these models, before highlighting a number of the key issues that will be addressed later in this thesis.

1.3. Psychological models of chronic pain

1.3.1. Psychodynamic models

The psychodynamic view of pain was an early alternative to the biomedical model. The psychodynamic approach does not provide an integrated theory of pain (Grzesiak, Ury & Dworkin, 1996), and instead consists of different theoretical models proposed by a number of authors, such as Szasz (1957) and Engel (1959). The main tenet of psychodynamic theory is that pain is a manifestation of an unconscious psychic conflict due to, for example, repressed hostility or aggression, guilt, or early childhood trauma (Grzesiak et al., 1996). Psychodynamic theories are characterised by the concept of "psychogenic" pain (Engel, 1959), which essentially adheres to a dualistic notion that pain is either organic or psychological in nature (Flor, Birbaumer & Turk, 1990; Novy, Nelson, Francis & Turk, 1995). According to this perspective, psychogenic pain accounts for pain being experienced without corresponding physical pathology (Gamsa, 1994b). Psychodynamic theorists have also argued in favour of a "pain-prone personality"; that is, the view that some individuals are predisposed to developing persistent pain conditions (e.g. Engel, 1959; Blumer & Heilbronn, 1982).

It has frequently been concluded that the empirical foundations for the concepts of psychogenic pain and the pain-prone personality are limited (e.g. Turk & Salovey, 1984;

Roy, 1985; Benjamin, Barnes, Berger, Clarke & Jeacock, 1988; Merskey, 1989; Gamsa, 1994bb; Nielson, 2001). As these authors have argued, the research in this area is characterised by poor methodology, including a reliance on anecdotal accounts, small sample sizes, and a lack of adequate control groups. In addition, studies have not supported the dualistic distinction between pain of psychological or somatic origin (Gamsa, 1994a; Novy, Nelson, Francis & Turk, 1995), and supporters of this perspective have often been criticised for not considering alternative explanations for the results of their studies (e.g. Turk & Salovey, 1984; Gamsa, 1994a). Despite this, it is important to note that psychodynamic thought played an important role in the growth of psychological approaches to pain by drawing attention to psychological issues during a period in which the biomedical model predominated (Gamsa, 1994b).

1.3.2. Behavioural models of pain

Behavioural models of pain emerged in the 1970s and were derived from the work of behavioural theorists in other areas of psychological research (e.g. Skinner, 1953). Behavioural theory focuses on observable behaviour, and is based on the principle that environmental events are the key determinants of human behaviour (Schwartz, 1989).

The operant model

The operant model of pain is concerned with observable "pain behaviours" as opposed to the private experience of pain (Turk & Nash, 1996). Pain behaviours include verbal responses (e.g. complaints of pain, moaning, sighing), nonverbal behaviours (e.g. limping, grimacing, or rubbing), consumption of medication, and time spent resting (Sanders, 2002). According to operant theory, pain behaviours are subject to the same influences as other behaviours; that is, they are sensitive to the effects of reinforcement contingencies (Fordyce, 1976). Fordyce, who pioneered the operant approach to chronic

pain, proposed that initially all pain behaviours could be classified as “respondent”; that is, they are reflexive actions in response to acute injury (e.g. limping in response to an ankle strain). However, he argued that over time the same behaviours become increasingly influenced by environmental factors. Consequently, pain behaviours eventually occur as a function of reinforcement contingencies, even after the original cause of the pain has been resolved.

More specifically, Fordyce (1976) identified three ways in which pain behaviours can be maintained or reinforced. Firstly, pain behaviour can be directly reinforced if it is followed by an outcome the individual considers positive or rewarding (positive reinforcement); for example, when a verbal expression of pain is followed by increased attention from a spouse. Secondly, pain behaviour can be reinforced indirectly if it leads to effective avoidance of an aversive consequence (avoidance learning); for example, when pain behaviour such as limping minimises pain, or allows avoidance of an unpleasant work situation. Finally, pain behaviour can be maintained if “well behaviour” (Fordyce, 1976, p. 69), such as attempting to be active despite pain, is punished (e.g. if a doctor criticises the individual for attempting a task), or is not sufficiently reinforced.

An influential contribution of the operant model has been the identification of the role of avoidance learning, particularly in the development of chronic musculoskeletal pain. Fordyce (1976) suggested that individuals learn to avoid activities which they have experienced as causing pain, and that the non-occurrence of pain reinforces the avoidance behaviour. It has been noted that avoidance of a wide array of normal activities (e.g. work, socialising, recreational interests) is very common among chronic pain patients (Philips, 1987). Avoidance of activities or situations in order to prevent pain is considered to be detrimental to recovery from injury as it leads to a vicious cycle in which physical deconditioning due to inactivity (or the "disuse syndrome"; Bortz, 1984) increases the

likelihood of pain when activity is attempted, leading to further avoidance and increasing levels of disability (Lethem, Slade, Troup & Bentley, 1983; Vlaeyen, Kole-Snijders, Boeren & van Eek, 1995a). Contemporary models of the development of disability in chronic musculoskeletal pain following physical injury assign avoidance a central role, particularly when avoidance of activity is due to fear of pain or fear of causing further damage (Lethem et al., 1983; Vlaeyen et al., 1995a). These "fear-avoidance" models, as they are often referred to, will be discussed in further detail in Chapter 2.

Despite the contributions of operant theory, some of the tenets of the operant model have been criticised (e.g. Turk & Flor, 1987; Turk, 1996b). In particular, the validity of the pain behaviour construct has been questioned (Turk & Flor, 1987; Turk, 1996b), and several authors have argued that the model does not take into account important physical, emotional, or cognitive influences on pain behaviour (Schmidt, 1985; Schmidt, Gierlings & Peters, 1989; Turk & Okifuji, 1997; Sharp, 2001). Studies purportedly providing support for the operant model (e.g. Cairns & Pasino, 1977; Block, Kremer & Gaylor, 1980) have also been criticised on methodological grounds (e.g. Paulsen & Altmaier, 1995; Jolliffe & Nicholas, 2004), and researchers have recommended that broader influences on pain behaviour (e.g. sociocultural factors) also be investigated (Blyth et al., 2001; Sanders, 2002). The effectiveness of operant treatments in improving functional status in chronic pain (Morley, Eccleston & Williams, 1999; van Tulder, Ostelo, Vlaeyen, Linton, Morley & Assendelft, 2000; 2001), has sometimes been cited as support for the operant model (e.g. Turk & Flor, 1984), although it has also been noted that evidence supporting the effectiveness of operant treatments does not prove that treatment change is the result of the manipulation of reinforcement contingencies (Linton & Gotestam, 1985; Sharp, 2001).

These concerns aside, overall the research does seem to indicate that operant conditioning factors can play a role in the development and maintenance of pain behaviours. For example, in an early laboratory study, Linton and Gotestam (1985) demonstrated that the pain reports of healthy individuals exposed to a noxious stimulus could be operantly conditioned to increase or decrease through verbal reinforcement even when the intensity of the painful stimulus was kept constant. They also reported that reports of increased pain could be elicited and maintained by verbal reinforcement even when the intensity of the stimulus decreased. In a similar paradigm with a larger sample and experimental design improvements, Jolliffe and Nicholas (2004) also found that the pain reports of university students could be influenced by reinforcement contingencies independent of the level of the aversive stimulus. Finally, Flor, Knost and Birbaumer (2002) compared a sample of chronic pain patients with a group of matched healthy controls and also found that the pain reports of both groups following an aversive stimulus (a brief electric shock) could be influenced by positive reinforcement. Importantly, Flor et al. also reported that the chronic pain group required more trials compared with the control group for their previously reinforced pain reports to be extinguished. Flor et al. argued that this result indicated that chronic pain patients are more influenced by operant conditioning and that this could play a role in the maintenance of their chronic pain problems. Other studies have demonstrated that the pain behaviours of chronic pain patients can also be reinforced by the responses of spouses (e.g. Flor, Kerns & Turk, 1987; Turk, Kerns & Rosenberg, 1992), providing support for the operant model outside of the laboratory-induced pain paradigms described above.

Respondent conditioning models

Respondent conditioning models of pain also focus on the learning of behaviour. Unlike operant theories, however, respondent models are based on the principles of classical conditioning; the theory that environmental stimuli previously not related to a response can come to trigger the response through the process of association (Schwartz, 1989).

The central argument of this approach to chronic pain is that classical conditioning of pain and physical tension may occur during the acute pain phase (Gentry & Bernal, 1977; Linton, Melin & Gotestam, 1984). As Linton et al. (1984) explain, the pain experienced during the acute phase of an injury is the unconditioned stimulus (UCS). The UCS automatically leads to the unconditioned response (UCR) of sympathetic activation, including muscle tension and anxiety. With repeated pairings of the UCS and external stimuli (e.g. activities or situations), the external stimuli become conditioned stimuli (CS) and may elicit the conditioned response (CR) of sympathetic activation and anxiety independently of the UCS. According to Linton et al., this conditioning process can contribute to ongoing pain being experienced, depending on the degree and duration of muscle tension, and the individual's vulnerability.

Fordyce, Shelton and Dundore (1982) also noted that classical conditioning can maintain avoidance behaviour. They hypothesised that if pain is repetitively paired with other stimuli (both internal and external cues) these stimuli may come to elicit avoidance behaviours such as guarding or limping because the individual associates these cues with the onset of pain. Through the phenomenon of stimulus generalisation the individual may learn to associate a range of activities with pain, leading to greater avoidance of activity and increasing disability (Flor, Birbaumer & Turk, 1990; Turk, 2001). The expectation of

pain and the fear of activities which is said to develop through classical conditioning is thought to lead to avoidance of these activities even after the original injury has healed (Lethem et al., 1983; Flor et al., 1990). In this way, the initial respondent conditioning process may be followed by operant learning in which avoidance of the conditioned stimuli is negatively reinforced when the experience of pain (and the associated anxiety) is avoided (Turk & Flor, 1999; Turk, 2001). Consistent with this, a number of authors (e.g. Flor et al., 1990; Sanders, 2002) have suggested that it is likely that operant and respondent conditioning processes interact to maintain avoidance in chronic pain states.

There is some empirical support for the role of classical conditioning mechanisms in chronic pain, mainly in the form of correlations between elevated state anxiety and reduced pain tolerance or spinal immobility (Pope, Rosen, Wilder & Frymoyer, 1980; Dolce, Crocker, Moletteire & Doleys, 1986; Flor et al., 1990). There is also evidence that at least some groups of chronic pain patients exhibit symptom-specific psychophysiological responses (e.g. elevated muscular reactivity at the site of injury) in response to stress (Flor & Turk, 1989). However, there is no support for an association between increased muscle tension and increased pain severity (Vlaeyen & Morley, 2005).

As is the case with the operant model of pain, respondent conditioning paradigms have also highlighted the potential role of fear and anxiety in chronic pain. In addition, by suggesting that individuals may anticipate or expect pain in certain situations, operant and respondent models also paved the way for the role of cognitive variables in chronic pain to be considered.

1.3.3. Cognitive-behavioural models

The shift towards incorporating cognitive constructs into models of chronic pain (e.g. Turk, Meichenbaum & Genest, 1983) emerged from similar developments in

theoretical and treatment approaches to depression and anxiety (Beck, 1976; Meichenbaum, 1977; Beck, Rush, Shaw & Emery, 1979). The cognitive-behavioural perspective on chronic pain emphasises the pivotal role of cognitive factors, while integrating the principles of operant and respondent conditioning from earlier approaches (Turk, 2002b).

According to the cognitive-behavioural model, individuals do not simply respond passively to reinforcement contingencies, but instead actively process and interpret their environment based on knowledge from prior experience (Turk, 2002b). Consequently, it is the individual's perception or interpretation of their situation that interacts with emotional, sensory, and behavioural factors to determine the response to pain (Turk & Rudy, 1986). Some of the cognitive variables that have been ascribed a role in this interpretative process include beliefs and attitudes, attributions and expectancies, attentional processes, coping self-statements, and images (Turk et al., 1983). For example, if an individual believes that the pain is indicative of having exacerbated an injury, or is a sign of undiagnosed cancer, they are more likely to experience the pain as distressing in comparison to an individual who views their pain as a minor injury that is likely to resolve (Turk & Rudy, 1992). The cognitive-behavioural model has been supported by a growing body of literature revealing significant relationships between a range of cognitive variables and adjustment to chronic pain.

Firstly, studies investigating negative thoughts experienced in response to pain (e.g. "I am useless", "I am going to become an invalid") have found that these kinds of responses are associated with reports of higher levels of pain severity and psychological distress (e.g. Gil, Williams, Keefe & Beckham, 1990). Stroud, Thorn, Jensen and Boothby (2000) reported that negative thoughts in response to pain accounted for a significant amount of the variance in general activity, interference in activities due to

pain, and affective distress, even after controlling for demographic variables, employment status, and pain severity. Similarly, specific types of maladaptive thoughts, often referred to as cognitive errors (Beck et al., 1979), have been found to be positively correlated with psychological distress in chronic low back pain patients (Smith, Aberger, Follick & Ahern, 1986). In another sample of chronic low back pain patients, when the contributions of pain severity, number of treatments for pain, and depression were controlled for, cognitive errors were also found to account for a significant amount of the variance in physical disability (Smith, Follick, Ahern & Adams, 1986).

The importance of specific beliefs about pain has also been examined. The belief that pain is indicative of harm, and that movement or activity could cause further damage has consistently been shown to be a better predictor of disability when compared with variables such as pain intensity and duration, or biomedical findings (e.g. Waddell, Newton, Henderson, Somerville & Main, 1993; Vlaeyen et al., 1995; Crombez, Vlaeyen, Heuts & Lysens, 1999). The belief that one is disabled by pain has also been reported to be strongly associated with higher levels of pain behaviours and physical disability (Jensen, Turner, Romano & Lawler, 1994; Jensen, Romano, Turner, Good & Wald, 1999). Believing that pain is mysterious or poorly understood, and believing in the permanence or constancy of pain (Williams & Thorn, 1989), is significantly associated with depression, less frequent use of coping strategies, and a lower likelihood of rating coping strategies as effective (Williams & Keefe, 1991; Williams, Robinson & Geisser, 1994). The belief that pain is permanent and constant is also positively correlated with higher anxiety and pain intensity (Herda, Siergeris & Basler, 1994), and is associated with lower levels of physical functioning (Williams, Robinson & Geisser, 1994). Consistent with the results of these studies, in a heterogeneous group of chronic pain patients, Turner, Jensen and Romano (2000) reported that beliefs about the permanency

of pain, and beliefs about whether one is disabled and whether medications and solicitous responses from others are appropriate, made statistically significant contributions to the prediction of physical disability after adjusting for age, sex, pain intensity, and other cognitive variables.

Studies have also indicated that the individual's perceptions of control over the pain are also important. For example, perceptions of helplessness or poor control over pain are significantly associated with depression, anxiety, and higher levels of pain and disability (e.g. Flor & Turk, 1988; Spinhoven, Ter Kuile, Linssen & Gazendam, 1989; Strong, Ashton, Cramond & Chant, 1990; Jacob, Kerns, Rosenberg & Haythornthwaite, 1993). Perceptions of poor control over pain predict a significant amount of the variance in psychological distress (Keefe, Crisson, Urban & Williams, 1990), and the relationship between pain and depression seems to be mediated at least partly by perceptions of lack of control over pain and life in general (Rudy, Kerns & Turk, 1988). In a recent study Samwel, Evers, Crul and Kraaimaat (2006) reported that perceptions of helplessness were the strongest predictor of pain intensity, and were a significant predictor of pain-related disability in a sample of chronic pain patients being treated at an interdisciplinary pain centre. Similarly, studies have reported that individuals who tend to believe that important outcomes are under their own control (i.e. internal locus of control; Rotter, 1966) report less pain (Toomey, Mann, Abashian & Thompson-Pope, 1991), and lower levels of pain-related distress and depressive symptoms (Skevington, 1983; Crisson & Keefe, 1988), when compared with individuals who exhibit an external locus of control (i.e. the tendency to attribute important outcomes to chance, luck, or the behaviour of others). External locus of control has also been linked to the use of maladaptive strategies to cope with pain (Crisson & Keefe, 1988; Harkapaa, 1991).

Related to perceptions of control is the concept of self-efficacy, that is, an individual's conviction that he or she is able to successfully perform a desired behaviour (Bandura, 1977). Studies investigating self-efficacy in chronic pain have revealed that low self-efficacy ratings (regarding ability to perform specific activities or cope with pain) are related to poor physical performance (Dolce et al., 1986; Council, Ahern, Follick & Kline, 1988; Rudy, Lieber, Boston, Gourley & Baysal, 2003), and depression (Anderson, Dowds, Pelletz, Edwards & Peeters-Asdourian, 1995). A number of studies have also suggested that self-efficacy may be a better predictor of disability in different groups of chronic pain patients (e.g. chronic low back pain, fibromyalgia) than biomedical variables, demographic variables, pain intensity, and fear-avoidance beliefs (Buckelew, Parker, Keefe, Deuser, Crews, Conway, Kay & Hewett, 1994; Lackner, Carosella & Feuerstein, 1996; Asghari & Nicholas, 2001; Ayre & Tyson, 2001; Denison, Asenlof & Lindberg, 2004). In fact, in a recent review of the literature on the role of psychological factors in chronic pain, Keefe, Rumble, Scipio, Giordano and Perri (2004) concluded that there is strong support for the importance of self-efficacy and that the consistency of results across populations is impressive.

Catastrophising is another cognitive construct that has received considerable attention in chronic pain research (DeGood & Tait, 2001). Catastrophising has been defined as "an exaggerated negative orientation toward pain stimuli and pain experience" (Sullivan, Stanish, Waite, Sullivan & Tripp, 1998, p. 253). Although there has been some theoretical debate about whether catastrophising is a coping strategy or an appraisal (e.g. Jensen, Turner, Romano & Karoly, 1991; Keefe, Kashikar-Zuck, Robinson, Salley, Beuapre, Caldwell, Baucom & Haythornthwaite, 1997; Sullivan, Thorn, Haythornthwaite, Keefe, Martin, Bradley & Lefebvre, 2001; Turner & Aaron, 2001), research has consistently indicated that it is associated with poor adjustment to chronic pain in several

chronic pain populations, including back pain (e.g. Flor & Turk, 1988), arthritis (e.g. Keefe, Brown, Wallston & Caldwell, 1989), and neuropathic pain (e.g. Sullivan, Lynch & Clark, 2005). In particular, catastrophising has been found to be associated with reports of higher pain intensity (e.g. Keefe et al., 1989; Harkapaa, 1991), greater disability (e.g. Martin, Bradley, Alexander, Alarcon, Triana-Alexander, Aaron & Alberts, 1996; Robinson, Riley, Myers, Sadler, Kvaal, Geisser & Keefe, 1997; Sullivan et al., 1998), and higher levels of psychological distress (e.g. Jensen, Turner & Romano, 1992; Geisser, Robinson & Henson, 1994; Geisser, Robinson, Keefe & Weiner, 1994; Turner, Dworkin, Mancl, Huggins & Truelove, 2001). Catastrophising has also been shown to be an important predictor of disability and distress even when demographic variables or pain intensity are taken into account (Severeijns, Vlaeyen, van den Hout & Weber, 2001; Turner, Jensen, Warms & Cardenas, 2002).

Although the empirical literature concerning the cognitive-behavioural model reflects an emphasis on investigating the role of cognitive factors in chronic pain, proponents of the model have stressed that it is not a linear, causal model; instead, it posits complex and reciprocal interrelationships amongst factors (Turk & Rudy, 1986). In other words, it is the interaction between cognitions, physiology, affect, and behaviour that contributes to the maintenance of distress and disability over time, and that accounts for individual variations in adjustment to chronic pain. However, as Novy and colleagues (1995) have pointed out, the research is dominated by correlational studies that do not elucidate causal relationships, and further research is required to determine if all factors are interrelated in the manner proposed by the cognitive-behavioural model. Notwithstanding the need to explicate causal relationships amongst cognitive and other variables, the cognitive-behavioural model is arguably the most widely accepted psychological model of chronic pain (Turk, 2002b).

1.4. Treatment

1.4.1. Behavioural and cognitive-behavioural treatments for chronic pain

Both behavioural and cognitive-behavioural models of chronic pain have led to the development of effective psychological treatments for chronic pain patients (Morley et al., 1999; van Tulder et al., 2000; Guzman, Esmail, Karjalainen, Malmivaara, Irvin & Bombardier, 2001; van Tulder et al., 2001).

Behavioural treatment approaches based on the operant model aim to achieve measurable changes in pain behaviour by modifying reinforcement contingencies operating in the individual's environment, as opposed to targeting the pain itself (Fordyce, Fowler & Delateur, 1968). Consequently, operant treatment programs are characterised by a focus on decreasing pain behaviours (e.g. excessive resting) by ignoring such behaviour, and positively reinforcing well behaviours (e.g. exercise) through attention, positive feedback, and praise (Fordyce et al., 1968; Fordyce, 1976). Typically, operant treatments incorporate an exercise program in which patients are encouraged to increase their performance of set exercises through gradually increasing quotas, and aim to reduce consumption of medication by shifting patients from pain-contingent to time-contingent schedules (Fordyce, 1976). Spouses or other family members are usually involved in treatment since they are considered an important reinforcer of pain behaviour.

Similarly to operant programs, cognitive-behavioural treatments do not attempt to eliminate pain, and instead focus on assisting patients to improve their ability to cope with and manage their pain (Turk et al., 1983; Holzman, Turk & Kerns, 1986). As these authors explain, a central goal of cognitive-behaviour therapy is to help the patient reconceptualise their pain condition as a problem that they can manage. Instruction in

coping strategies, such as relaxation techniques or methods of distraction, is aimed at increasing the patient's perceptions of having control over the pain experience. In addition, cognitive-behaviour therapy focuses on teaching patients to identify relationships between cognitive, affective, behavioural, and physiological responses to pain, in order to help them develop more adaptive ways of responding to pain. Elements of behaviour therapy (e.g. strategies to achieve behaviour change) are usually included in cognitive-behavioural treatments, although these "behavioural experiments" are conceptualised as providing patients with an opportunity to evaluate or test their beliefs about their pain and their ability to cope (Turk & Okifuji, 1999). One of the basic assumptions of this treatment approach is that modifying a patient's beliefs about their pain will lead to changes in functioning, distress, and the experience of pain (Turk, 2002b). This assumption has been supported by numerous studies showing that cognitive variables do change with cognitive-behavioural treatment, and that these cognitive changes are either predictive of, or mediate, changes in disability and distress (e.g. Jensen, Turner & Romano, 1994; McCracken & Gross, 1998; Jensen et al., 1999; Burns, Kubilus, Bruehl, Harden & Lofland, 2003; Spinhoven, Ter Kuile, Kole-Snijders, Hutten Mansfeld, Den Ouden & Vlaeyen, 2004; Woby, Watson, Roach & Urmston, 2004).

In practice, features of operant and cognitive-behavioural treatments are frequently combined, and consequently, the terms behaviour therapy and cognitive-behaviour therapy are often used interchangeably (Kole-Snijders, Vlaeyen, Goossens, Rutten-van Moelken, Heuts, van Breukelen & von Eek, 1999). In addition, the main therapeutic technique based on the respondent conditioning model (i.e. training in relaxation strategies to reduce muscle tension) is often included in chronic pain treatment programs, both as a coping strategy and as a way of increasing perceptions of control over pain (Turk & Okifuji, 1999). As a result, behavioural or cognitive-behavioural treatment

programs typically consist of multiple components (van Tulder et al., 2000; 2001), and are often embedded within broader multidisciplinary rehabilitation programs that also address physical and vocational issues (Turk & Okifuji, 1999).

1.4.2. Treatment issues

Several recent reviews support the efficacy of behavioural and cognitive-behavioural treatment approaches (Compas, Haaga, Keefe, Leitenberg & Williams, 1998; Morley et al., 1999; van Tulder et al., 2000; 2001). Morley and colleagues (1999) conducted a systematic review and meta-analysis of randomised controlled trials comparing the effectiveness of cognitive-behaviour therapy and behaviour therapy for chronic pain (excluding headache) with waiting-list and alternative-treatment control conditions. They reported that, compared with waiting-list control conditions, behaviour/cognitive-behaviour therapy produced significantly greater changes in pain-related measures (such as pain intensity) and positive cognitive coping/appraisal, and reduced behavioural expressions of pain (i.e. pain behaviours and activity level). Similarly, in another systematic review, van Tulder and colleagues (2000; 2001) concluded that there was strong evidence that operant and cognitive/cognitive-behavioural treatments result in positive changes in pain intensity and functional status, compared with waiting-list control conditions or no treatment. Consistent with these reviews, Vlaeyen and Morley (2005) observed that these treatments are now widely accepted, and have been adopted in multidisciplinary pain clinics worldwide.

Despite this, a need to improve the effectiveness of cognitive-behavioural treatments for chronic pain has been highlighted repeatedly (e.g. Turk, 1990; Vlaeyen & Morley, 2005). The aforementioned meta-analysis by Morley and colleagues failed to find significant differences between behaviour/cognitive-behaviour therapy and

alternative treatment conditions (e.g. physiotherapy) for variables such as depression, negative appraisals of pain (e.g. catastrophising), and social role functioning. Furthermore, it has been apparent for some time that not all patients benefit from cognitive-behaviour therapy to the same degree, and that a sizable proportion of patients do not complete treatment, or relapse (Turk, 1990; Turk & Rudy, 1991; Nicholas, 1992).

Turk and colleagues (e.g. Turk, 1990; Turk & Okifuji, 2002; Turk, 2005) have argued that one reason for this may be that many chronic pain treatment programs tend to treat all chronic pain patients as a homogeneous group, although in reality they may be quite different. Consequently, Turk (2005) has emphasised the importance of identifying the characteristics of patients who do not tend to benefit from existing treatments, and determining if these individuals are more likely to benefit from different combinations of existing treatment components, or even new treatment approaches developed specifically to meet the needs of sub-groups of patients. Vlaeyen and Morley (2005) have proposed that identifying variables which act as moderators and mediators of treatment outcome would also assist in achieving a better match between patient characteristics and specific treatments (or treatment components).

Consistent with these arguments, a number of investigators (e.g. Dahlstrom, Widmark & Carlsson, 1997; Epker & Gatchel, 2000) have attempted to identify patient sub-groups in samples of fibromyalgia and orofacial pain patients by using responses on the Multidimensional Pain Inventory (MPI; Kerns, Turk & Rudy, 1985) to classify patients according to the profiles derived from Turk and Rudy's (1987; 1988) Multiaxial Assessment of Pain (i.e. "Dysfunctional", "Interpersonally Distressed", "Adaptive Coper"). Studies comparing the responses of these sub-groups to treatment have supported the notion that sub-groups of patients do respond differently to treatment (e.g. Rudy, Turk, Kubinski & Zaki, 1995; Turk, Okifuji, Sinclair & Starz, 1998b).

In addition, researchers have also endeavoured to identify predictors of treatment outcome. Although some authors have concluded that this research has not identified any consistent predictors (e.g. Compas, Haaga, Keefe, Leitenberg & Williams, 1998), others have concluded that variables such as treatment credibility and the individual's readiness to change are potentially important factors (e.g. Kole-Snijders et al., 1999; Biller, Arnstein, Caudill, Federman & Guberman, 2000). Studies have also reported that psychopathology is predictive of poor outcome, and that depressed chronic pain patients are more likely to drop-out of treatment or relapse than non-depressed patients (Painter, Seres & Newman, 1980; Kerns & Haythornthwaite, 1988; Fricton & Olsen, 1996; Kole-Snijders et al., 1999). At least one study (e.g. Turk, Okifuji, Sinclair & Starz, 1998a) has reported that the nature of the onset of the pain condition (i.e. idiopathic versus identifiable onset) is associated with a differential response to treatment; however, relatively few researchers have investigated the role of this variable.

Taken together, these converging lines of evidence indicate that sub-groups of chronic pain patients may respond differently to treatment, and that variables such as the nature of the onset of the pain and psychopathology might be important predictors of treatment outcome. In recent years, a growing number of studies have focused on a sub-group of chronic pain patients for whom these specific issues are particularly relevant. Individuals who experience a sudden onset of pain following an injury or accident, particularly when the instigating event is experienced as psychologically traumatic, may present for treatment with high levels of distress, including symptoms consistent with a posttraumatic stress response. The impact of the nature of the onset of pain and posttraumatic stress symptoms on adjustment to chronic pain and treatment outcome is the focus of this thesis.

1.5. Thesis overview

The literature review presented in the following chapters will critically examine existing research regarding the influence of the nature of the onset of pain and posttraumatic stress symptoms on adjustment to chronic pain and treatment outcome, and will highlight important questions that will be addressed by the studies conducted.

As noted above, although a number of studies have investigated the role of the nature of the onset of pain, these studies are relatively few. Chapter 2 will review this research, and will argue that when comparing different types of onset (e.g. onset of pain following accidents versus a gradual onset of pain) the studies conducted to date have failed to consider potentially important characteristics of these types of events. In particular, the fact that some of the events typically associated with onset of pain are experienced as psychologically traumatic has been overlooked in the existing research.

Consequently, Chapter 3 examines the types of experiences that are considered to be potentially traumatic, and provides an overview of the most common posttraumatic disorder, that is, Posttraumatic Stress Disorder (PTSD).

In Chapter 4, the focus returns to the relevance of these issues for chronic pain patients by critically examining a growing body of literature interested in the link between chronic pain and PTSD. Studies investigating the relationship between the two conditions will be examined critically, and important limitations of the research (e.g. the lack of measures of posttraumatic stress that have been validated for use in chronic pain samples) will be delineated. The theoretical models which have been developed to account for the relationship between chronic pain and PTSD will also be described, and their empirical status will be evaluated.

Finally, Chapter 5 will explain the implications of these different, but related, areas of research for the treatment of chronic pain patients who present with posttraumatic

stress symptoms. Unfortunately, few studies have explored the impact of the nature of the onset of pain, or comorbid pain and PTSD on treatment outcome; therefore, currently unresolved issues regarding treatment will be identified.

Three studies have been conducted to address some of the limitations of the literature as expounded in the literature review, and these are presented in Chapters 6 to 8. Chapter 9 provides a general discussion of the findings of these studies, and outlines a number of recommendations for future research.

2. THE ROLE OF ONSET OF PAIN

2.1. Onset of pain

Both epidemiological and clinical studies indicate that chronic pain develops in a variety of circumstances (Crook, Rideout & Browne, 1984; Crombie, Davies & Macrae, 1998; Blyth et al., 2003a; Nicholas, 2005). These studies show that some individuals report that their pain originally developed following a specific incident, such as a physical injury, surgery, or an illness. Other individuals report that they are unable to identify a specific precipitating event (often referred to as spontaneous onset, e.g. Turk, Okifuji, Starz & Sinclair, 1996). The remaining group of individuals also report that the onset of their pain was not associated with a specific event, and that the pain developed gradually over a period of time (also referred to as an insidious onset, e.g. Turk & Okifuji, 1996). These different types of onset are depicted in Figure 2.1 below.

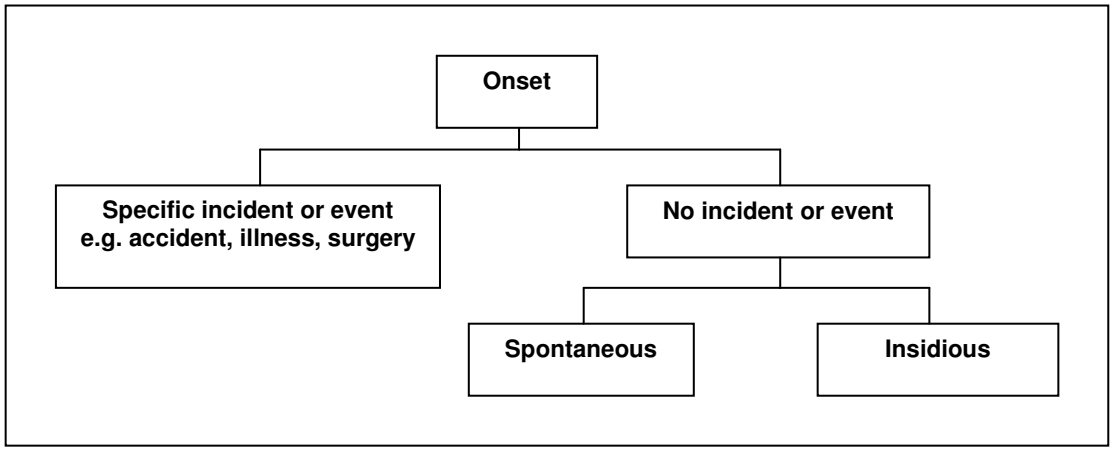

Figure 2.1. Different types of onset of pain reported in epidemiological and clinical studies

The proportion of individuals with chronic pain who report these different types of onset of pain varies depending on the sample being studied (Crombie et al., 1998; Blyth et al., 2003a). The available data suggest that while injuries are a common precipitant of chronic pain in Australia, many individuals with chronic pain also describe a spontaneous or insidious onset of pain. At a population level, data collected in the 2001 National Health Survey indicated that approximately 24% of chronic musculoskeletal and connective tissue disorders were the result of an injury (Australian Bureau of Statistics, 2001a). Blyth et al. (2003a) surveyed over 2000 participants randomly selected from a region of New South Wales and reported that 38% of respondents with chronic pain reported that their pain had started with some type of injury or accident (e.g. a sports injury, work accident, or car accident). Another 19% attributed the pain to a health problem (e.g. an illness or another health problem such as a medical procedure). At the same time, 33% of individuals stated that there was no clear reason for the onset of their pain, or that they did not know what had caused it. A similar picture emerges from Australian clinical data. In a study of a large sample of chronic pain patients presenting to a multidisciplinary pain clinic, Nicholas (2005) reported that over 43% of patients attributed their pain to an accidental injury, and over 15% attributed it to illness or surgery. However, at least 22% could not identify a specific cause for their pain.

The observation that individuals with chronic pain report these different types of onset has led to consideration of the potential impact of this variable on adjustment to chronic pain. This chapter will critically examine the research investigating the role of the onset of pain.

2.2. Onset of pain and adjustment to chronic pain

2.2.1. Onset of pain related to a specific incident

A small number of studies have compared onset of pain related to a specific incident or event with onset of pain unrelated to a specific incident or event. These studies have compared individuals who report that their pain started following some kind of physical trauma (i.e. an injury, accident, illness, or surgery) with individuals who experienced a spontaneous or insidious onset of pain. Most of these studies have been conducted in specific sub-groups of chronic pain patients.

Two studies examining onset of pain have been conducted in samples of fibromyalgia patients. The characteristic features of fibromyalgia are chronic, generalised or widespread pain, and tenderness on palpation at specific body locations (Wolfe, Smythe, Yunus, Bennett, Bombardier, Goldenberg, Tugwell, Campbell, Abeles, Clark & et al., 1990). Although the etiology of fibromyalgia is not well understood (Okifuji & Turk, 1999; Al-Allaf, Dunbar, Hallum, Nosratzadeh, Templeton & Pullar, 2002), fibromyalgia patients often attribute the onset of their symptoms to a precipitating event, such as an injury or illness (White, Speechley, Harth & Ostbye, 1999; Al-Allaf et al., 2002).

Greenfield, Fitzcharles and Esdaile (1992) reviewed the medical files of 127 fibromyalgia patients to obtain information about the onset of their pain. Patients were classified as meeting criteria for what the investigators labelled “reactive” fibromyalgia if they attributed the onset of their problems to a specific event, and were classified as having “primary” fibromyalgia if they could not identify a specific event as the trigger for their symptoms. The two groups were compared on a range of variables, including demographic variables, duration of symptoms, employment status at the time of the study,

loss of employment as a result of the pain, disability benefit status, and physical activity levels before and after the onset of the fibromyalgia symptoms.

Greenfield et al. reported that 23% of their sample met criteria for reactive fibromyalgia, with events such as motor vehicle accidents (MVAs), back injuries, surgery, and illnesses reported as the events associated with the onset of problems in this group. Compared with the primary fibromyalgia group, the reactive fibromyalgia group was more likely to be unemployed due to pain and was more likely to be receiving disability benefits. The physical activity levels of the reactive fibromyalgia group were also more likely to have declined compared with the primary fibromyalgia group. There were no statistically significant differences between the two groups in terms of age, gender, or duration of symptoms.

While these results suggest that onset of pain related to a specific incident is associated with higher levels of disability, there are a number of problems with this study. Firstly, it is difficult to interpret the differences in physical activity levels between the two groups because a standardised measure of physical activity or disability due to pain was not used. Instead, Greenfield et al. coded descriptions of activity levels before and after the onset of pain from the patients' medical file notes as involving either: (1) minimal physical activity beyond personal care; (2) physical activity limited to normal household activities or work; or (3) additional leisure or sporting activity. These categories are relatively broad and are open to interpretation. Despite this, the authors do not comment on the reliability of the ratings made by the different coders. In addition, data regarding the degree of physical pathology identified in the two groups was not collected, so it is possible that the difference in physical activity levels between the two groups was at least partly related to differences in physical pathology. Similarly, the

influence of compensation-related factors cannot be excluded given that 34% of the reactive fibromyalgia group reported receiving disability benefits for pain.

In another fibromyalgia study, Waylonis and Perkins (1994) compared 56 patients diagnosed with fibromyalgia due to a physical trauma (e.g. an injury, accident, or surgery) with over 500 primary fibromyalgia patients who had participated in an earlier study (Waylonis & Heck, 1992). Waylonis and Perkins concluded that the two groups were to a great extent identical, and that there were no notable differences between fibromyalgia associated with trauma and fibromyalgia with a spontaneous or insidious onset. However, as Turk, Okifuji, Starz and Sinclair (1996) have pointed out, Waylonis and Perkins reported significant differences in symptom levels between the two groups on almost half of the symptoms assessed, and significant differences in reported aggravating factors on 60% of the variables assessed. Turk and colleagues argued that despite Waylonis and Perkins' conclusions, the data clearly revealed a number of differences between the two groups.

The results reported by Greenfield et al. (1992) and Waylonis and Perkins (1994) offer some support for the notion that there are differences between patients who develop fibromyalgia following a specific event, and those who experience a spontaneous or insidious onset of symptoms. However, it is important to note that the diagnosis of fibromyalgia has been a source of controversy (e.g. Fitzcharles, 1999; Russell, 1999), and its validity as a distinct clinical entity has often been challenged (e.g. Cohen, 1999; Makela, 1999; Wessely & Hotopf, 1999). In addition, the participants in both studies were almost all female. Consequently, it might not be appropriate to generalise the findings of these two studies to other groups of chronic pain patients.

Another study examining onset of pain in a specific group of chronic pain patients was conducted in a sample of 124 individuals referred to a specialist clinic for work-

related upper extremity disorders (Himmelstein, Feuerstein, Stanek, Koyamatsu, Pransky, Morgan & Anderson, 1995). These disorders are a diverse group of diagnoses involving pain and associated symptoms, such as numbness or stiffness, in the fingers, wrist, arm, shoulder, and neck (Feuerstein, Huang & Pransky, 1999). The patients were categorised as having pain due to "acute trauma" if they attributed the onset of their pain to a specific incident in the workplace. Himmelstein et al. reported that 38% of the sample identified a specific work-related incident as the cause of their injury. Compared with patients who had continued working, patients who were not working due to their pain were more likely to report such an incident.

However, it is worth noting that the authors pointed out that some of the patients categorised as "acute trauma" might also have had other pain conditions unrelated to the specific incident they reported (e.g. repetitive-strain injuries that could be considered to have had an insidious onset). This casts some doubt on the classification of pain onset used in the study, and consequently, the results that were reported. In addition, Himmelstein and colleagues reported that 40% of the patients referred to the clinic were excluded from the study, mainly due to lack of approval of payment for the assessment by the workers' compensation insurer. As a result, the degree to which the final sample was representative of chronic pain patients with this type of disorder is uncertain.

To the best of the author's knowledge, the only study to have compared onset of pain following a specific event to pain with an insidious or spontaneous onset in a heterogeneous sample of chronic pain patients was conducted by Turk and Okifuji (1996). Unlike the study conducted by Greenfield and colleagues (1992), only patients who were not receiving compensation were included in the analyses, so that this variable could not influence the results. Turk and Okifuji based this decision on the results of other comparisons they conducted in this study, which indicated that patients receiving or

seeking compensation reported higher levels of pain, and higher levels of pain-related disability and affective distress than patients who were not involved in a compensation claim. Standardised psychometric measures (the Center of Epidemiological Study-Depression Scale, Radloff, 1977; the Owestry Disability Scale, Fairbank, Couper, Davies & O'Brien, 1980; the Multidimensional Pain Inventory, Kerns et al., 1985) were used to collect data regarding pain severity, perceived interference in life activities, affective distress (including symptoms of depression), and general physical activity. The analyses also included a standardised method of quantifying the extent of physical findings (i.e. the Medical Examination and Diagnostic Information Coding System, MEDICS; Rudy, Turk, Brena, Stieg & Brody, 1990) in order to compare the two groups on this variable.

Thirty-seven out of 63 participants (59%) reported that their pain began with a specific physical trauma, such as a work-related injury, motor-vehicle accident, other type of accident, or surgery. Turk and Okifuji reported that, compared with the insidious onset group, the traumatic onset group reported significantly higher levels of pain, interference in activities due to pain, and affective distress. Importantly, there were no significant differences between the two groups in terms of physical findings. It is also important to note that there were no significant differences between the two groups on levels of depressive symptoms or level of activity. Although the traumatic-onset group reported being just as active as the insidious-onset group, they perceived the interference in their life due to pain as being more extreme than the insidious-onset group.

In a study focused primarily on the role of pain-related fear in chronic back pain disability, Crombez, Vlaeyen, Heuts and Lysens (1999) investigated an earlier finding from a study examining the role of fear of movement/(re)injury in back pain (Vlaeyen, Kole-Snijders, Rotteveel, Ruesink & Heuts, 1995b). Although onset of pain was not the focus of the study, Vlaeyen et al. (1995b) observed that participants who reported a

sudden onset of pain in which they had experienced fear scored significantly higher on a measure of fear of movement/(re)injury than participants who had reported that their pain started gradually. Consequently, Crombez et al. (1999) compared participants who were able to identify a specific date for onset of their pain (labelled the "sudden onset" group) with those who were not able to recollect such a date. It was assumed that the onset of pain in the second group must have been gradual.

Seventeen out of 31 participants (55%) were able to specify a date for the onset of their pain. As Crombez et al. predicted, these individuals reported significantly higher levels of fear of movement/(re)injury when compared with the individuals who could not identify a date of onset. However, contrary to their expectations, the latter group reported higher levels of pain-related disability.

Potential explanations for this unexpected finding were not provided by Crombez and colleagues because onset of pain was not the focus of the study, but may be related to the small sample size, or the method used to categorise subjects into groups. All patients who could not identify a specific date for the onset of their pain were allocated to the other group. This may result in different groups of participants to asking individuals whether they attribute their pain to a specific event, which is the method used in the majority of studies. In particular, participants who had experienced a spontaneous onset of pain (and could thus identify a specific date on which it developed) would have been allocated to the "sudden onset" group in the Crombez et al. study, unlike the other studies in this area.

2.2.2. Onset of pain related to accidents

Another group of studies has examined the impact of onset of pain related to accidents. These studies have either compared accident-related pain to all pain that is not

associated with an accident, or have compared accident-related pain to pain with an insidious or spontaneous onset.

Turk et al. (1996) compared fibromyalgia patients who attributed their symptoms to an accident and patients who reported a gradual or spontaneous onset to their condition. Given that the aim of the study was to investigate onset related to accidents, patients who attributed their pain to other specific events (such as illness or surgery) were excluded from the analysis (unlike the studies reviewed in the previous section). Turk and colleagues used the MEDICS and the same standardised psychometric measures as Turk and Okifuji (1996) to obtain data regarding extent of physical pathology, pain severity, perceived interference in life activities, affective distress (including symptoms of depression), and general physical activity. In addition, Turk et al. collected data concerning compensation status, treatment history, and prescribed medication.

Turk et al. reported that 46 patients from the sample of 152 (30%) attributed the onset of their pain to an accident; another 17% attributed the onset of pain to illness or surgery. The remaining 81 patients (53%) could not identify a specific precipitating event. As would be expected, a greater proportion of the accident-onset group were receiving or seeking compensation, so this variable was treated as a covariate in the analyses comparing the two groups.

Turk et al. reported that the patients in the accident-onset group reported significantly higher levels of pain severity, affective distress, perceived disability and life interference due to pain, and lower levels of activity compared to the individuals who experienced a spontaneous or insidious onset of pain. Importantly, there were no significant differences between the two groups on the extent of physical findings, so the group differences could not be attributed to differences in physical pathology. Despite the comparability of the two groups in terms of physical pathology, a significantly higher

number of patients in the accident-onset group (five times as many) were being prescribed opioid medications compared with the insidious/spontaneous-onset group. Similarly, the accident-onset group was also more likely to have received nerve block, transcutaneous electrical nerve stimulation (TENS), and physical therapy. Interestingly, the two groups did not differ in levels of depressive symptomatology, although Turk et al. noted that the accident-onset group's scores on the psychometric measures were indicative of higher levels of general psychological distress. Unfortunately, as was argued in the previous section, this study also focused on fibromyalgia patients, so it is not clear if these results can be generalised to other chronic pain groups.

In a large investigation of a heterogeneous sample of chronic pain patients presenting to an Australian multidisciplinary pain management centre, Nicholas (2005) compared MVA victims with patients who reported a spontaneous or insidious onset of pain. Twelve percent of the sample of over 4000 participants attributed their pain to a motor-vehicle accident, while 22% reported an insidious or spontaneous onset of pain. Standardised psychometric measures of disability (a modified version of the Roland and Morris Disability Questionnaire; Asghari & Nicholas, 2001), affective distress (the Depression Anxiety Stress Scales; Lovibond & Lovibond, 1995b), catastrophising (Pain-Related Self-Statements; Flor & Turk, 1988), self-efficacy (Pain Self-Efficacy Questionnaire; Nicholas, 1989), and fear-avoidance beliefs (Tampa Scale for Kinesiophobia; Kori, Miller & Todd, 1990) were administered.

Nicholas found that the MVA group reported significantly higher levels of pain, distress, and disability compared with the insidious/spontaneous-onset group. The accident-onset group also scored significantly higher on the self-report measures of catastrophising and fear-avoidance beliefs, and lower on the measure of self-efficacy.

Nicholas also repeated the analyses using a slightly different method of categorising type of onset. For the second group of analyses, patients who attributed the onset of their pain to a work-related accident were included in the accident-onset group, and were compared with the patients who had reported a spontaneous or insidious onset. The results of the second group of analyses revealed the same differences between the two groups on all of the self-report measures. This indicates that the differences detected in the first set of analyses were due to having experienced onset of pain following an accident, and were not unique to motor-vehicle accidents. Thus, this study extends the findings obtained by Turk et al. (1996) to a heterogeneous group of chronic pain patients, and indicates that there may also be differences between individuals experiencing accident-related and insidious/spontaneous onset on a range of cognitive variables.

Unfortunately, Nicholas did not control for the effect of compensation status, despite the fact that a comparison of the MVA victims involved in a compensation claim with the MVA victims who were not involved in such a claim revealed that the compensation group reported higher levels of disability and distress.

Finally, Geisser, Roth, Bachman and Eckert (1996) compared pain associated with an accident with all non accident-related pain. 241 chronic pain patients presenting to a multidisciplinary pain management centre were grouped according to type of onset of pain (accident-related and non accident-related). Standardised measures of pain severity (McGill Pain Questionnaire; Melzack, 1975), affective distress (Brief Symptom Inventory; Derogatis & Melisaratos, 1983), and disability (Pain Disability Index; Tait, Chibnall & Krause, 1990) were administered. Geisser and colleagues reported that 38% of the sample attributed the onset of their pain to an accident of some kind. Initial comparisons indicated that the accident-onset group were younger and were more likely to be involved in litigation or receiving compensation compared with the non-accident

group. As Geisser et al. pointed out, the age difference may be attributable to the fact that older individuals are more likely to develop pain conditions that do not typically develop following accidents (e.g. arthritis). Consequently, age, compensation, and litigation status were treated as covariates in the analyses. Similarly, pain severity was also included as a covariate because the accident-onset group reported significantly higher levels of pain compared with the non-accident group. Geisser et al. reported that the patients who attributed their pain to an accident reported higher levels of disability than the patients who did not attribute their pain to an accident. It is worth noting that Geisser and colleagues also investigated other issues related to onset of pain, and their findings will be discussed in further detail in Chapter 4.

2.2.3. Summary

In summary, the research conducted to date provides some support for the hypothesis that there are differences between chronic pain patients who experience the onset of pain following a specific incident, and those who experience a spontaneous or insidious onset of pain. Specifically, onset of pain following a specific incident appears to be associated with higher levels of pain, disability, interference in activities, and affective distress when compared with an insidious or spontaneous onset of pain. Although a number of the studies reviewed above have involved specific patient groups, particularly fibromyalgia patients, the studies conducted in multidisciplinary pain management centres with heterogeneous samples of chronic pain patients have reported similar findings. Similarly, later studies which have improved upon earlier investigations by employing larger samples and standardised outcome measures, and by controlling for the effects of other variables, have also identified differences between patients reporting different types of pain onset. Despite the results of these studies, it is not clear how the

onset of pain might influence adjustment to chronic pain. This issue will be considered in the following section.

2.3. Potential explanations for the differences observed between onset groups

The studies reviewed in the section above suggest that onset of pain following a specific event is associated with poor adjustment to chronic pain when compared with onset of pain that is spontaneous or insidious. These studies also suggest that accident-related pain is associated with poor adjustment to chronic pain when compared with other types of onset of pain. There are two ways in which the results of these studies can be interpreted. One interpretation is that experiencing the onset of pain following a specific incident, regardless of the exact nature of that incident (i.e. whether it was an accident, illness, or due to surgery), is associated with poor adjustment to chronic pain. An alternative possibility is that it is the experience of developing pain following an accident that is associated with poor adjustment. It is possible that studies which have reported differences between onset related to a specific incident and spontaneous or insidious onset have only identified these differences because the specific-incident group tends to include sizeable numbers of individuals who developed pain following an accident. In other words, the first interpretation highlights the importance of pain developing following any specific incident, while the second proposes that developing pain following an accident is the important variable. The evidence in support of each of these possibilities will be examined in turn.

2.3.1. Onset of pain related to a specific incident

The existing literature has forwarded several possible explanations for the onset of pain related to a specific incident being associated with higher rates of disability than spontaneous or insidious onset of pain. Firstly, as Turk et al. (1996) have noted,

individuals who have sustained an injury, who have been ill, or who have undergone surgery often experience a relatively sudden decrease in physical activity. In the early stages following these events reducing activity may be necessary or appropriate; however, as outlined in Chapter 1, continued avoidance of activity can lead to further pain and contribute to maintenance of disability in the longer-term. In the case of fibromyalgia, it has been argued that a rapid decrease in activity may contribute directly to the development of the fibromyalgia symptoms (Greenfield et al., 1992). Although the argument appears to be that the impact of the decrease in activity is greater when the pain is triggered by a specific event because it is often a rapid and significant change in functioning, to date no studies have directly examined this hypothesis.

Respondent conditioning principles have also been forwarded to account for the higher levels of disability found in individuals who have developed pain following a specific event (Turk & Okifuji, 1996). Unlike pain of insidious or spontaneous onset, when pain develops following an identifiable incident the individual has a particular set of circumstances (i.e. a situation or activity) that may become associated with pain. In other words, an association may develop between the experience of pain and features of the original incident. For example, feelings of distress experienced at the time of the incident may become associated with the situation or activity the individual was engaged in at the time, and the distress may be triggered when the individual confronts the situation or attempts the activity later on. Consequently, the individual may avoid these situations or activities in order to avoid the feelings of distress, contributing to ongoing disability (Turk & Okifuji, 1996).

Although no studies have compared respondent conditioning processes in groups of patients with different types of pain onset, as stated in Chapter 1, there is support for the role of respondent conditioning in chronic pain in general. Indirect support for an

association between respondent conditioning and onset of pain can also be found in the literature on stressful and psychologically traumatic events, which indicates that some individuals do avoid situations or activities reminiscent of the event due to the intense feelings of distress that these reminders can elicit (Wilson, 2004).

Other investigators have hypothesised that onset of pain following a specific incident is associated with increased disability because many of these incidents involve some kind of physical injury or trauma. Turk and Holzman (1986) and Turk (2002a) have argued that when pain is attributed to a physical injury the belief that engaging in activity might exacerbate the pain or cause further damage becomes especially pertinent. Thus, chronic pain attributed to an injury may lead to higher levels of disability than chronic pain attributed to other factors by facilitating the development of fear-avoidance beliefs.

The studies by Crombez and colleagues (1999) and Nicholas (2005) described above provide support for this argument. As already noted, Nicholas reported that patients who associated the onset of their pain with an accident reported significantly higher levels of fear of movement/(re)injury compared with patients who reported an insidious or spontaneous onset of pain. Similarly, Crombez et al. reported that patients who could recall a specific date of onset also reported higher levels of fear-avoidance beliefs than patients who could not recall such a date.

In the same way that fear of causing further harm through movement or activity could be considered to be an understandable concern following a physical injury, it is plausible that similar beliefs about the desirability of avoiding activity in order to promote recovery would also be applicable in the context of pain experienced due to illness or surgery. However, no mention has been made in the literature about whether these fears also play a role in the development of disability when chronic pain develops following these types of events. This is an important issue given that the studies reporting

differences between individuals who attribute their pain to a specific event and those who have experienced a spontaneous or insidious onset of pain have often included individuals with illness- or surgery-related pain in the specific incident group. If fear-avoidance beliefs are more pertinent when pain begins following an injury, as Turk and colleagues (Turk & Holzman, 1986; Turk, 2002a) suggested, individuals who have experienced the onset of pain following injury should report higher levels of fear-avoidance beliefs than individuals who have developed pain following illness or surgery. To date, this specific hypothesis does not appear to have been investigated.

It has also been suggested that sustaining an injury may modify the way in which sensory information is processed, leading to changes in pain perception and sensitivity (Turk et al., 1996; Turk, 2002a). Research indicating that there may be differences in pain tolerance between patients with whiplash injuries and healthy control subjects has been cited in support of this hypothesis. For example, Lee, Giles and Drummond (1993) reported that compared with healthy controls, patients with chronic whiplash reported higher pain intensity during a cold pressor procedure (i.e. pain induced by immersing the participant's hand into a mixture of ice and cold water), and tolerated the test for shorter periods. Several studies have also indicated that compared with healthy control subjects, individuals with chronic whiplash injuries display hypersensitivity (i.e. lower pain detection thresholds) to a variety of sensory stimuli, including induced experimental pain (Koelbaek Johansen, Graven-Nielsen, Schou Olesen & Arendt-Nielsen, 1999), electrical stimulation (Curatolo, Petersen-Felix, Arendt-Nielsen, Giani, Zbinden & Radanov, 2001) and non-noxious stimulation such as pressure or vibration (Moog, Quintner, Hall & Zusman, 2002; Sterling, Treleaven, Edwards & Jull, 2002), even in parts of their body in which they do not usually experience pain.

Further evidence that widespread hypersensitivity to sensory stimuli is associated with having experienced an injury comes from a study comparing chronic whiplash-injured patients and individuals with chronic idiopathic neck pain (or neck pain with an insidious onset). Scott, Jull and Sterling (2005) reported that only the whiplash group exhibited widespread hypersensitivity (i.e. in other areas in addition to the cervical spine) to pressure, heat and cold stimuli. The idiopathic neck pain group only showed hypersensitivity to pressure over the cervical spine. Scott et al. pointed out that the experience of a sudden injury in the whiplash group is one of the main differences between the two conditions and that this may account for the differences in hypersensitivity.

However, other studies cast some doubt on the hypothesis that the experience of an injury leads to changes in processing of sensory information. In a prospective study Kasch, Qerama, Bach and Jensen (2005) exposed whiplash-injured patients and individuals with ankle injuries to a cold pressor procedure one week and one, three, six and twelve months after experiencing the injuries. They found that patients with whiplash injuries who had not recovered at one year exhibited reduced pain tolerance compared with whiplash-injured patients who had recovered at one year and individuals with ankle injuries (who had all recovered). Interestingly, the responses of the recovered whiplash patients and the ankle injury group were not significantly different, indicating that reduced pain tolerance was associated with the chronicity of the pain rather than having experienced an injury.

Consistent with this, as Curatolo, Arendt-Nielsen and Petersen (2004) have pointed out, hypersensitivity to sensory stimuli is not unique to whiplash injuries, and may be a feature of a range of chronic pain conditions. For example, a number of studies have reported similar differences in hypersensitivity between chronic pain patients and healthy

controls in fibromyalgia (e.g. McDermid, Rollman & McCain, 1996; Staud, Vierck, Cannon, Mauderli & Price, 2001), temporomandibular joint pain (Svensson, List & Hector, 2001), osteoarthritis (Bajaj, Bajaj, Graven-Nielsen & Arendt-Nielsen, 2001), and post-mastectomy pain (Gottrup, Andersen, Arendt-Nielsen & Jensen, 2000). Given that some of these pain conditions can have a spontaneous or insidious onset the results of these studies also suggest that changes in processing of sensory stimuli may not be solely the result of injury.

Finally, it is also important to consider that studies investigating these issues are typically cross-sectional in design. Consequently, it is not possible to exclude the possibility that patients exhibited these responses prior to the onset of their pain condition (Curatolo et al., 2004).

Overall, while there is evidence for changes in processing of sensory stimuli amongst groups of chronic pain patients, it is not clear that these changes are associated only with injury-related pain. Alternatively, it is possible that cognitive aspects of the processing of physical sensations, including pain, may change following injury. For example, Turk (2002a) argued that hypervigilance for all bodily sensations may be heightened by the increased anxiety associated with a physical injury, and that following an injury individuals experiencing pain may be more likely to interpret bodily sensations as being abnormal or harmful.

A growing number of studies have investigated potential biases in the cognitive processing of pain-related information by chronic pain patients. Most of these studies have focused on the potential existence of an attentional bias towards pain-related information by employing experimental paradigms such as the modified Stroop (Williams, Mathews & MacLeod, 1996b) or dot-probe tasks (MacLeod, Mathews & Tata, 1986). As other researchers have noted (e.g. Pincus & Morley, 2001; Asmundson, Wright

& Hadjistavropoulos, 2005a) the findings of these studies have tended to be inconsistent, with some studies reporting that chronic pain patients, particularly those who report elevated levels of pain-related fear and anxiety, exhibit selective attention towards some types of pain-related information (e.g. Pearce & Morley, 1989; Snider, Asmundson & Wiese, 2000; Dehghani, Sharpe & Nicholas, 2003), and others failing to detect such a bias (e.g. Pincus, Fraser & Pearce, 1998; Crombez, Hermans & Adriaensen, 2000; Asmundson, Carleton & Ekong, 2005b). Alternatively, rather than exhibiting an attentional bias towards pain-related stimuli there is recent evidence that chronic pain patients may have difficulty shifting attention away from all types of threatening stimuli (Asmundson et al., 2005a). Other investigators have explored the processing of somatic sensations, hypothesizing that some chronic pain patients may selectively attend to all physical sensations. However, the small numbers of studies that have addressed this question have also reported mixed findings (e.g. Peters, Vlaeyen & van Drunen, 2000; Peters, Vlaeyen & Kunnen, 2002). Unfortunately, despite progress in all of these areas of research, none of the studies investigating the processing of pain-related information in chronic pain patients have considered the potential impact of type of onset of pain. Consequently, at this stage, empirical support for Turk's hypothesis that the cognitive processing of physical sensations is modified when pain is specifically associated with injury is limited.

2.3.2. Onset of pain related to accidents

As outlined above, it is possible that studies which have reported differences between individuals who attribute their pain to a specific incident and those who have experienced a spontaneous or insidious onset of pain have only identified these differences because the first group tends to include individuals who developed pain

following an accident. This explanation underscores the importance of accidental injury. A growing body of literature supports the notion that accidental injury due to motor-vehicle accidents, industrial or domestic accidents, and other events leading to unexpected physical injury (e.g. assaults) is associated with a wide range of long-term, adverse physical, psychological, and social outcomes.

For example, numerous studies have found high levels of psychopathology in individuals who have sustained an accidental injury. In an Australian study, O'Donnell, Creamer, Pattison and Atkin (2004) assessed 363 accident victims during their initial hospitalisation using structured clinical interviews and self-report measures. Most participants (74%) had been involved in a motor-vehicle accident. O'Donnell et al. reported that just prior to discharge, 17% of patients reported moderate to severe levels of anxiety, and 15% reported moderate to severe levels of depression. Similar findings have emerged from studies conducted in other countries. In the United States, Richmond and Kauder (2000) reported that 32% of 109 patients hospitalised with serious physical injuries due to motor-vehicle accidents, incidents involving a gun or knife, assaults, and other accidents, reported high levels of psychological distress. In a British study, Mason, Wardrope, Turpin and Rowlands (2002) reported that almost 48% of their sample of 210 male accident and emergency department patients met criteria for a psychiatric disorder at six weeks post-injury according to self-report measures.

Importantly for the issue of impact of onset on adjustment to chronic pain, the research also indicates that these psychological sequelae often persist. In the British study mentioned above, over 43% of participants still met criteria for a psychiatric disorder six months post-injury (Mason et al., 2002). O'Donnell et al. (2004) reported that over 20% of their sample still met the criteria for at least one psychiatric diagnosis at the 12-month follow-up, with 46% of this distressed group being diagnosed with more than one

disorder. In an early study, Malt (1988) assessed 107 accidentally injured adults during their initial hospitalisation, and repeated the assessments over an average follow-up period of 28 months. He reported an incidence of psychiatric disorders of over 22% during the follow-up period, with depressive disorders being the most common diagnosis. Even three years after injury, Malt, Blikra and Høivik (1989) found that almost 20% of their sample of 551 accidentally injured adults continued to report decreased psychological health due to the accident. The long-term consequences for this sample of patients were not purely of a psychological nature. More than 30% reported decreased physical functioning due to ongoing physical complications of their injuries, and 18% reported decreased capacity for work due to the injury. Likewise, in a large study of over 1000 hospitalised trauma patients, Holbrook, Anderson, Sieber, Browner and Hoyt (1999) reported that only 18% obtained scores on a standardised quality of life measure that were above the norm for a healthy population at 12 months post-injury. At the 18-month follow-up 80% of patients obtained quality of life scores below the healthy norm.

Similar outcomes have also been reported in studies investigating the consequences of motor-vehicle accidents. Using clinical interviews and self-report measures in a sample of 100 patients admitted to an intensive care unit after motor-vehicle accidents, Matsuoka, Nishi, Nakajima, Kim, Homma and Otomo (2008) found that over 30% met criteria for a psychiatric disorder (mainly depression) 4-6 weeks after the accident. Mayou, Bryant and Duthie (1993) reported that almost one quarter of 188 motor-vehicle accident victims who had presented to a hospital emergency department reported clinical levels of anxiety and depression one year after the accident. They also noted that ongoing psychological problems were associated with continuing medical, work-related, and financial problems. In two large studies (n > 1000 and 500, respectively) of the long-term consequences of motor-vehicle accidents, Mayou and Bryant (2001; 2002) reported that

one year after the accident over 50% of the patients reported clinically significant medical, psychological, social, or legal problems due to the accident. Furthermore, three years post-accident 21% of participants reported ongoing pain associated with their injuries, and 26% continued to report ongoing psychiatric problems (Mayou & Bryant, 2002).

Although a number of explanations have been forwarded to account for the high proportion of negative outcomes following accidental injury, a full review of this research is beyond the scope of this chapter. However, three potential explanations that are relevant to chronic pain are worth highlighting.

The first involves attributions regarding responsibility and blame by individuals who have experienced stressful life events, such as illness or death of a loved one. Studies investigating these attributions and their consequences have consistently reported poor adjustment to the stressful event among individuals who blame others for their circumstances (Tennen & Affleck, 1990). This relationship has been reported in studies of cancer survivors (e.g. Timko & Janoff-Bulman, 1985), women who have experienced a miscarriage or serious illness of a child (e.g. Tennen, Affleck & Gershman, 1986; Madden, 1988), victims of serious motor-vehicle accidents (Delahanty, Herberman, Craig, Hayward, Fullerton, Ursano & Baum, 1997; Hickling, Blanchard, Buckley & Taylor, 1999), survivors of other serious accidents (Schnyder, Wittmann, Friedrich-Perez, Hepp & Moergeli, 2008), hospitalised burn patients (Lambert, Difede & Contrada, 2004), and patients with a spinal cord injury following an accident (Bulman & Wortman, 1977). One explanation for these findings is that blaming others restricts the range of coping strategies available to the individual, and may heighten distress by challenging the beliefs of some individuals in a fair world (Tennen & Affleck, 1990). DeGood and Kiernan (1996) have hypothesised that attributing fault or blame detracts from an individual's

efforts to develop adaptive strategies to cope with their situation because these attributions focus the individual's attention on issues of retribution, justice, or entitlement to compensation (when these issues are relevant).

This issue has also been of interest to chronic pain researchers. McParland, Whyte and Murphy-Black (2005) recruited 62 individuals with a range of chronic pain conditions from advertisements and interviewed them as part of a qualitative study investigating adjustment to chronic pain. The interviews revealed that attributing responsibility to other people for one's pain was associated with having experienced more negative emotions towards the pain since its onset. McParland et al. speculated that these attributions may contribute to poor adjustment by reducing an individual's perception of control over their pain. Alternatively, blaming others may also be associated with unwillingness to accept responsibility for one's condition, leading to poor compliance with treatment (McParland et al., 2005).

Evidence in support of this latter explanation comes from an earlier study conducted with chronic pain patients. DeGood and Kiernan (1996) examined the relationship between perception of fault (defined as the belief of some chronic pain patients that someone else, such as an employer or the other driver in a motor-vehicle accident, is to blame for their pain and suffering) and disability, distress and response to treatment. The sample consisted of 188 chronic pain patients admitted to an outpatient pain management program. Approximately one third of the sample faulted their employer or a generic "other" (mainly doctors or other drivers). Patients who attributed fault to their employer reported significantly higher levels of distress compared with those who attributed fault to the generic "other", who in turn reported significantly higher levels of distress than patients who did not ascribe fault to anyone. The groups did not differ in terms of pain intensity, location of pain, duration of pain, or degree of interference in

activities due to pain. Despite the similarities between the groups on these variables, DeGood and Kiernan found that perception of fault was strongly associated with poor response to past treatments and lower expectations about the future benefits of treatment.

Turk et al. (1996) have hypothesised that issues regarding perception of fault may be particularly relevant for individuals who have developed pain following an accidental injury, and have cited this factor as a possible explanation for differences in distress levels between individuals who develop pain following an accident, and those who experience a spontaneous or insidious onset of pain. Unfortunately, this hypothesis has not been tested as there are no studies to date comparing perceptions of fault in groups of chronic pain patients who have experienced different types of onset of pain.

The second potential explanation for the negative consequences of accidental injury is involvement in a compensation claim. Although early research in this area provided conflicting results (Turk & Okifuji, 1996), recent studies have indicated that compensation status may be an important predictor of outcome. For example, two recent prospective studies of large cohorts of Australian patients with acute injuries (specifically acute low back pain and orthopaedic trauma patients) have revealed that compensation is is one of the strongest predictors of poor prognosis (Gabbe, Cameron, Williamson, Edwards, Graves & Richardson, 2007; Henschke, Maher, Refshauge, Herbert, Cumming, Bleasel, York, Das & McAuley, 2008). Likewise, meta-analysis of the surgical literature has revealed a strong association between compensation status and poor outcome after surgery (Harris, Mulford, Solomon, van Gelder & Young, 2005). In studies of individuals with chronic pain, there is evidence that those involved in compensation or litigation report higher levels of pain, pain-related disability, and psychological symptoms, and are less likely to benefit from treatments or rehabilitation (Carron, DeGood & Tait, 1985;

Rohling, Binder & Langhinrichsen-Rohling, 1995; Turk & Okifuji, 1996; Rainville, Sobel, Hartigan & Wright, 1997; Blyth, March, Nicholas & Cousins, 2003).

The third potential explanation for the negative consequences of accidental injury is based on the observation that many accidents are potentially psychologically traumatic events. There is now considerable evidence that posttraumatic stress symptoms, including symptoms of sufficient severity to meet diagnostic criteria for PTSD, are a common sequelae of accidental injury, particularly motor-vehicle accidents (e.g. Blanchard & Hickling, 1997; Koren, Arnon & Klein, 1999; Mayou & Bryant, 2001; 2002; O'Donnell, Creamer, Bryant, Schnyder & Shalev, 2003; O'Donnell et al., 2004). The implications of this finding for research on the role of onset of pain will be discussed in the following section.

2.3.3. Summary

Some researchers have proposed that developing pain following a specific event, particularly if it involves injury or physical trauma, facilitates processes that are thought to contribute to poor adjustment to chronic pain, such as avoidance, fear of movement/(re)injury, or modified cognitive processing of pain and physical sensations. However, few studies have compared these variables in groups of patients with different types of onset of pain, and consequently, many of these hypotheses remain largely unexplored. Other investigators have pointed out that accidental injury is associated with long-term, negative psychosocial consequences, and have argued that certain aspects of being injured in an accident (e.g. issues of responsibility for the accident, the risk of developing PTSD) may account for the heightened distress and disability reported by accident victims. However, the existing research has not indicated whether it is the experience of developing chronic pain following an accident in particular or *any* specific

event that contributes to the observed differences between chronic pain patients with pain related to these events and patients who have experienced a spontaneous or insidious onset of pain.

2.4. Conclusions and implications for research

Studies examining the association between onset of pain and adjustment to chronic pain have typically included patients with accident-related pain in the group of patients who attribute their pain to any specific event. As a result, it is not clear whether the results of these studies can be interpreted as support for the importance of developing pain following an accident, or the importance of developing pain following any specific event. This question could be addressed by comparing three groups of patients: (1) individuals who attribute their pain to an accident; (2) individuals who attribute their pain to a specific incident that is not an accident; and (3) individuals who report that they experienced a spontaneous or insidious onset of pain. Conducting these comparisons is one of the aims of Study 2, presented in Chapter 7 of this thesis.

A second question arising from the research reviewed above is the degree to which the disability and distress reported by chronic pain patients with accident-related pain is attributable to the finding that many accidents are potentially psychologically traumatic events. Sustaining a physical injury, and subsequently developing a chronic pain condition, following an event that could potentially lead to the development of a posttraumatic stress response may impact negatively upon adjustment to chronic pain, even in the absence of significant posttraumatic stress symptoms. This hypothesis has not been investigated to date, since as far as the author is aware, previous studies have not compared individuals with chronic pain related to a potentially traumatic event, with

individuals who did not develop pain in the context of such an event. This approach to categorising types of onset of pain is depicted in Figure 2.2 below.

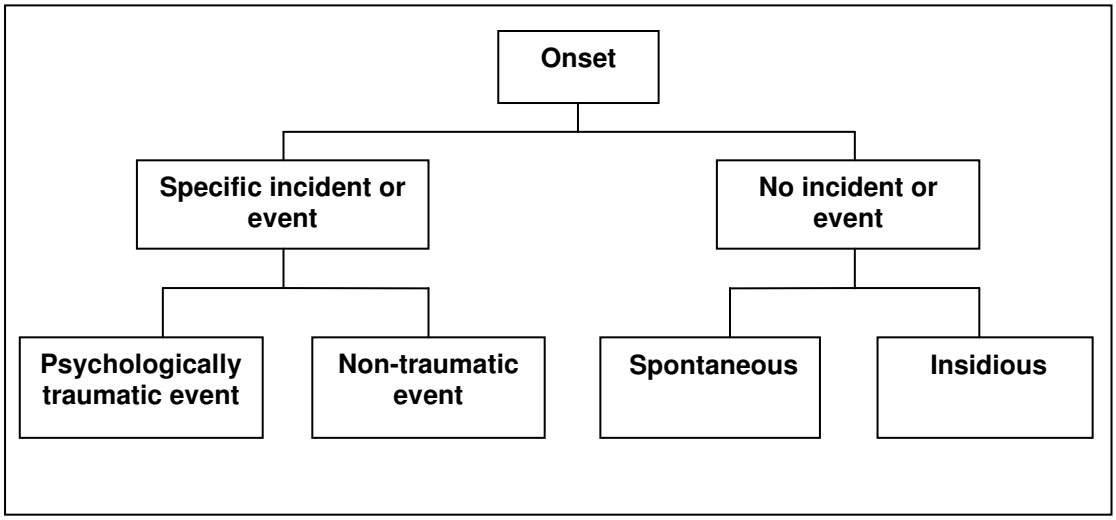

Figure 2.2. Types of onset of pain derived from previous research

Accordingly, the second aim of Study 2 was to compare chronic pain patients who developed pain in the context of a psychologically traumatic event, with patients who developed pain following a specific event that could not be considered traumatic. Both of these groups were also compared with patients who experienced a spontaneous or insidious onset of pain. In order to group participants according to these pain onset categories, it was necessary to identify the types of experiences that could be considered to be traumatic. The literature addressing this issue, and the nature of posttraumatic stress responses, is reviewed in the following chapter.

3. OVERVIEW OF TRAUMATIC EVENTS AND PTSD

3.1. The definition of a traumatic event

Since PTSD was first introduced into the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980), Criterion A or the "stressor criterion" (which provides the definition of a traumatic event) has been the subject of controversy and debate (e.g. Breslau & Davis, 1987; Davidson & Foa, 1991; March, 1993; Kilpatrick, Resnick, Freedy, Pelcovitz, Resick, Roth & van der Kolk, 1998; Brewin, 2003; McNally, 2003a; Spitzer, First & Wakefield, 2007; Weathers & Keane, 2007). One of the main points of contention has centred around how broadly (or narrowly) to define a traumatic event (Kilpatrick et al., 1998; Weathers & Keane, 2007). As Weathers and Keane (2007) have noted, developing a universal definition of a traumatic event has proved a challenge to the field because stressors lie on a continuum of severity; consequently, it is difficult to draw distinct boundaries between common stressors and those of a traumatic nature.

At the time of the publication of the DSM-III there was little empirical data concerning responses to traumatic events (Yehuda & McFarlane, 1995; Young & Yehuda, 2006). Since then research in the area of traumatic stress has burgeoned, and accordingly, the wording of the stressor criterion has been modified in the light of evidence challenging initial assumptions about the defining features of a traumatic event (Kilpatrick et al., 1998).

The DSM-III indicated that PTSD developed following events that were "generally outside the range of usual human experience" and stated that these types of events "would evoke significant symptoms of distress in most people" (American Psychiatric Association, 1980, p. 236). This definition conceptualised PTSD as a normal

response to an extraordinary or abnormal event (Yehuda & McFarlane, 1995). As such, the DSM-III excluded experiences considered to be common (e.g. "simple bereavement, chronic illness, business losses, or marital conflict") from the definition of a traumatic stressor (American Psychiatric Association, 1980, p. 236).

The revised edition of the DSM-III (DSM-III-R; American Psychiatric Association, 1987) described in more detail the types of events that could lead to PTSD (e.g. events that involved a serious threat to one's life), but upheld the DSM-III assumptions that traumatic events were uncommon and would cause significant distress in most people (Weathers & Keane, 2007). The DSM-III and DSM-III-R highlighted the importance of the magnitude or severity of the stressor as being central to the issue of whether an event can be considered traumatic (Weathers & Keane, 2007). This has been supported by the research, which has consistently shown that trauma severity is a significant predictor of the development of PTSD in groups of individuals exposed to a wide variety of events (March, 1993; Brewin, Andrews & Valentine, 2000). For example, being threatened with death or physical injury, being severely injured, being harmed or injured intentionally by another person, being exposed to grotesque sights, learning that one has been exposed to a noxious agent, or experiencing the violent or sudden death of a loved one are all event characteristics which have been associated with an increased risk for PTSD (Green, 1990; March, 1993).

However, it has also become apparent that subjective factors, such as the individual's perception of, and response to, the event, and factors that operate after the event are also important predictors of the likelihood of developing PTSD (Brewin et al., 2000). In recent meta-analyses the individual's perception of the degree to which their safety was threatened, and peritraumatic dissociation (dissociative experiences during or immediately after the event; Ozer, Best, Lipsey & Weiss, 2003) emerged as strong

predictors, as did the perceived level of social support received after the event (Brewin et al., 2000; Ozer et al., 2003). In other words, while the objective characteristics of the event are important, so too are individual and contextual factors (Keane & Barlow, 2002).

Consistent with this, the stressor criterion was modified in DSM-IV (American Psychiatric Association, 1994) to take into account both objective and subjective aspects of the traumatic experience (Kilpatrick et al., 1998). DSM-IV defined a traumatic event using two necessary criteria: firstly, the person must have "experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others" (Criterion A1); and secondly, the person must have responded with "intense fear, helplessness, or horror" (Criterion A2; American Psychiatric Association, 1994, pp. 427-8).

The second part of this definition shifted the emphasis away from the assumption made explicit in the DSM-III and DSM-III-R definition that a traumatic event would distress most people (Weathers & Keane, 2007). In addition to being criticised for not being empirically supported, the DSM-III and DSM-III-R definition has also been criticised for requiring a difficult and potentially unreliable judgement on the part of the clinician as to whether an event was outside usual human experience or would be distressing to almost anyone (Davidson & Foa, 1991; Kilpatrick et al., 1998). Criterion A2 was also included in the DSM-IV definition to take into account evidence gathered in the DSM-IV field trial that subjective distress at the time of the event was one of the important features that later distinguished between the presence or absence of PTSD (Kilpatrick et al., 1998).

For the first time DSM-IV criteria also stated that an individual could be traumatised by being confronted with an event they had not directly experienced or witnessed, for example, hearing about the traumatic experience of a loved one. The

experience of being diagnosed with a life-threatening illness (e.g. cancer) was also added to the list of traumatic stressors in DSM-IV. Examples of the types of events commonly identified as being consistent with the DSM-IV definition of a traumatic stressor are provided in Table 3.1. below.

Table 3.1: Examples of traumatic events according to DSM-IV Criterion A

Military combat	Being diagnosed with a life-threatening illness
Physical assault	Witnessing a dead body or body parts
Sexual assault or rape	Robbery
Being kidnapped or taken hostage	Torture
Being a prisoner of war or in a concentration camp	Terrorist attack
Motor vehicle accidents	Witnessing the serious injury, assault or unnatural death of another person
Natural disasters (e.g. floods, earthquakes, bushfires)	Learning that a loved one has experienced a violent assault or serious injury
Technological disasters (e.g. industrial accidents)	Accidental injury

It is obvious that some of the events listed in the table above are not as uncommon as DSM-III and DSM-III-R had initially stated, and this was another feature of the initial conceptualisation which was refuted by emerging epidemiological data (Young & Yehuda, 2006). In two large surveys employing the DSM-III-R definition of a traumatic stressor, Breslau, Davis, Andreski and Peterson (1991) and Norris (1992) reported that 39.1% and 69% of their community samples had experienced a traumatic event in their lifetime. Later studies confirmed these findings. For example, in Australia, the 1997 Australian National Survey of Mental Health and Well-being surveyed a representative, national sample of over 10,000 adults and found that 64.6% of males and 49.5% of females reported having experienced at least one traumatic event, with the majority of these adults having experienced more than one event (Creamer, Burgess & McFarlane, 2001). The most common events reported were witnessing someone being badly injured

or killed, being involved in a life-threatening accident, and being involved in a natural disaster.

Similarly, the National Comorbidity Survey (NCS; Kessler, Sonnega, Bromet, Hughes & Nelson, 1995) of a large sample representative of the US general population reported that 60.7% of men and 51.2% of women reported at least one traumatic event. As was the case in the Australian survey, the majority of the adults who reported experiencing a traumatic event had actually experienced two or more traumatic events. The most common events cited in the NCS were similar to those reported as being most common in the Australian study (Creamer et al., 2001). Comparably high rates of trauma exposure have also been reported in other large-scale community studies in the US (e.g. Resnick, Kilpatrick, Dansky, Saunders & Best, 1993; Breslau, Kessler, Chilcoat, Schultz, Davis & Andreski, 1998), Canada (Stein, Walker, Hazen & Forde, 1997), Mexico (Norris, Murphy, Baker, Perilla, Rodriguez & Rodriguez, 2003), and Sweden (Frans, Rimmo, Aberg & Fredrikson, 2005).

There is clearly some overlap between the types of events considered to be traumatic and the types of events often associated with the onset of chronic pain (e.g. motor-vehicle accidents). It could also be argued that other injuries or accidents associated with the onset of chronic pain could be considered traumatic if the incident involved serious injury or a serious threat to the individual's physical safety (e.g. workplace injuries involving falling from a significant height, being trapped under a large, heavy object, or being struck by a moving vehicle, flying objects, or moving machinery).

In summary, the current DSM definition of a traumatic stressor encompasses both objective aspects of the event and the individual's subjective response. Exposure to a potentially traumatic event is a common experience in the general population, and may be

even more prevalent among individuals with chronic pain given that some of the incidents associated with the onset of pain satisfy the DSM-IV criteria.

The focus of this chapter will now shift from the nature of a traumatic event to the consequences of exposure to a traumatic stressor. At this point it is important to note that although PTSD is only one of a number of psychological disorders which can develop in response to a traumatic event (Briere, 2004), it is considered the hallmark posttraumatic syndrome (Breslau, 1998). Consequently, it is the focus of the current investigation of the impact of a traumatic onset of pain on adjustment to chronic pain. The following sections provide an overview of PTSD, with a focus on the issues relevant to the topic of this thesis.

3.2. Posttraumatic Stress Disorder

As described in the previous section, the DSM-IV stipulates that the individual must have experienced a traumatic event to satisfy the criteria for a diagnosis of PTSD. According to the DSM-IV, the characteristic features of PTSD fall into three symptom clusters: (1) reexperiencing of the trauma; (2) avoidance of reminders of the event and general numbing of responsiveness; and (3) hyperarousal. A diagnosis of PTSD requires that the individual experiences at least one reexperiencing symptom (i.e. intrusive thoughts or memories of the event, nightmares, flashbacks, and intense psychological distress and physiological reactivity when exposed to internal or external stimuli that resemble the event), at least three avoidance or numbing symptoms (i.e. avoidance of thoughts and conversations about the trauma, or activities and places associated with the traumatic event, an inability to recall an important aspect of the event, diminished interest in activities, restricted range of affect, and a sense of a foreshortened future), and at least

two hyperarousal symptoms (i.e. difficulty falling or staying asleep, irritability or anger, difficulty concentrating, hypervigilance, and exaggerated startle response).

The duration of these symptoms must be longer than one month after the trauma, and the symptoms must cause significant distress and interference in the individual's functioning to qualify for the diagnosis. The DSM-IV classifies the condition as chronic if it has lasted for longer than three months. A specifier for delayed onset of symptoms (i.e. the symptoms develop six months after the trauma) is also provided; however, the evidence suggests that only a small minority of PTSD cases have a delayed onset of symptoms (Bryant & Harvey, 2002; Gray, Bolton & Litz, 2004). Research has also demonstrated that, in addition to the individuals who meet full diagnostic criteria for PTSD, there is a subset of individuals with "partial" or "subsyndromal" PTSD (Stein et al., 1997). This group do not report symptoms that meet the above diagnostic criteria, but nonetheless present with sufficient symptoms to cause significant distress and disability (Stein et al., 1997).

3.2.1. Prevalence of PTSD

As a result of the development of ideas concerning the definition of a traumatic event, changes to diagnostic criteria for PTSD over time, and diversity in methodologies across studies (e.g. use of different sampling methods or assessment procedures to enquire about exposure to trauma), figures regarding the prevalence of PTSD in the community have varied between studies (Kessler, 2000; Creamer et al., 2001; McFarlane, 2004). Despite this, a number of consistent findings have emerged from epidemiological research (Norris et al., 2003; Norris & Slone, 2007).

Although only a minority of exposed individuals develop persistent PTSD, given the high rates of exposure to traumatic events a significant number of people in the

general population are affected by PTSD (Norris & Slone, 2007). In the 1997 Australian National Survey of Mental Health and Well-being cited above, the prevalence of PTSD over the previous 12 months was 1.33% (Creamer et al., 2001), making it the second most common anxiety disorder in Australia (Andrews, Henderson & Hall, 2001). The 12-month prevalence of PTSD in the US replication of the NCS using DSM-IV diagnostic criteria was reported to be 3.5% (Kessler, Chiu, Demler, Merikangas & Walters, 2005), while lifetime prevalence was 6.8% (Kessler, Berglund, Demler, Jin, Merikangas & Walters, 2005). Although relatively few studies have been conducted in developing countries (de Girolamo & McFarlane, 1996), the available data indicates that the lifetime prevalence of PTSD is higher in poor or economically developing countries and in war-torn countries (e.g. de Jong, Komproe, Van Ommeren, El Masri, Araya, Khaled, van De Put & Somasundaram, 2001; Cardozo, Kaiser, Gotway & Agani, 2003; Norris et al., 2003; Cardozo, Bilukha, Crawford, Shaikh, Wolfe, Gerber & Anderson, 2004).

Not surprisingly, when compared with figures obtained from general population surveys, rates of PTSD are higher in groups of individuals who have all been exposed to trauma. For example, studies of Vietnam War combat veterans in both Australia and the US reveal significantly higher rates of PTSD in these veterans when compared with Vietnam era (non-combat) veterans and civilian samples (e.g. Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar & Weiss, 1990; O'Toole, Marshall, Grayson, Schureck, Dobson, Ffrench, Pulvertaft, Meldrum, Bolton & Vennard, 1996). For example, the National Vietnam Veterans Readjustment Study conducted in the US found that the lifetime and current prevalence rates of PTSD were five to ten times higher in Vietnam combat veterans than noncombat veterans and civilians (Kulka et al., 1990). Similarly, veterans of other wars and military personnel deployed to peacekeeping missions also exhibit elevated rates of PTSD (e.g. Litz, Orsillo, Friedman, Ehlich & Batres, 1997;

Hoge, Castro, Messer, McGurk, Cotting & Koffman, 2004; Ikin, Sim, Creamer, Forbes, McKenzie, Kelsall, Glass, McFarlane, Abramson, Ittak, Dwyer, Blizzard, Delaney, Horsley, Harrex & Schwarz, 2004).

Posttraumatic stress symptoms and PTSD have also been found to be common among victims of violent crime (e.g. Kilpatrick, Saunders, Veronen, Best & Von, 1987; Resnick et al., 1993), with victims of sexual and physical assault being at increased risk of developing PTSD compared with individuals exposed to other traumatic events (Kilpatrick, Saunders, Amick-McMullan, Best, Veronen & Resnick, 1989; Breslau et al., 1991; Resnick et al., 1993; Breslau et al., 1998; Creamer et al., 2001; Rosenman, 2002). Other groups identified as being at risk of developing PTSD include individuals affected by natural disasters (e.g. Green, Lindy, Grace, Gleser, Leonard, Korol & Winget, 1990; Ironson, Wynings, Schneiderman, Baum, Rodriguez, Greenwood, Benight, Antoni, LaPerriere, Huang, Klimas & Fletcher, 1997), motor-vehicle accidents victims (e.g. Blanchard, Hickling, Taylor, Loos & Gerardi, 1994; Ehlers, Mayou & Bryant, 1998; Harvey & Bryant, 1999; Koren et al., 1999; Mayou & Bryant, 2002), and occupational groups routinely exposed to traumatic events, such as police officers (e.g. Carlier, Lamberts & Gersons, 1997; Maia, Marmar, Metzler, Nobrega, Berger, Mendlowicz, Coutinho & Figueira, 2007) and firefighters (e.g. McFarlane, 1988; Bryant & Harvey, 1996; Wagner, Heinrichs & Ehlert, 1998). Following the inclusion of life-threatening illness in DSM-IV's definition of a traumatic stressor, it has also become apparent that being diagnosed with a life-threatening illness (e.g. cancer or HIV/AIDS), and the experience of sudden and life-threatening physical events (e.g. myocardial infarction or stroke) are also associated with PTSD (Tedstone & Tarrier, 2003).

3.2.2. Prognosis and associated features

Initial stress reactions following exposure to a traumatic event appear to be common, but largely transient, with the majority of symptoms remitting spontaneously within weeks (Bryant, 2006). For example, Rothbaum, Foa, Riggs, Murdock and Walsh (1992) reported that while 94% of the rape victims in their study reported symptoms that met criteria for PTSD in the first two weeks following the assault (excluding the duration criterion of one month), by 12 weeks only 47% reported symptoms that were sufficient for a diagnosis. Blanchard, Hickling, Barton, Taylor, Loos and Jones-Alexander (1996b) found that half of their sample of MVA victims with PTSD had remitted by the six month follow-up, and two-thirds had remitted by the one year follow-up.

However, for those individuals who develop chronic symptoms PTSD often persists for many months or years after the traumatic event (Davidson, 2004). For example, in an early general population survey, Davidson, Hughes, Blazer and George (1991) reported that 46% of all identified PTSD cases were chronic (i.e. had persisted for at least six months). Similarly high proportions of chronic cases have been reported in other general population surveys (e.g. Breslau & Davis, 1992; Kessler et al., 1995; Norris et al., 2003) and in surveys of groups of individuals exposed to trauma (e.g. Australian firefighters exposed to the 'Ash Wednesday' bushfires in 1983; McFarlane, 1988). Studies of war veterans and victims of disasters have revealed that individuals may endure symptoms of PTSD for several decades (e.g. Goldberg, True, Eisen & Henderson, 1990; Green et al., 1990; Kulka et al., 1990; O'Toole et al., 1996; Schnurr, Ford, Friedman, Green, Dain & Sengupta, 2000).

The research has also indicated that PTSD is associated with high rates of psychiatric comorbidity (Brady, Killeen, Brewerton & Lucerini, 2000). In the 1997 Australian Survey of Mental Health and Well-being, Creamer and colleagues (2001)

reported that 85.2% of males and 79.7% of females with PTSD met diagnostic criteria for another DSM-IV Axis 1 disorder. Furthermore, nearly 50% of females and over 60% of males with PTSD met criteria for two or more additional diagnoses. The disorders most commonly comorbid with PTSD include major depression, substance abuse disorders, and other anxiety disorders (Breslau et al., 1991; Kessler et al., 1995; Mills, Teesson, Ross & Peters, 2006). While there are different views regarding the nature of the relationship between PTSD and these disorders, it is clear that individuals with PTSD and other diagnoses are more distressed and dysfunctional than individuals with PTSD alone (Brady et al., 2000). Several studies have also found that individuals with PTSD are at a significantly higher risk of attempting suicide compared with individuals with other anxiety disorders or without PTSD (e.g. Davidson et al., 1991; Kotler, Iancu, Efroni & Amir, 2001).

It has also been noted that individuals with PTSD present with a variety of other problems. For example, there is considerable evidence that PTSD is associated with an increased prevalence of self-reported health problems and chronic medical conditions (Schnurr, Green & Kaltman, 2007). McFarlane, Atchison, Rafalowicz and Papay (1994) studied a group of over 400 Australian firefighters exposed to severe bushfires and reported significantly higher rates of cardiovascular, respiratory, musculoskeletal and neurological symptoms in firefighters diagnosed with PTSD. In the NCS, PTSD was strongly associated with an increased likelihood of neurological, vascular, gastrointestinal, metabolic or autoimmune, and bone or joint conditions, even more so than other anxiety disorders assessed in the survey (Sareen, Cox, Clara & Asmundson, 2005). This association has been reported repeatedly in both civilian and veteran samples (e.g. Shalev, Bleich & Ursano, 1990; Davidson et al., 1991; Beckham, Moore, Feldman, Hertzberg, Kirby & Fairbank, 1998; Engel, Liu, McCarthy, Miller & Ursano, 2000;

Wagner, Wolfe, Rotnitsky, Proctor & Erickson, 2000; Ouimette, Cronkite, Henson, Prins, Gima & Moos, 2004; Gillock, Zayfert, Hegel & Ferguson, 2005). Consistent with this, compared with control groups without PTSD, individuals with PTSD use significantly more health care services (e.g. Switzer, Dew, Thompson, Goycoolea, Derricott & Mullins, 1999; Stein, McQuaid, Pedrelli, Lenox & McCahill, 2000; Walker, Katon, Russo, Ciechanowski, Newman & Wagner, 2003; Gillock et al., 2005). Accordingly, PTSD has also been associated with higher health care costs, even when the effects of depression, chronic medical illness, and demographic differences are taken into account (Walker et al., 2003).

Given the prevalence of PTSD in the community, associated physical and psychiatric comorbidity, and high health care utilisation rates, PTSD has been described as a significant economic and social burden (Solomon & Davidson, 1997; Kessler, 2000; Chan, Medicine, Air & McFarlane, 2003).

3.2.3. Risk factors for the development of PTSD

Two meta-analyses (Brewin et al., 2000; Ozer et al., 2003) have summarised the considerable literature on risk factors for the development of PTSD. The variables examined in these meta-analyses included pretrauma variables (e.g. gender, age, race, previous trauma, prior psychological adjustment, family history of psychopathology), trauma variables (e.g. trauma severity, perceived life threat during the trauma, peritraumatic emotional responses and dissociation) and posttrauma variables (e.g. social support, additional life stress). As Brewin et al, Ozer et al and others (e.g. Vogt, King & King, 2007) have noted, the effect sizes of many of the analysed variables were modest, and often varied with differences in methodology and the population being studied,

indicating that it is unlikely that one set of factors will provide a causal framework across all populations.

This issue aside, the analyses revealed that pretrauma demographic factors such as female gender, younger age at the time of the trauma, lower socioeconomic status, lower education, and belonging to a racial or ethnic minority are associated with an increased risk for developing PTSD (Brewin et al., 2000). Psychiatric history and previous exposure to a traumatic event also predict the development of PTSD (Brewin et al., 2000; Ozer et al., 2003). Interestingly, the meta-analyses revealed that trauma-related variables (particularly trauma severity and peritraumatic dissociation) and factors operating after the trauma (specifically, level of social support) were the strongest predictors of PTSD.

3.3. PTSD following traumatic injury

As O'Donnell, Bryant, Creamer and Carty (2008) have pointed out, there are numerous aspects of the experience of being injured that could potentially be experienced as traumatising. For example, at the time of the injury the individual may experience fear or horror, or perceive that they are helpless to prevent the injury. After the injury they may have to undergo stressful or painful medical procedures, and in the longer-term may have to adjust to permanent sequelae of the injury, such as disability or pain (O'Donnell et al., 2008). Thus, Shalev (2002) has distinguished between the "primary stressors" faced by injury survivors (e.g. the accident itself), and "secondary stressors" (e.g. the longer-term disability or pain). All of these facets of being injured are thought to contribute to the experience of the injury as traumatic (Shalev, 2002).

The traumatic events which have been studied in the context of physical injury include motor vehicle accidents, other accidents, workplace injuries, and assaults (see e.g. O'Donnell et al., 2004; O'Donnell, Elliott, Lau & Creamer, 2007). As is the case with all

stressors, physical injuries have the potential to be traumatising (because of the factors outlined above), but not all individuals experience them as traumatic (i.e. they do not respond with fear, helplessness, or horror). Consequently, the term "traumatic injury" is used in the literature to refer to injuries as "potentially traumatic events" (Shalev, 2002; O'Donnell et al., 2008).

Large-scale epidemiological studies of the general population indicate that the experience of a potentially traumatic physical injury is common. Many of these studies assume that accidents are associated with serious injury and enquire about exposure to accidents and injuries together. In the 1997 Australian Survey of Mental Health and Well-being, Creamer et al. (2001) found that 28.3% of men and 13.6% of women had been involved in a life-threatening accident. Almost identical figures for exposure to life-threatening accidents were reported in the NCS (25% of men and 13% of women; Kessler et al., 1995). Similarly, in a general population survey in Mexico, Norris et al. (2003) reported that more than 32% of the sample had experienced a life-threatening accident. Breslau et al.'s (1991) large survey of young adults revealed that 9.4% of the sample had experienced a sudden injury or serious accident, making it the most common traumatic event the participants had experienced in their lifetime.

Although epidemiological surveys have indicated that the risk of developing PTSD following serious accidents or injuries is low relative to other traumatic events (Kessler et al., 1995; Breslau et al., 1998), because they occur frequently they are commonly associated with PTSD (O'Donnell, Creamer, Bryant, Schnyder & Shalev, 2006). Creamer et al. (2001) found that 24.7% of the participants with PTSD surveyed for the Australian Survey of Mental Health and Well-being attributed their symptoms to a life-threatening accident. Breslau and colleagues (1998) reported that 10.6% of cases of PTSD attributed their symptoms to a serious motor-vehicle accident or other serious accident.

Apart from these epidemiological studies, most of the knowledge about PTSD and traumatic injury has emerged from research focusing on individuals who present to hospital emergency departments following a serious injury. PTSD prevalence rates have varied considerably across these studies, largely due to methodological differences, such as assessment methods or sampling variations (O'Donnell et al., 2003). For example, an Australian study that used a well-established structured clinical interview to diagnose PTSD (The Clinician Administered PTSD Scale for DSM-IV; Weathers, Keane & Davidson, 2001) in a sample of over 300 severely injured trauma survivors, reported a PTSD rate of 9% at three months post-trauma (Creamer, O'Donnell & Pattison, 2004). In contrast, Matthews and Chinnery (2005) used a self-report measure of PTSD symptoms (the PTSD checklist or PCL; Weathers, Litz, Herman, Huska & Keane, 1993) in 69 Australian injured trauma survivors and reported a PTSD rate of over 17% at an average of eight months post-trauma.

Studies conducted overseas using structured clinical interviews to diagnose PTSD in samples of hospitalised trauma survivors have reported higher rates than Australian studies. For example, Shalev, Freedman, Peri, Brandes, Sahar, Orr and Pitman (1998) reported a PTSD rate of 30% at one month post-trauma in their Israeli study, and Mellman, David, Bustamante, Fins and Esposito (2001) reported a PTSD rate of 24% at six weeks post-trauma in their US study.

As is the case with other PTSD groups, only a minority of injured trauma survivors experience posttraumatic stress symptoms that persist for several months (O'Donnell et al., 2007). However, those individuals whose symptoms do meet diagnostic criteria at 12 months experience an escalation of their symptoms over time (O'Donnell et al., 2007). Estimates of the prevalence of PTSD in injured trauma victims at 12 months post-trauma

have varied between 2% (Schnyder, Moergeli, Klaghofer & Buddeberg, 2001) and 30% (Zatzick, Kang, Muller, Russo, Rivara, Katon, Jurkovich & Roy-Byrne, 2002).

3.4. Psychological models of PTSD

Although numerous psychological models of PTSD have been developed since its introduction into the DSM-III, this section will focus on the models that are relevant to the discussion in subsequent chapters on the impact of PTSD on adjustment to chronic pain. This includes models based on conditioning theories and cognitive models of anxiety.

3.3.1. Learning theory model of PTSD

The main model of PTSD based on theories of conditioning is the learning theory model of Keane and colleagues (Keane, Fairbank, Caddell, Zimering & Bender, 1985; Keane, Zimering & Caddell, 1985), which was initially forwarded to account for the experiences of Vietnam veterans. This model applied Mowrer's (1960) two-factor theory, which proposed that fear is acquired through classical conditioning, and avoidance maintained through operant conditioning. Keane et al. hypothesised that individuals are exposed to a variety of stimuli during a traumatic event (e.g. sights, sounds, smells, cognitions) that become associated with the intense anxiety experienced during the event, so that after the trauma these stimuli elicit memories of the event and the anxiety response in the absence of the event itself. By repeated pairing of new stimuli with the trauma-related stimuli after the event, the individual may learn to respond with fear to an increasingly wide range of stimuli (a process known as higher-order conditioning), particularly those that resemble the trauma-related stimuli (the principle of stimulus generalisation). Keane and colleagues argued that these classical conditioning processes could account for the intense emotional response experienced by Vietnam veterans when

faced with both direct reminders of combat duty, and with everyday stimuli that resembled combat-related stimuli (e.g. a car backfiring being similar to the sound of gunfire). Furthermore, internal experiences (e.g. memories of the traumatic event) could also become conditioned stimuli.

Applying the second aspect of Mowrer's theory, Keane and colleagues proposed that individuals with PTSD attempt to avoid the stimuli that elicit the conditioned response, leading to pervasive avoidance behaviour of both external and internal stimuli as the range of conditioned stimuli increases. Avoidance of the conditioned stimuli prevents the aversive conditioned response, thereby reinforcing the avoidance behaviour. Keane et al. suggested that because of this avoidance behaviour, exposure to the conditioned stimuli (including the memory of the trauma itself) is brief and incomplete, thus preventing the conditioned response from being extinguished.

Keane and colleagues also identified other factors that prevent complete exposure to the traumatic memory. Firstly, state-dependent memory (Bower, 1981) is used to explain why individuals with PTSD are often unable to recall important aspects of the traumatic event. That is, recall is impaired because a traumatic memory is encoded in an emotional, physiological, and cognitive state that is significantly different to the state in which trauma survivors typically attempt to recall the event. Recall is thought to improve only if all of the state-related cues are provided. Consistent with this, there is strong evidence that increased arousal and activation of the fear response is associated with greater benefit from treatment protocols that include imaginal recall of the trauma memory (Cahill & Foa, 2007). Secondly, Keane et al. argued that limited opportunities to discuss combat experiences during the Vietnam War, and afterwards due to political factors, also prevented the veterans from engaging with the traumatic memory for any length of time.

Keane and Barlow (2002) also proposed a model of the aetiology of PTSD which utilises both the tenets of the learning theory model and Barlow's (2002) theory of fear and anxiety. Keane and Barlow's model postulates that individuals who develop PTSD in response to a traumatic stressor bring to the event the two basic vulnerabilities Barlow delineated: (1) a generalised psychological vulnerability; and (2) a generalised biological vulnerability. The biological vulnerability refers to a genetic tendency to experience intense negative affect. The psychological vulnerability refers to a learned tendency to feel that events and one's reaction to them are unpredictable and uncontrollable. Barlow also distinguished between true and false alarms, both of which trigger physiological arousal. A false alarm occurs when the physiological response is elicited by non-threatening stimuli. Keane and Barlow proposed that a true alarm occurs at the time of the traumatic event, leading to learned (false) alarms when the individual is exposed to stimuli that are reminiscent of the event. In the face of this physiological arousal the inclination to perceive things as uncontrollable leads to intense negative affect and hence, attempts to avoid the alarms and the stimuli which trigger them. Keane and Barlow asserted that attempts to avoid the affect elicited by false alarms accounts for the emotional numbing symptoms of PTSD.

A key strength of the learning theory model of PTSD is its ability to explain many of the symptoms of PTSD (Brewin & Holmes, 2003). However, the model has also been criticised for neglecting some symptoms, such as numbing of general responsiveness and exaggerated startle responses (Foa, Steketee & Rothbaum, 1989; Taylor, 2006; Cahill & Foa, 2007), or for providing mechanisms that are only applicable to the experience of Vietnam veterans (Taylor, 2006; Cahill & Foa, 2007). In addition, although Keane and colleagues make reference to the potential role of familial factors, pretrauma history, coping strategies, and social support systems, they do not attempt to delineate underlying

mechanisms, or explain why only some individuals who are exposed to a traumatic event develop PTSD (Jones & Barlow, 1990). Finally, subsequent research has demonstrated the importance of cognitive factors in the development and maintenance of PTSD (see Section 3.3.2 below), and Keane et al.'s model does not incorporate cognitive variables (Brewin & Holmes, 2003; Taylor, 2006). Consequently, the conditioning principles highlighted by the learning theory model of PTSD have been integrated into other models (Brewin & Holmes, 2003; Taylor, 2006).

3.3.2. Ehlers and Clark's (2000) cognitive model of PTSD

Ehlers and Clark's (2000) cognitive model of PTSD is based on cognitive models of anxiety that conceptualise anxiety as being the result of appraisals of threat (see e.g. Eysenck, 1997). Ehlers and Clark proposed that PTSD persists if the individual's interpretations of the traumatic event and/or its sequelae lead to a sense of current threat. These interpretations are idiosyncratic, can be about different aspects of the event (i.e. the event itself, the individual's response during and after the event, other people's reactions to the event, or negative consequences of the trauma), and can represent either external or internal threats.

For example, appraisals of the traumatic event itself may involve overgeneralising and concluding that even normal daily activities are more dangerous than they are in reality (an external threat). Alternatively, the individual may appraise their feelings or behaviour during the event in a way which challenges their view of themselves as competent and able to cope with life (e.g. because they cried or tried to escape at the time). This is an example of an appraisal that generates an internal threat.

Ehlers and Clark stated that threat appraisals can also be related to the individual's PTSD symptoms. For example, they may interpret their initial intrusive thoughts or

images as a sign of mental illness, instead of attributing them to a normal process of recovery following a traumatic event. Importantly for the role of physical injury in the maintenance of PTSD, Ehlers and Clark have pointed out that threat appraisals may also be about the consequences of the trauma, including long-term physical consequences. For example, the individual may believe that their body is permanently changed, or that they will never be able to live a normal life again because of their injuries.

In addition to these idiosyncratic threat appraisals, Ehlers and Clark hypothesised that the nature of the traumatic memory and its relationship to the individual's other autobiographical memories can also produce a sense of current threat. According to Ehlers and Clark, unlike other autobiographical memories, the trauma memory is fragmented and poorly elaborated, and is not completely integrated into its context among other autobiographical memories. This is thought to lead to the individual's difficulties in intentionally recalling the event, to the intrusive reexperiencing symptoms, and to the sense that the event is happening again when the memory is activated. Ehlers and Clark argued that these features of the trauma memory are related to the way in which the individual processes the traumatic event at the time. That is, the memory will be fragmented and poorly elaborated if the individual engages in predominantly data-driven processing (i.e. processing mainly sensory impressions) rather than conceptual processing (i.e. processing the meaning of the situation in an organised way) during the trauma.

Drawing upon conditioning theories and the work of Foa and colleagues (e.g. Foa & Kozak, 1986; Foa et al., 1989), Ehlers and Clark also suggested that traumatic memories are characterised by strong stimulus-stimulus and stimulus-response associations, so that memories of the event and associated stimuli and responses are easily triggered. In particular, their model ascribes a role to strong perceptual priming; that is, a reduced perceptual threshold for stimuli that were temporally associated with the

trauma. Consequently, even stimuli which only resemble trauma-related stimuli can trigger the trauma memory, contributing further to the sense of current threat. Furthermore, in the presence of these reminders, individuals with PTSD exhibit a tendency to selectively attend to threat-related cues, particularly those that confirm their appraisals. In this way, the cognitive process of selective attention contributes to the maintenance of the sense of threat.

According to the model, the sense of threat generated by the trauma memory and the individual's appraisals leads to the symptoms characteristic of PTSD (e.g. reexperiencing symptoms, hyperarousal, and feelings of anxiety), and motivates a range of behavioural and cognitive responses that are intended to reduce the sense of threat and the individual's distress. According to Ehlers and Clark, although these responses are successful in achieving these aims in the short-term, in the longer-term they contribute to increased PTSD symptoms, prevent modification of the underlying threat appraisals, or prevent change in the nature of the trauma memory, thereby maintaining the PTSD symptoms. For example, avoiding thinking about the trauma because the individual believes that intrusive thoughts indicate they are going crazy only increases the frequency of those thoughts. Safety behaviours (i.e. actions designed to prevent a feared consequence from occurring; Salkovskis, 1996) prevent changes in the individual's appraisals of the trauma or its consequences by maintaining their belief that the feared consequence would occur if they did not engage in the safety behaviour. Finally, avoidance of reminders of the trauma maintains the nature of the trauma memory by preventing access to cues that might assist with the elaboration and integration of the memory into autobiographical memory.

As others have noted, there is good empirical support for many aspects of Ehlers and Clark's cognitive model of PTSD (Brewin & Holmes, 2003; Taylor, 2006). For

example, studies have revealed a relationship between PTSD symptoms and threat appraisals, including negative interpretations of the trauma and its consequences (Dunmore, Clark & Ehlers, 2001; Ali, Dunmore, Clark & Ehlers, 2002), and negative interpretations of PTSD symptoms (Ehlers et al., 1998; Halligan, Michael, Clark & Ehlers, 2003). There is also evidence that the maladaptive coping strategies identified by Ehlers and Clark (e.g. avoidance and safety behaviours, thought suppression) predict persistence of PTSD symptoms (Clohessy & Ehlers, 1999; Steil & Ehlers, 2000; Dunmore et al., 2001). Also, experimental paradigms such as the modified Stroop and dot-probe tasks have confirmed that individuals with PTSD exhibit an attentional bias towards trauma-related threat words (e.g. McNally, Kaspi, Riemann & Zeitlin, 1990; Foa, Feske, Murdock, Kozak & McCarthy, 1991; Cassiday, McNally & Zeitlin, 1992; Bryant & Harvey, 1995; 1997). Finally, recent studies have provided support for the role of disordered autobiographical memory processes (Kleim & Ehlers, 2008), and enhanced perceptual priming (Michael, Ehlers & Halligan, 2005; Michael & Ehlers, 2007). Importantly, a recent prospective study of MVA survivors found that cognitive variables highlighted by the cognitive model (e.g. cognitive processing during the MVA, memory disorganisation, negative appraisals of the trauma and its sequelae, safety behaviours, and thought suppression) predicted PTSD severity at six months better than initial symptom levels and other variables identified as significant predictors in Ozer et al.'s (2003) meta-analysis (Ehring, Ehlers & Glucksman, 2008).

The evidence in favour of the cognitive model aside, a number of problems with Ehlers and Clark's conceptualisation have been identified. Firstly, Brewin and Holmes (2003) have argued that the ecological validity of the cognitive processing paradigms used to investigate data-driven versus conceptual processing is questionable given the complexity of responses to traumatic events in the "real world". Secondly, Taylor (2006)

has questioned the model's emphasis on data-driven processing at the time of the trauma, pointing out that some degree of conceptual processing must occur given that the perceptions of life-threat during the trauma have consistently been shown to be significant predictors of the development of PTSD. Finally, the model has been criticised for not providing sufficient explanation of why only some individuals develop PTSD (Dalgleish, 2004; Taylor, 2006).

3.5. Treatment of PTSD

Over the last two decades significant advances have been made in the treatment of PTSD (Taylor, 2004). The focus of the following discussion is cognitive-behavioural therapy (CBT) because it has been the most thoroughly evaluated (Foa & Meadows, 1997; Harvey, Bryant & Tarrier, 2003), and it is considered the psychological treatment of choice for PTSD (Bryant, 2006).

A number of different protocols for the delivery of CBT for PTSD have been developed over the years, some of them targeting specific groups of PTSD patients, for example, combat-related PTSD, motor-vehicle accident victims, or rape victims (e.g. Keane, Fisher, Krinsley & Niles, 1994; Blanchard & Hickling, 1997; Foa & Rothbaum, 1998). These CBT packages tend to have a number of treatment components in common; namely, psychoeducation, anxiety management training, exposure therapy, and cognitive restructuring (Harvey et al., 2003).

Psychoeducation typically involves providing the patient with information about typical responses to traumatic events, a model to account for posttraumatic stress responses and the core symptoms of PTSD, and a rationale for treatment (see e.g. Blanchard & Hickling, 1997; 2004). Anxiety management involves training the patient to utilise a range of coping skills (e.g. breathing retraining, applied relaxation, grounding

techniques) to effectively manage physiological arousal in daily life and during the exposure phase of treatment (Taylor, 2006).

Exposure therapy entails both prolonged imaginal exposure to the patient's memory of the traumatic event and in vivo graded exposure to feared trauma-related situations and places (see e.g. Foa & Rothbaum, 1998). During prolonged imaginal exposure the therapist assists the patient in developing a verbal (e.g. Foa & Rothbaum, 1998) or written (e.g. Resick & Schnicke, 1993) narrative of their traumatic experience that integrates all of the sensory, cognitive, and affective details necessary to generate a vivid sense of reliving the event. Prolonged imaginal exposure sessions typically last for longer than an hour (especially initially) and are accompanied by homework tasks which involve repeatedly reading or listening to an audiotape of the narrative. From the perspective of Mowrer's two factor theory, exposure promotes habituation of the conditioned fear response to the trauma memory and disrupts the associated avoidance behaviour (Foa & Meadows, 1998). According to emotional processing paradigms of PTSD, exposure therapy promotes the emotional processing of the trauma memory by introducing and incorporating corrective information into the trauma memory (Foa & Kozak, 1986). Exposure is also thought to help the patient to distinguish between the trauma memory and the event itself, and to allow the patient to realise that anxiety will not persist indefinitely when confronting trauma-related stimuli (Foa & Meadows, 1998).

Cognitive restructuring consists of identifying and modifying the cognitions and beliefs about the traumatic event and its sequelae that are conceptualised as maintaining the patient's maladaptive coping strategies and distress (see e.g. Ehlers, Clark, Hackmann, McManus & Fennell, 2005). It is worth noting that additional treatment modules (e.g. anger management training or communication skills training) are

sometimes included in CBT to address problems specific to certain populations (Taylor, 2006).

In practice, many of the above treatment components (particularly exposure and cognitive restructuring) are interwoven together (Harvey et al., 2003), and different protocols approach this slightly differently depending on the theoretical stance of the researchers. For example, while Ehlers et al.'s (2005) variant of cognitive therapy utilises many well-established techniques (e.g. imaginal reliving combined with cognitive restructuring), the focus is on targeting the key maintaining variables as identified by Ehlers and Clark's (2000) cognitive model. CBT protocols which have been empirically evaluated usually involve 8-12 individual therapy sessions delivered on an outpatient basis (Bisson, Ehlers, Matthews, Pilling, Richards & Turner, 2007).

As the results of several meta-analyses and systematic reviews attest, there is strong empirical support for the efficacy of CBT for PTSD (Van Etten & Taylor, 1998; Foa, Keane & Friedman, 2000; Bradley, Greene, Russ, Dutra & Westen, 2005; Bisson et al., 2007). These reviews have consistently found CBT (and particularly exposure therapy) to be superior to waiting-list control and other active treatment conditions across a diverse range of trauma groups. In the most recent meta-analysis, Bisson et al. (2007) identified 38 randomised controlled trials of psychological treatment for chronic PTSD for review. They concluded that, compared with waiting-list and usual care conditions, CBT produced clinically important gains on all measures of PTSD symptoms. The analysis also provided limited support for the superiority of CBT over supportive/non-directive therapies. Several authors have noted that the ability of trials to detect differences between CBT and other active treatments has been hampered by insufficient statistical power due to small sample sizes (e.g. Harvey et al., 2003; Resick, Monson & Gutner, 2007).

These reviews have also highlighted a number of important issues for future research. In particular, a need to improve the efficacy of CBT for PTSD has been highlighted repeatedly (e.g. Harvey et al., 2003; Bradley et al., 2005; Resick et al., 2007). While the exact figures vary across studies, 15-50% of participants still meet diagnostic criteria for PTSD post-treatment or at follow-up (Harvey et al., 2003; Taylor, 2006; Resick et al., 2007), or continue to report residual symptoms despite no longer meeting diagnostic criteria (Bradley et al., 2005). Consequently, recent clinical trials have focused on either investigating the effects of combining or integrating treatments to enhance treatment gains, or dismantling the components of CBT in order to ascertain the aspects most responsible for treatment change (Resick et al., 2007). For example, Bryant, Moulds, Guthrie, Dang and Nixon (2003) investigated the degree to which combining cognitive restructuring with imaginal exposure would lead to greater treatment gains compared with providing imaginal exposure alone. They found that the combination was superior to imaginal exposure alone in reducing PTSD symptoms in 58 survivors of non-sexual assault or motor-vehicle accidents. In addition to increased attention to these issues, recommendations have also been made for larger, multi-site trials focused on demonstrating the generalisability of previous findings, and testing the effectiveness of CBT in real clinical practice (Harvey et al., 2003; Bisson et al., 2007; Resick et al., 2007). The need to minimise drop-out rates by improving the tolerability of exposure therapy has also been noted (Bisson et al., 2007). Relevant to the topic of this thesis, the high rate of comorbidity in PTSD underscores the importance of including participants with other conditions in clinical trials and broadening the range of treatment outcome variables targeted by CBT (Bradley et al., 2005; Spinazzola, Blaustein & van der Kolk, 2005; Bisson et al., 2007; Resick et al., 2007).

3.6. Chapter summary

Exposure to trauma is a common experience in the general population and PTSD is prevalent among individuals exposed to traumatic events. The course of PTSD is often chronic, and is associated with significant psychiatric and medical comorbidity. Numerous psychological models of PTSD have been formulated, mainly based on cognitive and behavioural theories of psychopathology. These models have led to the development of empirically supported cognitive-behavioural treatments to ameliorate PTSD symptoms across a broad range of trauma survivors. Despite advances in treatment, little is known about optimal treatment of PTSD when individuals also present with chronic pain. This issue is the focus of the final chapter of this literature review.

4. CHRONIC PAIN AND PTSD

As discussed in the previous chapter, PTSD is a common outcome of exposure to potentially traumatic events, including motor-vehicle accidents and accidental injuries. Considering the overlap between these events and the events typically associated with the onset of chronic pain it is likely that some chronic pain patients who initially experience a sudden onset of their pain also develop PTSD. Therefore, it is possible that the higher rates of disability and distress observed in chronic pain patients who attribute their pain to an accident or another specific event (if it satisfies the DSM-IV definition of a traumatic stressor) can be at least partly explained by an interaction between the problems typically experienced in chronic pain and those related to PTSD. In fact, several authors have claimed that when chronic pain and PTSD co-occur the two conditions interact and influence each other in a way that maintains the disability and distress associated with each (Bryant, Marosszeky, Crooks, Baguley & Gurka, 1999; Sharp & Harvey, 2001; Asmundson, Coons, Taylor & Katz, 2002; Sharp, 2004).

This chapter will review the evidence that chronic pain and PTSD are frequently comorbid conditions, and will critically examine the research exploring the relationship between them. This will include an outline of the models which have been proposed to account for the association between chronic pain and PTSD, and a review of the supporting empirical data.

4.1. Comorbidity of chronic pain and PTSD

Early reports of a link between chronic pain and PTSD consisted mainly of brief case studies describing patients presenting with both diagnoses (e.g. Pilowsky, 1985; Rapaport, 1987; Lebovits, Yarmush & Lefkowitz, 1990; Schreiber & Galai-Gat, 1993). As a result of these case studies, research efforts have shifted towards identifying both the

prevalence of PTSD in chronic pain samples and the proportion of PTSD patients who report chronic pain.

4.1.1. Prevalence of PTSD in chronic pain

There is substantial evidence from large-scale epidemiological surveys world-wide that PTSD is a common condition among individuals in the general population who experience chronic pain. A large survey (i.e. N = 85,088) of pooled data from 17 countries revealed that individuals with chronic neck or back pain are almost three times more likely to have PTSD than those with no pain (Demyttenaere, Bruffaerts, Lee, Posada-Villa, Kovess, Angermeyer, Levinson, de Girolamo, Nakane, Mneimneh, Lara, de Graaf, Scott, Gureje, Stein, Haro, Bromet, Kessler, Alonso & Von Korff, 2007). In the US NCS, McWilliams, Cox and Enns (2003) examined the relationship between individuals who reported having "severe arthritis, rheumatism, or another bone or joint disease" (p. 128) and PTSD diagnosed with the revised Diagnostic Interview Schedule's PTSD module (Robins, Helzer, Cottler & Golding, 1989). They reported that, compared with individuals who did not report pain, individuals in the chronic pain group were more likely to have experienced symptoms meeting criteria for PTSD in the past year. Specifically, 10.7% of the chronic pain group was diagnosed with PTSD, compared with only 3.3% of the non-pain group. Similarly, in the revision of the NCS which applied DSM-IV criteria, the 12-month prevalence of PTSD in individuals reporting chronic spinal pain (i.e. "chronic back or neck problems", p. 332) was 7.3% (Von Korff, Crane, Lane, Miglioretti, Simon, Saunders, Stang, Brandenburg & Kessler, 2005).

Studies that have investigated the prevalence of PTSD in samples of chronic pain patients also indicate that the two conditions often co-occur. As would be expected, the reported prevalence rates have varied according to the population being studied. The

lowest prevalence rates have been reported in studies conducted with heterogeneous groups of chronic pain patients or with patient groups not referred specifically for pain problems associated with a traumatic event. Muse (1985) reported that 9.4% of 64 patients presenting to an outpatient pain clinic met his criteria for "stress-related, posttraumatic chronic pain syndrome" (p. 296). That is, 9.4% of participants had developed a posttraumatic stress condition following a traumatic incident that also initiated their pain problem. Benedikt and Kolb (1986) conducted a retrospective review of the file notes of 225 veterans referred to a pain clinic and reported that 10% met DSM-III criteria for PTSD. Aghabeigi, Feinmann and Harris (1992) used the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon & First, 1990) to assess the prevalence of PTSD in 34 patients with facial pain and found that 6% met diagnostic criteria for PTSD in relation to the same event that was associated with the onset of their pain. A larger study of 141 orofacial pain patients, which also used the SCID, reported that 11.3% had a current diagnosis of PTSD and 4.3% reported symptoms below the diagnostic cut-off (Sherman, Carlson, Wilson, Okeson & McCubbin, 2005).

Not surprisingly, rates of PTSD appear to be higher in patient groups referred specifically for pain problems associated with a potentially traumatic event. In a sample of 41 patients presenting for assessment of headaches related to head or neck injuries sustained in motor-vehicle accidents, Chibnall and Duckro (1994) diagnosed 29.3% with PTSD, and noted that another 19.5% reported levels of PTSD symptoms below the diagnostic threshold. Hickling and Blanchard (1992) assessed 20 patients referred to a psychology private practice for treatment of pain or headaches following motor-vehicle accidents, and found that the symptoms reported by half of the patients met full criteria for PTSD. They also reported that another three patients were experiencing significant symptoms that did not meet diagnostic requirements, and concluded that 65% of the

sample presented with significant levels of posttraumatic stress symptoms. Hickling, Blanchard, Silverman and Schwarz (1992a) conducted their study in the same private practice with a similar patient group referred following motor-vehicle accidents and reported that 16 out of the 20 patients (80%) reported significant PTSD symptoms (15 patients were diagnosed with PTSD and one patient reported symptoms not sufficient for the diagnosis). More recently, Sterling and Kenardy (2006) reported that 13% of a sample of 76 motor-vehicle accident victims with whiplash injuries reported moderate levels of PTSD symptoms six months post-injury.

High rates of pain and PTSD have also been reported in other groups of individuals exposed to potentially traumatic events (e.g. patients living with HIV/AIDS). Smith, Egert, Winkel and Jacobson (2002) reported that 53.8% of 145 HIV/AIDS patients reporting pain could be diagnosed with PTSD based on a cut-off score on a self-report measure of PTSD symptoms.

As far as the author is aware, only one study has investigated the prevalence of PTSD among patients with injury-related chronic pain. Asmundson, Norton, Allerdings, Norton and Larsen (1998) recruited 139 injured workers referred to a tertiary-care rehabilitation setting, of which 87% reported chronic pain due to a variety of work-related accidents and injuries. Based on a self-report measure of PTSD symptoms the investigators reported that 34.7% of the workers reported symptoms that met criteria for PTSD, and 18.2% reported symptoms consistent with "partial PTSD" (i.e. significant levels of PTSD that did not satisfy criteria for a diagnosis). Unfortunately, given that 13% of the sample did not experience chronic pain, these figures only provide an estimate of the prevalence of PTSD symptoms in relation to injuries in general, and not chronic pain specifically.

4.1.2. Prevalence of chronic pain in PTSD

In addition to the studies cited in Chapter 3 highlighting the association between PTSD and self-reported health problems there is also evidence of an elevated rate of chronic pain problems in patients presenting with PTSD. Beckham, Crawford, Feldman, Kirby, Hertzberg, Davidson and Moore (1997) reported that 80% of a sample of 129 Vietnam Veterans presenting with PTSD reported chronic pain. Amir, Kaplan, Neumann, Sharabani, Shani and Buskila (1997) compared the prevalence of fibromyalgia in 29 patients with PTSD related to a range of traumatic events and a matched control group of 37 subjects. They reported that 20% of the PTSD group (compared with 0% of the control group) reported symptoms consistent with a diagnosis of fibromyalgia.

In an Australian study, Bryant and colleagues (1999) assessed the occurrence of PTSD and chronic pain (defined as pain experienced at least once a week for six months) in 96 patients who had sustained a severe traumatic brain injury. They found that significantly more patients reporting chronic pain (37%) met criteria for PTSD compared with those who did not report chronic pain (15%). In another Australian study, Bryant and Harvey (2002) investigated PTSD in a prospective study of motor-vehicle accident survivors and found that significantly more participants with PTSD (100%) reported chronic pain compared with the participants without PTSD (56%).

Unfortunately, many of the prevalence studies cited above, particularly those conducted in chronic pain settings, are characterised by methodological problems. In particular, there has been a tendency to employ small, unrepresentative samples. For example, Hickling and colleagues (Hickling & Blanchard, 1992; Hickling et al., 1992a) studied treatment-seeking patients presenting to a private practice, while Chibnall and Duckro (1994) recruited participants through media advertisements. Furthermore, assessment methods and diagnostic criteria have varied between studies, so that it is

difficult to ascertain the exact prevalence of comorbid chronic pain and PTSD (Otis, Pincus & Keane, 2006). Despite these limitations, the research from both the chronic pain and PTSD fields suggests that a sizeable proportion of patients present to clinical settings with both conditions.

4.2. Impact of the co-occurrence of chronic pain and PTSD on pain and adjustment-related variables

The literature also indicates that the co-occurrence of chronic pain and PTSD is associated with reports of higher levels of pain, disability, and affective distress than chronic pain alone.

4.2.1. Pain severity

Several studies have reported that chronic pain patients with PTSD report higher levels of pain severity when compared with patients without PTSD. In the Geisser et al. (1996) study described in Chapter 2, patients presenting with accident-related pain were compared with patients who did not attribute their pain to an accident (No Accident group). The accident-related group was divided further into patients reporting high levels and low levels of PTSD symptoms on the basis of a median split of scores on a self-report measure of PTSD symptoms (The Posttraumatic Chronic Pain Test; Muse & Frigola, 1986). Geisser et al. reported that the Accident/High PTSD group reported significantly higher levels of pain compared with both the Accident/Low PTSD group and the No Accident group. Similar results have been noted in other chronic pain patient groups. Sherman, Turk and Okifuji (2000) and Smith et al. (2002) found that both fibromyalgia and HIV/AIDS patients reporting PTSD symptoms on self-report measures reported higher levels of pain than the patients who did not report PTSD symptoms. Only one study, Chibnall and Duckro (1994), did not find any significant differences in headache-

related variables between patients with and without a diagnosis of PTSD. However, as noted above, the sample for this study was limited to 42 patients recruited through media advertisements and this may have influenced their findings.

4.2.2. Pain-related disability and affective distress

There is also evidence that chronic pain patients reporting PTSD symptoms report higher levels of pain-related disability, and higher levels of distress when compared with patients who do not report PTSD symptoms. Two studies of fibromyalgia patients suggest that, compared with individuals who do not present with PTSD symptoms, patients who endorse PTSD symptoms on self-report measures, or who meet diagnostic criteria for PTSD using structured clinical interviews, report significantly greater levels of life interference and perceived disability due to pain (Sherman et al., 2000; Cohen, Neumann, Haiman, Matar, Press & Buskila, 2002), and are more likely to be unemployed (Cohen et al., 2002). Higher levels of pain-related disability and distress in pain patients reporting PTSD symptoms have also been reported in HIV/AIDS patients (Smith et al., 2002), in chronic pain related to motor-vehicle accidents (Duckworth & Iezzi, 2005), and in orofacial pain patients (Sherman et al., 2005).

Two studies have not found differences in pain-related disability, reporting differences between the PTSD and non-PTSD groups only on measures of distress (Chibnall & Duckro, 1994; Geisser et al., 1996). The limitations of Chibnall and Duckro's study have already been noted. Geisser and colleagues' study is of particular interest as the results shed light on the relative impact of type of onset of pain and posttraumatic stress symptoms on pain-related disability and distress. Geisser et al.'s comparisons between accident-related pain and PTSD symptom status revealed that the High PTSD/Accident group reported significantly higher scores, compared with both the

Low PTSD/Accident and No Accident groups, on self-report measures of depression and general affective disturbance. In contrast, as noted in Chapter 2, both accident groups reported higher levels of disability than the No Accident group. Although this finding has only been reported in one study it is a potentially important result as it suggests that while pain related to an accident is associated with greater disability than pain of gradual onset, it could be the additional burden of PTSD that leads to increased levels of distress.

An association between PTSD symptoms and pain-related disability and distress has also been noted in the studies investigating the prevalence of chronic pain in PTSD patients. McFarlane et al. (1994) noted that firefighters with PTSD who also complained of physical symptoms (of which musculoskeletal symptoms were the most common) were more likely to be depressed than those who did not report physical problems. They also reported that experiencing both physical symptoms and PTSD was correlated positively with the severity of PTSD symptoms, particularly intrusive symptoms. Similarly, Beckham et al. (1997) reported that PTSD symptoms (especially reexperiencing symptoms) in Vietnam Veterans were associated with higher levels of pain and pain-related disability. Asmundson, Wright and Stein (2004) found that interference due to pain and being bothered by pain over the last four weeks contributed significantly to the prediction of reexperiencing symptoms in female veterans with PTSD. These studies suggest that reexperiencing symptoms may be particularly important in understanding the impact of PTSD on adjustment to chronic pain. Finally, Amir et al. (1997) also reported that patients diagnosed with both PTSD and fibromyalgia reported more interference in quality of life and higher levels of depression and anxiety than the patients presenting only with PTSD.

Three studies have attempted to compare posttraumatic stress symptoms across different groups of chronic pain patients (Asmundson, Bonin, Frombach & Norton, 2000;

Sherman et al., 2000; Beck, Gudmundsdottir & Shipherd, 2003). These studies have used responses on the Multidimensional Pain Inventory (MPI; Kerns et al., 1985) to classify patients according to the profiles derived from Turk and Rudy's (1987; 1988) Multiaxial Assessment of Pain (MAP). The MPI is a self-report measure of chronic pain designed to assess a range of psychosocial variables, including the individual's perceptions of the pain and the degree to which pain interferes in daily activities.[1] The MAP taxonomy classifies patients into three subgroups. "Dysfunctional" patients are characterised by relatively high levels of pain severity, affective distress, and interference in activities due to pain, and low levels of perceived life control. The "Interpersonally Distressed" profile is characterised by perceptions of low levels of social support. "Adaptive Copers" report relatively low levels of pain severity, affective distress and interference in activities, and relatively high levels of perceived life control.

Applying this classification system to the sample of fibromyalgia patients described earlier, Sherman et al. (2000) reported that 85% of the PTSD group was classified as dysfunctional or interpersonally distressed, while 50% of the non-PTSD group was classified as an adaptive coper. This difference was statistically significant.

Asmundson et al. (2000) classified 155 patients presenting with chronic pain due to work-related injuries and reported that, compared with patients in the interpersonally distressed and adaptive coper groups, dysfunctional patients reported significantly higher levels of PTSD reexperiencing and avoidance symptoms. Interestingly, compared with the adaptive coper group, the dysfunctional group also reported significantly higher levels of PTSD arousal symptoms; however, there were no differences between the dysfunctional and interpersonally distressed groups on this PTSD symptom cluster. Similarly, there was no difference between the dysfunctional and interpersonally

[1] Further information regarding the MPI is provided in the Method section for Study 1 (Chapter 6).

distressed groups in the proportion of patients diagnosed with PTSD; although more patients from these two groups were diagnosed compared with patients in the adaptive coper group.

Slightly different results were obtained by Beck et al. (2003) with 85 motor-vehicle accident victims with chronic pain who were presenting for treatment of PTSD. These investigators found no differences between the dysfunctional and interpersonally distressed groups across all PTSD symptom clusters. Consistent with the other two studies, adaptive copers reported fewer PTSD symptoms, and lower levels of anxiety and depression, compared with the dysfunctional and interpersonally distressed groups. Beck et al. attributed the differences in their results and those of Asmundson et al. to differences in the samples, noting that the relationship between chronic pain and PTSD may vary with different traumatic events. This highlights the importance of investigating the relationship between the two conditions in different settings and with different patient groups. In addition, these studies demonstrate the importance of examining the relationship between pain-related variables and the different PTSD symptom clusters (Asmundson et al., 2004).

4.2.3. Summary

The studies described above have demonstrated that patients who present with both chronic pain and PTSD report higher levels of pain, pain-related disability, and affective distress than patients who present with chronic pain alone. There is also evidence that individuals with both conditions report higher levels of PTSD symptoms, and that the relationship between pain-related variables and PTSD symptom clusters may vary across different patient groups.

4.3. Potential explanations of the relationship between chronic pain and PTSD

This research has prompted numerous authors to conclude that chronic pain and PTSD are often intricately connected (Beckham et al., 1997; Sharp & Harvey, 2001; Asmundson et al., 2002; Otis, Keane & Kerns, 2003; Asmundson & Taylor, 2006; Otis et al., 2006). As Asmundson et al. (2002) clarify, there are four possible relationships between chronic pain and PTSD. Firstly, the two conditions could co-occur, but be unrelated. Secondly, one could cause the other. Alternatively, each condition could influence the other in some way; or finally, a third factor could cause both. It has been argued that the consistency of the findings in the studies described above indicate that chronic pain and PTSD are related in some way (Otis et al., 2003; Asmundson & Taylor, 2006). The following sections will review the studies and theoretical models that are beginning to shed light on the nature of the relationship between chronic pain and PTSD.

4.3.1. The aetiology of comorbid chronic pain and PTSD

Chronic pain as a stressor

A fundamental question raised by the finding that chronic pain and PTSD are commonly comorbid conditions is whether one causes the other. Specifically, could the experience of chronic pain be a traumatic stressor that leads to the development of PTSD?

As discussed in Chapter 3, the definition of a traumatic stressor has been a contentious issue. Several authors (e.g. Solomon & Canino, 1990; Avina & O'Donohue, 2002; Gold, Marx, Soler-Baillo & Sloan, 2005; Mol, Arntz, Metsemakers, Dinant, Vilters-Van Montfort & Knottnerus, 2005) have reported that many individuals develop posttraumatic stress symptoms in response to stressful events that have not traditionally been considered potentially traumatic (e.g. sexual harassment, death or illness of a loved one, and relationship, employment, or financial problems), and that as a result, the

definition of a traumatic stressor should be broadened to include any event that produces the constellation of symptoms conceptualised as PTSD. With respect to chronic pain, Schreiber and Galai-Gat (1993) described the case of a male patient who lost an eye during his military service and subsequently developed chronic headaches and PTSD symptoms. A detailed assessment of this patient revealed that his PTSD symptoms were not related to the incident in which his eye was injured. Instead, the stressor appeared to be seven hours of severe and uncontrolled pain he experienced while awaiting eye surgery. Schreiber and Galai-Gat argued that although pain did not meet the DSM-III-R definition of a traumatic stressor, it could at times be sufficiently distressing so as to lead to the development of PTSD.

Banks and Kerns (1996) highlighted numerous aspects of the chronic pain experience that are arguably unique and distinguish it as an extraordinarily stressful experience (although they were not arguing in favour of it being defined as a traumatic stressor). For example, they pointed out that pain is a noxious and aversive sensation that is intrinsically associated with affective distress, and signals of danger, threat, and potential or actual injury. As such, the challenging nature of pain is both sensory and related to its instinctive meaning. Banks and Kerns hypothesised that chronic pain is particularly stressful and demanding, not only because of the amount of aversive stimulation the individual must cope with, but also because of the anxiety and fear it can generate. They cited the common fears of chronic pain sufferers in support of this point; for example, fear that the pain will never end, fear of physical deterioration and paralysis, and fear of having an undetected, insidious disease, and noted the sense of uncertainty, helplessness, and loss of control often described by chronic pain patients. Thus, the issue arises as to whether these aspects of the chronic pain experience are consistent with the DSM-IV definition of a traumatic stressor.

Despite the points raised by Banks and Kerns, the chronic pain experience is not consistent with the type of event typically conceptualised as a traumatic stressor. Although chronic pain is sometimes perceived as uncontrollable and unpredictable, and it is arguable that it is sometimes perceived as a threat to the individual's physical integrity, it is clearly not life-threatening in its nature as are other medical conditions which have been included in the DSM-IV definition (such as cancer or myocardial infarction). Also, while chronic pain is often characterised by similar cognitive, emotional, and behavioural features as responses to traumatic events (e.g. perceptions of uncontrollability and helplessness, feelings of anxiety, and avoidance behaviour), it does not necessarily follow that it is a traumatic stressor as there may be other explanations for these similarities. Several authors (McNally, 2003a; b; Monson, Stevens & Schnurr, 2004; Weathers & Keane, 2007) have cautioned against excessive broadening of the definition of a traumatic stressor (what McNally, 2003a refers to as "conceptual bracket creep") on the basis that it will dilute the initial intent of the PTSD diagnosis, and hinder important conceptual and theoretical research. Although this view has been disputed (e.g. Brewin, 2003; Taylor, 2006), there is limited empirical support for the notion that chronic pain is a traumatic stressor.

A more parsimonious explanation for the role that chronic pain may have in the development of PTSD is that the experience of chronic pain could be considered a secondary stressor according to Shalev's (2002) conceptualisation (as described in Chapter 3). That is, an individual who is injured in the context of a traumatic event and who subsequently develops a chronic pain condition is likely to experience a range of additional stressors, including strained relationships, loss of employment and financial problems, and potentially invalidating experiences in the medical system (Banks & Kerns, 1996). As Shalev (2002) has argued, these secondary stressors contribute to the

experience of the injury as traumatic and may contribute to the development of PTSD. Consistent with this, several studies have revealed that stressful life events, including persisting pain and other medical problems, experienced after a traumatic event are significant predictors of the development of PTSD and PTSD symptom severity (e.g. King, King, Fairbank, Keane & Adams, 1998b; Maes, Mylle, Delmeire & Janca, 2001), particularly in groups of injured trauma survivors (e.g. Ehlers et al., 1998; Bryant & Harvey, 2002; Carty, O'Donnell & Creamer, 2006).

Shared vulnerability model

The shared vulnerability model proposed by Asmundson and colleagues (Asmundson et al., 2002; Asmundson & Taylor, 2006) ascribes the concept of anxiety sensitivity a central role in the development of comorbid chronic pain and PTSD. The model asserts that individuals high in anxiety sensitivity are particularly vulnerable to developing both chronic pain and PTSD in response to a traumatic event that leads to physical injury.

The concept of anxiety sensitivity originated from the expectancy model of fear (Reiss & McNally, 1985; Reiss, 1991), and refers to individual differences in the fear of anxiety (Reiss & McNally, 1985). Reiss and McNally proposed that anxiety sensitivity consists of a set of learned beliefs about the consequences of anxiety; in particular, beliefs that anxiety symptoms have negative physical, social, or psychological consequences. For example, an individual high in anxiety sensitivity may believe that a rapid heart beat indicates a heart attack, or that dizziness is a sign of an imminent stroke. Anxiety sensitivity is conceptualised as a dispositional variable that signifies a tendency to respond with fear when anxiety-related symptoms are experienced (McNally, 1999). Accordingly, anxiety sensitivity is considered to be a cognitive risk factor for the

development of anxiety disorders by predisposing individuals to respond fearfully to bodily sensations, thus augmenting anxious and fearful responses to potentially anxiety-provoking stimuli (Reiss & McNally, 1985; McNally, 1999). Evidence for an association between anxiety sensitivity and anxiety disorders comes from investigations of levels of anxiety sensitivity across different diagnostic groups. When compared with non-clinical samples, elevated levels of anxiety sensitivity have consistently been reported in patients with panic disorder with and without agoraphobia (McNally & Lorenz, 1987; Taylor, Koch & McNally, 1992; Cox, Parker & Swinson, 1996), social phobia (Orsillo, Lilienfeld & Heimberg, 1994; Hazen, Walker & Stein, 1995), and generalised anxiety disorder (Taylor et al., 1992; Sandin, Chorot & McNally, 1996). Anxiety sensitivity is measured by the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1993) and an expanded version developed by Taylor and Cox (Anxiety Sensitivity Scale - Revised, ASI-R; 1998) to address some of the limitations of the original scale.

Taylor and colleagues (Taylor et al., 1992; Taylor, Rabian & Fedoroff, 1999; Taylor, 2003) have argued that anxiety sensitivity also plays an important role in PTSD, and the results of two studies indicate that when compared with other anxiety disorder groups and controls, anxiety sensitivity is elevated in PTSD patients (Taylor et al., 1992; Lang, Kennedy & Stein, 2002). According to Taylor (2003), anxiety sensitivity contributes to the development of PTSD by increasing the intensity of the individual's response to the traumatic stressor. Preliminary support for the role of anxiety sensitivity in the development of PTSD can be found in a prospective study of pregnant women which found that pre-natal anxiety sensitivity was significantly associated with PTSD symptoms following childbirth, even when other postnatal psychological symptoms (e.g. depression) were taken into account (Keogh, Ayers & Francis, 2002). The existing research also supports the notion that anxiety sensitivity plays a role in the maintenance

of PTSD. As Ehlers and Clark's (2000) cognitive model of PTSD predicts, individuals with PTSD endorse items on the ASI that indicate a tendency to be fearful of internal cognitive phenomena (Cox, Borger & Enns, 1999), and these items have been found to be predictive of PTSD symptom severity in a study of women exposed to intimate partner violence (Lang et al., 2002). Furthermore, Federoff and colleagues (2000) reported that in a sample of motor-vehicle accident victims with PTSD, anxiety sensitivity was a significant predictor of PTSD symptom severity, and that a reduction in anxiety sensitivity following CBT for PTSD was related to a reduction in PTSD symptoms.

Anxiety sensitivity has also been implicated in the development of fear of pain and pain-related avoidance in chronic pain (Keogh & Asmundson, 2004). Several studies have revealed an association between anxiety sensitivity and poor adjustment to chronic pain in a range of chronic pain samples, including chronic back pain, patients with recurring headaches, and samples of heterogeneous chronic pain patients (e.g. Asmundson & Norton, 1995; Plehn, Peterson & Williams, 1998; Asmundson, Norton & Veloso, 1999). These studies have also indicated that anxiety sensitivity is related to fear of pain and pain-related avoidance, and evidence from other studies (e.g. Asmundson & Taylor, 1996; Zvolensky, Goodie, McNeil, Sperry & Sorrell, 2001; Greenberg & Burns, 2003; Norton & Asmundson, 2004) support Asmundson and Taylor's (1996) prediction that anxiety sensitivity may actually mediate the relationship between fear of pain and pain-related escape/avoidance behaviour, thereby contributing to ongoing disability following injury (Keogh & Asmundson, 2004).

These converging lines of evidence from the chronic pain and PTSD fields point to a role for anxiety sensitivity as the link between the two conditions (Asmundson et al., 2002). The shared vulnerability model predicts that in the case of a stressor that could potentially lead to the development of both chronic pain and PTSD the individual is more

likely to develop both conditions because they are likely to respond fearfully to both the pain associated with the injury and the arousal symptoms experienced during and following the event (Asmundson et al., 2002). The research provides preliminary evidence of an interaction between anxiety sensitivity, PTSD, and chronic pain. Two studies indicate that higher levels of anxiety sensitivity are reported by individuals presenting with both chronic pain and PTSD, and that anxiety sensitivity is associated with pain-related dysfunction and PTSD symptom severity in this group of patients (e.g. Asmundson et al., 1998; Asmundson et al., 2000).

However, the studies are cross-sectional in design and consequently, do not provide proof of the "shared vulnerability" model of chronic pain and PTSD because in the absence of prospective studies it is not possible to confirm that the anxiety sensitivity actually predated the chronic pain and PTSD (Asmundson & Taylor, 2006; Asmundson, Abrams & Collimore, 2008). The only prospective study to date that has included pain-related variables, anxiety sensitivity, and PTSD symptoms assessed these variables in a sample of 134 chronic pain patients prior to the patients undergoing general surgery (Martin, Dzyuba, Halket, Asmundson & Katz, 2007). PTSD symptoms contributed 6% of unique variance to post-surgical pain-related disability scores after controlling for anxiety sensitivity, fear of pain, catastrophising, escape/avoidance behaviour, and pain intensity prior to surgery. Anxiety sensitivity was not a significant predictor in the model, but the investigators noted that it could have influenced disability indirectly by influencing escape/avoidance behaviour. Although the prospective design of this study is an important methodological advantage over previous research, and it suggests that PTSD symptoms influence pain-related disability, it does not provide much support for the shared vulnerability model. Furthermore, the participants in the study already had chronic

pain, and so the analyses do not shed light on the role of these variables in the development of comorbid pain and PTSD following a traumatic stressor.

The relationships evident in the cross-sectional studies investigating the relationship between anxiety sensitivity, PTSD symptoms, and pain-related variables can also be explained by conceptualising anxiety sensitivity as contributing to the maintenance of chronic pain and PTSD. That is, instead of being involved in the development of chronic pain and PTSD, it is possible that anxiety sensitivity increases as a result of the co-occurrence of the two conditions, and becomes a factor involved in their maintenance (Asmundson et al., 2008). Anxiety sensitivity could potentially do so by exacerbating the individual's inclination to misinterpret both pain and the arousal symptoms related to PTSD (Sharp & Harvey, 2001; Sharp, 2004; Asmundson & Taylor, 2006). This possibility will be discussed in further detail in Section 4.3.2. in the context of Sharp and Harvey's (2001) mutual maintenance model of the relationship between chronic pain and PTSD.

Triple vulnerability model

The triple vulnerability model (Otis et al., 2003; Otis et al., 2006) is another attempt to explain the co-occurrence of chronic pain and PTSD from the perspective of an underlying predisposition to develop both conditions. It is based on Keane and Barlow's (2002) model of PTSD outlined in Chapter 3. Applied to chronic pain, the model claims that the development of a chronic pain condition is preceded by the general biological vulnerability, the individual's belief that their pain is uncontrollable and unpredictable (i.e. the general psychological vulnerability), and a lowered threshold for physiological arousal in response to an alarm. These vulnerabilities are said to contribute to low self-efficacy, decreased expectations of being able to cope effectively with pain in the future,

and negative affect, prompting the individual to avoid situations or activities that they believe will elicit their pain. In turn, avoidance contributes to further deterioration in self-efficacy and perceptions of control, leading to increasing disability over the longer term.

As reviewed in Chapter 1, there is substantial evidence that cognitive variables, such as perceptions of control and self-efficacy, play a crucial role in influencing adjustment to chronic pain, and fear-avoidance is central to contemporary models of pain-related disability. Although the triple vulnerability model applies Keane and Barlow's terminology to these concepts (e.g. referring to the development of true or false alarms), it does not offer additional explanatory power beyond existing and more established accounts of the development of pain-related disability and distress (i.e. cognitive-behavioural models). Furthermore, given that the model was forwarded in the context of an attempt to explain the relationship between chronic pain and PTSD, the main weakness of Otis and colleagues' model is that it is essentially a model of the development of chronic pain only, and does not explicate any links between chronic pain and PTSD.

The shared vulnerability and triple vulnerability models are both consistent with Turk's (2002a) diathesis-stress model of chronic pain and disability following traumatic injury (Asmundson & Taylor, 2006). Although Turk's model is predominantly concerned with accounting for the development of pain-related disability, it focuses on the onset of pain following an accident or physical injury and shares features with the other two models discussed in this section. Turk also discussed pre-existing vulnerabilities, proposing that they interact with behavioural and cognitive processes to influence adjustment to the instigating event. Like the other two models, anxiety sensitivity is identified as a key dispositional variable; that is, individuals are at risk of developing ongoing pain and disability if they have a tendency to respond to stressors with fear. In

particular, the model asserts that anxiety sensitivity predisposes individuals to be hypervigilant for pain and other atypical sensations, and to interpret pain and other sensations as abnormal or harmful. These attentional processes and interpretations of physical sensations interact with other cognitive processes (anticipation of pain and injury, catastrophising, and self-efficacy) to contribute to avoidance behaviour and therefore, disability.

The conceptual similarities between all of these models suggest that the notion of an underlying vulnerability that is activated by a traumatic stressor could be a worthwhile avenue for further investigation through prospective studies. In the absence of such studies the evidence supporting the diathesis aspect of these models is limited. It does appear, however, that anxiety sensitivity could be an important individual difference factor important in the relationship between chronic pain and PTSD, whether as a dispositional variable, maintaining variable, or both.

4.3.2. The mutual maintenance model

The mutual maintenance model (Sharp & Harvey, 2001; Sharp, 2004) is concerned with elucidating the interaction between co-occurring chronic pain and PTSD. The model proposes that certain features of chronic pain maintain or exacerbate the symptoms of PTSD and vice versa, and specifies the cognitive, physiological and behavioural processes by which disability and distress are maintained when the two conditions occur together. The evidence for the role of the mechanisms advanced by Sharp and Harvey's (2001) model, and those forwarded by other researchers, is examined in the following sections.

Pain as a reminder of trauma

It has been hypothesised that when the onset of pain and the traumatic event coincide pain may become a reminder of the traumatic event (Sharp & Harvey, 2001). If the individual attempts to avoid situations or activities that trigger the pain in order to avoid the distress and arousal associated with memories of the trauma this is likely to lead to ongoing disability, as well as maintenance of the posttraumatic stress response (Sharp & Harvey, 2001). McFarlane et al's (1994), Beckham et al's (1997) and Asmundson et al.'s (2004) findings that the intrusive symptoms of PTSD are positively correlated with reports of severity of pain and pain-related disability provide preliminary support for the notion that these two variables are associated with the intrusive symptoms of PTSD.

In addition to pain potentially serving as a reminder of the trauma, it has also been suggested that in some circumstances pain experienced during a traumatic event may be re-experienced as a "somatic flashback" (Asmundson et al., 2002; Salomons, Osterman, Gagliese & Katz, 2004). Salomons et al. have presented two case studies of women who regained consciousness while under general anaesthesia and subsequently developed PTSD in response to the experience. They reported that in both cases the PTSD symptoms included pain similar in quality and location to the pain the women had experienced when they gained awareness during the surgery. Interestingly, these pain sensations were able to be triggered by stimuli associated with the trauma (e.g. blue scrub suits one of the patients had seen during the surgery). Stimulus generalisation had also occurred in this case, with other blue objects eventually eliciting the pain, suggesting a conditioned response (Salomons et al., 2004).

For pain to serve as a reminder of the traumatic event or to be experienced as a somatic flashback it must be associated with the traumatic event in some way.

Unfortunately, as will be discussed below, many of the studies investigating the relationship between chronic pain and PTSD do not consistently link traumatic events, onset of pain, and posttraumatic stress symptoms so that temporal associations are often hard to establish. Asmundson and colleagues (2002) argued that examination of the temporal relationship between pain and PTSD is essential for a more complete understanding of the potential contribution of pain to the re-experiencing of a traumatic event and recommended that future research address this issue.

Attentional biases

The empirical work described in earlier chapters suggests that attentional biases have been observed in both chronic pain and PTSD (Sharp & Harvey, 2001; Asmundson et al., 2002; Otis et al., 2003; Asmundson & Taylor, 2006). Accordingly, if pain does become a reminder of the trauma individuals presenting with both problems should exhibit an attentional bias towards both pain-related and trauma-related stimuli (Sharp & Harvey, 2001). An attentional bias towards pain-related stimuli (because they are ostensibly a reminder of the trauma) has been identified as one way in which PTSD may contribute to a focus on pain sensations, thereby amplifying the pain experience (Bryant et al., 1999; Sharp & Harvey, 2001).

Only one study to date (Beck, Freeman, Shipherd, Hamblen & Lackner, 2001) has investigated the hypothesis that individuals with both pain and PTSD will exhibit an attentional bias towards both types of stimuli. These researchers used a modified Stroop paradigm to explore information processing biases in three groups of MVA victims: (1) individuals with both PTSD and pain; (2) individuals with pain only; and (3) individuals without pain or any psychiatric condition. As predicted by Sharp and Harvey (2001), the results revealed that the PTSD and pain group showed a significant processing bias

towards both accident and pain-related stimuli compared with the other two groups, while the pain only group demonstrated a bias only towards pain-related stimuli (Beck et al., 2001). It is important to note that it is not clear from this study whether the comorbid pain and PTSD group exhibited a bias towards pain-related stimuli due to their experience of pain, or because the pain served as a reminder of trauma. Also, as the researchers acknowledged, the study would have been improved by the inclusion of a group of subjects with PTSD but no pain. These issues aside, the study does provide some preliminary support for the presence of an attentional bias in individuals with chronic pain and PTSD.

Anxiety and pain perception

There is now considerable evidence that anxiety influences the experience of pain (Vlaeyen & Linton, 2000). As many have observed, since anxiety is a core feature of PTSD the occurrence of PTSD in the context of pain could adversely impact on pain perception, including pain threshold and tolerance (Bryant et al., 1999; Sharp & Harvey, 2001; Asmundson et al., 2002). Although this specific hypothesis has not been examined, there is evidence from prospective studies that acute posttraumatic stress symptoms are significant predictors of the maintenance of pain symptoms in whiplash injuries following motor-vehicle accidents (Drottning, Staff, Levin & Malt, 1995; Sterling, Kenardy, Jull & Vicenzino, 2003; Sterling, Jull, Vicenzino, Kenardy & Darnell, 2005). Drottning et al. (1995) reported that high levels of acute posttraumatic stress symptoms in the hours following the motor-vehicle accident were significantly associated with ongoing pain symptoms four weeks later. Sterling et al. (2005) found that acute posttraumatic stress symptoms assessed within the first month following the motor-vehicle accident were a significant predictor of the persistence of pain symptoms six months post-injury.

Similarly, Sterling et al. (2003) reported that whiplash patients with moderate to severe symptoms at six months were distinguished from patients who had recovered or who were only experiencing mild symptoms by higher levels of posttraumatic stress symptoms, both initially and throughout the follow-up period. This complex clinical picture (i.e. physical and psychological dysfunction) continued up to 2-3 years post-injury (Sterling, Jull & Kenardy, 2006). These studies suggest that heightened anxiety in the early stages following a traumatic injury may contribute to the development of chronic pain.

Impact on cognitive functioning

Another way in which PTSD has been thought to exacerbate the pain experience is by impacting upon the ability to effectively apply pain coping strategies (Bryant et al., 1999; Sharp & Harvey, 2001). Sharp and Harvey noted that both chronic pain and PTSD are characterised by symptoms that place considerable cognitive demands on individuals (e.g. re-experiencing symptoms in PTSD and catastrophising in chronic pain). Bryant and colleagues suggested that this may limit the cognitive capacity available to employ effective coping strategies.

Thomas, Iezzi, Duckworth, Archibald and Klinck (2000) investigated the impact of PTSD symptoms on the cognitive functions commonly reported as being a problem by chronic pain patients (e.g. memory and concentration). After controlling for education and pain severity they found that PTSD symptoms accounted for significant variance in attention, concentration, and memory amongst chronic pain patients. Importantly, they also reported that this relationship was mediated by general levels of daily activity, indicating that patients who remained active despite pain and high levels of PTSD symptoms were less likely to exhibit cognitive deficits. Importantly, the researchers

pointed out that levels of daily activity may have been influenced by depression, and were unable to exclude the possibility that depressive symptoms influenced the relationship between PTSD symptoms and cognitive functioning.

The role of depression

In addition to potentially influencing cognitive functioning, depression has been forwarded as a factor that may contribute to ongoing disability when chronic pain and PTSD co-occur through decreases in activity levels due to fatigue and lethargy (Sharp & Harvey, 2001). The results of several studies confirm that it could be important to examine the interaction between chronic pain, PTSD and depression. Asmundson et al. (1998) reported that in their sample of injured workers depressive symptoms were the only significant predictor of PTSD symptom frequency. Another study conducted with fibromyalgia patients indicated that the relationship between fibromyalgia symptoms and PTSD was mediated by a diagnosis of depression (Roy-Byrne, Smith, Goldberg, Afari & Buchwald, 2004). Alternatively, Bryant et al. (1999) reported that the relationship between pain severity and depression was mediated by the severity of PTSD in patients with a traumatic brain injury. Collectively, these studies all suggest that there is interplay between pain, PTSD, and depression following traumatic injury, and further research is required to clarify this relationship.

Coping style

It is apparent from the literature reviewed in previous chapters that avoidance has been implicated in the maintenance of both chronic pain and PTSD. Consequently, a number of investigators have predicted that patients with chronic pain and PTSD exhibit an inclination towards an avoidant coping style, and have postulated that this places these patients at risk of becoming disabled and distressed in response to a traumatic injury

(Bryant et al., 1999; Sharp & Harvey, 2001; Asmundson et al., 2002; Otis et al., 2003). Consistent with this, Bryant and colleagues reported that when the effects of PTSD severity were controlled for, the only predictor of pain severity in patients with a traumatic brain injury and PTSD was an avoidant coping style. In their prospective study of whiplash patients Sterling et al. (2003) found that the responses of the moderate/severe whiplash group on a measure of posttraumatic stress symptoms (Impact of Events Scale, IES; Horowitz, Wilner & Alvarez, 1979) indicated a persistence of an avoidant response to the trauma throughout the six month follow-up period.

Other cognitive variables which have been highlighted as being important in both chronic pain and PTSD are catastrophising and perceptions of life control (Bryant et al., 1999; Palyo & Beck, 2005). At this stage only the latter variable has been investigated.

Palyo and Beck (2005) applied structural equation modeling to examine the relationship between PTSD symptoms, pain severity, and perceived life control in a sample of 183 motor-vehicle accident victims with chronic pain and PTSD. The investigators found support for their hypothesis that perceptions of life control mediated the relationship between pain and PTSD, and impairment in psychosocial and physical functioning. Interestingly, this study also revealed that pain and PTSD symptom severity were associated with different aspects of posttrauma functioning. The investigators reported that while both PTSD symptomatology and pain severity were related to psychosocial impairment, after controlling for the relationship between PTSD and pain, only pain was associated with impairments in physical functioning. Importantly, Paylo and Beck's research illustrates a point made by Asmundson and colleagues (2002) regarding the relationship between chronic pain and PTSD. These authors pointed out that considering the multidimensional nature of both conditions it is plausible that not all variables associated with both problems will be causally related in some way.

Anxiety sensitivity

In addition to being forwarded as a vulnerability factor, anxiety sensitivity has also been identified as potentially contributing to the maintenance of chronic pain and PTSD when they co-occur (Sharp & Harvey, 2001; Asmundson et al., 2002; Sharp, 2004; Asmundson & Taylor, 2006). As Asmundson and colleagues have argued, it is important to distinguish between anxiety sensitivity as a dispositional variable and as a maintaining factor, and the two are not necessarily mutually exclusive. That is, it is possible that anxiety sensitivity can act as a dispositional variable that places individuals at risk of responding ineffectively to traumatic events, and then serve as a maintaining factor once the relevant condition has developed by exacerbating the individual's inclination to misinterpret both pain and the arousal symptoms related to PTSD. This dual role can be found in theoretical views of the role of anxiety sensitivity in panic disorder and PTSD (e.g. Reiss, 1991; Taylor, 2003), and may help to account for the apparent interaction between anxiety sensitivity, chronic pain, and PTSD in the studies described in Section 4.3.1 above.

A PTSD symptom that may be closely linked to anxiety sensitivity is hypervigilance (Asmundson & Taylor, 2006). As Turk (2002a) has pointed out in the context of the diathesis-stress model discussed above, anxiety sensitivity predisposes individuals to be hypervigilant for pain and other atypical sensations. Consistent with this, anxiety sensitivity has been shown to be related to vigilance for bodily sensations (Schmidt, Lerew & Trakowski, 1997; Zvolensky & Forsyth, 2002). In addition, McFarlane, Weber and Clark (1993) have demonstrated that some individuals with PTSD exhibit difficulties evaluating the significance of stimulus change, and consequently, have an impaired ability to identify relevant information. McFarlane and colleagues (1993; 1994) have claimed that this may lead the individual to focus on, and misinterpret the

meaning of, physical symptoms. Accordingly, the hypervigilance experienced as part of PTSD may contribute to the maintenance of chronic pain and PTSD by increasing the individual's chances of detecting subtle and benign bodily sensations (Asmundson & Taylor, 2006).

There is growing support for the hypothesis that hypervigilance is an important aspect of the interaction between chronic pain and PTSD. Firstly, Asmundson et al. (2004) found that interference in daily activities due to pain accounted for the largest portion of the variance in hyperarousal symptoms in a sample of female veterans with PTSD. Secondly, Asmundson, Wright, McCreary and Pedlar (2003) conducted a factor analytic study in which they compared over 700 United Nations peacekeepers with and without chronic pain. Two models of PTSD symptoms provided a good fit to the data for both groups: (1) four interrelated factors of reexperiencing, avoidance, numbing, and hyperarousal; and (2) a hierarchical 2-factor model in which the two lower-order factors were intrusion-avoidance and numbing-hyperarousal. Importantly for the current discussion, the final models for the pain and no pain group contained statistically significant different factor loadings for the following symptoms: (1) physical reactions to reminders of the trauma; (2) emotional numbing; (3) a sense of foreshortened future; and (4) hypervigilance. Asmundson et al. noted that although the differences were statistically significant, only the difference on the hypervigilance symptom was of practical significance. They interpreted this finding as an indication that chronic pain may exacerbate PTSD symptoms by heightening hypervigilance. Finally, Buitenhuis, de Jong, Jaspers and Groothoff (2006) found that the hyperarousal symptom cluster of PTSD as assessed in the acute stage of whiplash injury was predictive of persistence of the whiplash complaint six and 12 months after the motor-vehicle accident. Although these studies indicate hyperarousal may be important in co-occurring chronic pain and PTSD

the mechanisms underlying this possible link are yet to be identified (Asmundson et al., 2002; Asmundson et al., 2003; Asmundson & Taylor, 2006). Nevertheless, Asmundson et al. (2008) speculated that chronic arousal (and possibly autonomic nervous system dysregulation) may be a feature shared by chronic pain and PTSD.

4.3.3. Summary

Several models have been proposed to account for the relationship between chronic pain and PTSD. Some of these theoretical accounts are concerned with explaining the aetiology of comorbid chronic pain and PTSD, while others focus on the interplay between the two conditions when they co-occur. Although there is preliminary support for many of the hypotheses generated by these models, overall the research is in its early stages. In addition, there are a number of methodological issues which need to be addressed in future research. These will be discussed in the following section.

4.4. Limitations of the research on chronic pain and PTSD

It is important to point out that the existing chronic pain/PTSD literature is limited by several methodological issues that are important to consider when interpreting the purported relationship between chronic pain and PTSD.

Firstly, as already noted, some studies (particularly earlier research) employ small samples which are not necessarily representative of chronic pain patients in general. Also, as many investigators have acknowledged (e.g. Geisser et al., 1996; Beckham et al., 1997; Sherman et al., 2000) all of the research conducted to date has been cross-sectional in design, which does not permit conclusions regarding causation.

Perhaps the most important problems with the literature concern the methods used to assess PTSD in samples of chronic pain patients. These problems include: (1) the use of self-report measures of posttraumatic stress symptoms which have not been adequately

validated in a chronic pain context; (2) the posttraumatic stress symptoms reported by subjects are not always linked to a specific event, and in particular, are not always linked to the event associated with the onset of the pain; (3) the degree to which the event was experienced as traumatic is typically not assessed; and (4) the apparent overlap between some symptoms of PTSD and the problems commonly associated with chronic pain has not been adequately addressed.

4.4.1. Self-report measures of posttraumatic stress symptoms

While the studies conducted in PTSD treatment settings (e.g. Beckham et al., 1997; Beck et al., 2003; Palyo & Beck, 2005) have been more likely to use well-researched structured clinical interviews, such as the Clinician Administered PTSD Scale (CAPS; Blake, Weathers, Nagy, Kaloupek, Gusman, Charney & Keane, 1995), to determine PTSD diagnostic status almost all of the studies conducted in chronic pain settings have used interviews conducted in clinical settings (e.g. Muse, 1985; Hickling et al., 1992; Chibnall and Duckro, 1994), or self-report measures of posttraumatic stress symptoms (e.g. Geisser et al., 1996; Sherman et al., 2000). The only exceptions to this include the epidemiological surveys already described (McWilliams et al., 2003; Von Korff et al., 2005; Demyttenaere et al., 2007), and the studies of Aghabeigi et al. (1992), Cohen et al. (2002) and Sherman et al. (2005). The main problem with the self-report measures used to date is that none of the questionnaires have been adequately validated for use with chronic pain patients.

Geisser and colleagues (1996) used the Posttraumatic Chronic Pain Test (PCPT; Muse & Frigola, 1986), a self-report measure consisting of only six items answered in a true-false format that was initially developed as a quick screening instrument for use in clinical settings. Although the original article by Muse and Frigola claims that the

measure has good psychometric properties and adequately identifies chronic pain patients at risk of posttraumatic stress, this evaluation of the instrument was conducted using a sample size of 20 subjects, and the authors acknowledged that the measure required further validation. Geisser et al. noted that they determined that the PCPT had good internal consistency using the responses of 91 subjects from their study. This brief report and Muse and Frigola's initial evaluation cannot be considered a thorough psychometric evaluation of the instrument. Furthermore, the PCPT was only designed to be a brief screening questionnaire, and thus in six items it does not cover all of the symptoms associated with PTSD. Finally, Geisser et al. changed the true-false format to a 7-point Likert scale, and then used a median split to categorise their subjects into the high and low PTSD symptom groups. Without appropriate normative data it is difficult to interpret the scores reported for the different PTSD symptom groups.

Similar concerns can also be expressed regarding the self-report measure used by Sherman et al. (2000) in their study of fibromyalgia patients. The measure used in this study was the Crime-Related (CR) PTSD scale, an empirically derived measure based on the Symptom Checklist-90-Revised (SCL-90R; Saunders, Arata & Kilpatrick, 1990). As Sherman et al. noted, while the SCL-90R has been widely used and studied in medical settings the PTSD subscale is a relatively recent addition. Furthermore, it has been argued that the precision of the scale as a measure of posttraumatic stress remains uncertain (Norris & Hamblen, 2004). Sherman and colleagues cite their own unpublished data in support of the subscale's validity and diagnostic utility with temporomandibular disorder patients; however, this is appears to be the only information available about the performance of the measure in a chronic pain setting.

While Geisser et al. and Sherman et al. did not attempt to obtain a diagnosis of PTSD using these self-report measures and instead focused on posttraumatic stress

symptoms, Asmundson and colleagues (Asmundson et al., 1998; Asmundson et al., 2000) and Smith et al. (2002) both used self-report measures of PTSD to obtain a diagnosis by applying a cut-off score or diagnostic algorithm derived from research in other PTSD populations. Although reliance on self-report measures is not considered the optimal method for making diagnostic judgements, the use of empirically validated scoring guidelines can be a useful method for classifying subjects into broad diagnostic groups (Norris & Hamblen, 2004). Nonetheless, the validity of the measures and accompanying diagnostic guidelines applied in these three studies has not been adequately investigated in a chronic pain population.

Specifically, Asmundson and colleagues used the Modified PTSD Symptom Scale (MPSS; Falsetti, Resnick, Resick & Kilpatrick, 1993), a self-report measure of the frequency and severity of PTSD symptoms over the past week. Asmundson and colleagues applied the diagnostic algorithm recommended by the scale's original authors, and stated that the diagnostic utility of this scoring method has been confirmed in community and clinical samples. Given the apparent overlap between symptoms of PTSD and the problems commonly associated with chronic pain (see section 4.4.4. below) it is not appropriate to assume that diagnostic scoring guidelines obtained in one trauma group are applicable in all groups. This has particularly proved to be the case in the assessment of PTSD symptoms, with authors noting that different measures are suitable for different settings, and that interpretations of scores on self-report measures vary across different trauma groups (Carlson, 1997; Norris & Hamblen, 2004).

Smith et al. (2002) also applied a cut-off score that has not been derived from research in chronic pain samples, this time with the PTSD Checklist (Weathers et al., 1993), a self-report measure of posttraumatic stress symptoms based on the DSM-IV criteria for PTSD. Unlike the MPSS, the PCL has been used in another study in a chronic

pain setting (Sherman et al., 2005). This study was conducted with a sample of 141 orofacial pain patients, and indicated that when compared with PTSD diagnoses obtained using the SCID, the recommended cut-off score on the PCL accurately classified 89% of patients with a sensitivity (i.e. the chance that a condition that is present will be detected) of 0.82, and specificity (i.e. the chance that a condition that is not present will be found to be absent) of 0.92. Although promising, further validation of the PCL in larger and more general chronic pain samples is still required. This was the aim of the study presented in Chapter 6 of this thesis.

Finally, given that the self-report measures only obtain information about PTSD symptoms and not the traumatic event in question the use of self-report measures to obtain a diagnosis of PTSD must be accompanied by a thorough assessment of the events themselves. As argued in the next two sections, this is often not the case in the research on chronic pain and PTSD.

4.4.2. Association of symptoms to specific traumatic events

The second problem associated with the research on chronic pain and PTSD concerns the relationship between the traumatic events, pain, and posttraumatic stress symptoms reported by the subjects. Beckham et al. (1997), Amir et al. (1997), Cohen et al. (2002) and Sherman et al. (2005) identified the traumatic event associated with the PTSD symptoms reported by their subjects, but did not determine if the pain reported by the subjects was actually related to that traumatic event or another incident (or no incident in the case of spontaneous or gradual onset). The studies conducted in chronic pain settings have sometimes identified the event associated with the onset of pain but have not always determined if the PTSD symptoms reported by the subjects are related to that incident or other potentially traumatic events (e.g. Benedikt & Kolb, 1986; Smith et al.,

2002). Asmundson et al. (2004) measured PTSD symptoms using the PCL-C and did not enquire about the event(s) associated with either the PTSD symptoms or the pain reported by their participants.

The study by Sherman and colleagues (2000) effectively illustrates the interpretative problems that this issue raises. The authors reported that 84% of the participants provided data regarding the onset of their fibromyalgia symptoms. From this sub-group of patients, 49% of the participants reporting PTSD symptoms reported that an injury preceded the development of their symptoms. This figure suggests that at least half of the patients reporting PTSD symptoms were reporting symptoms associated with events not related to the onset of their pain. Consequently, it is not possible to determine the degree to which the relationships between pain and PTSD found in the study were due to an interaction between pain and PTSD related to the same event, or were due to an interaction between pain and PTSD due to prior (or subsequent) traumatic events.

It could still prove instructive to obtain an understanding of the interaction between pain and PTSD symptoms regardless of whether a temporal connection between the two conditions exists. It is possible that whatever the cause of either problem, the co-occurrence of pain and PTSD may still be associated with greater problems with adjustment. For example, Young and Yehuda (2006) have claimed that PTSD is exacerbated by comorbid conditions, even if the comorbid condition developed first. From a clinical perspective, identifying chronic pain patients with PTSD is important regardless of the event that instigated the posttraumatic stress symptoms. Nevertheless, establishing the temporal connections between chronic pain and PTSD symptoms is important for identifying the processes that may place individuals at risk of developing chronic problems in response to a potentially traumatic event. Asmundson and colleagues (2002) have also argued that attention to temporal associations could aid in clarifying the

interactions between the two disorders once they develop. For example, when the pain and PTSD have developed in response to different events this may alter the interaction between the two disorders and different maintaining mechanisms may be involved to when the two have developed in the context of the same event.

The studies presented in Chapters 6-8 of this thesis have attempted to overcome this methodological problem by linking the onset of the participants' chronic pain with the posttraumatic stress symptoms they report. Further details of how this was achieved are provided in Chapter 6.

4.4.3. Evaluation of the nature of the events reported

Although some studies have evaluated the events reported by subjects to ensure that they qualify as traumatic events (e.g. Cohen et al., 2002; Smith et al., 2002; Beck et al., 2003), many studies have not described doing so. Several of the studies that have used self-report measures of PTSD (e.g. Geisser et al., 1996; Asmundson et al., 1998; Asmundson et al., 2000; Sherman et al., 2000) have not attempted to confirm that the events are traumatic according to the posttraumatic stress literature or formal diagnostic criteria. As Sherman and colleagues (2000) recognised, this prevents confirmation that the symptoms being reported are actually related to a traumatic event. Considering current diagnostic criteria for PTSD it is important to obtain information about the nature of the events reported by subjects in PTSD-related research to ensure that the symptoms being studied are actually responses to traumatic events and not generally stressful experiences. Given that some of the events associated with the onset of chronic pain could not be considered traumatic events, particular attention was paid to this issue in the second and third studies that were conducted for this thesis. Further details are provided in the Method section of Chapter 7.

4.4.4. Overlap between chronic pain and PTSD

Some overlap between the features of chronic pain and PTSD is apparent when diagnostic criteria and descriptions of the two disorders are briefly surveyed. Problems with anxiety and increased physiological arousal, avoidance behaviour, and increased attention to physical symptoms are prevalent in both chronic pain and PTSD (Asmundson et al., 2002). Many of the difficulties frequently described by chronic pain patients, such as disturbed sleep (e.g. Pilowsky, Crettenden & Townley, 1985; Morin, Gibson & Wade, 1998), problems with anger and irritability (Fernandez & Turk, 1995; Okifuji, Turk & Curran, 1999), and difficulty concentrating (Jamison, Sbrocco & Parris, 1988) are all diagnostic features of PTSD.

This symptom overlap is an important issue to consider when interpreting the responses of chronic pain patients on self-report measures of PTSD as scores may be inflated by patients endorsing items related to their chronic pain experience. For example, it is important to distinguish between intrusive recollections of a traumatic event and voluntary rumination about the event and its sequelae, including pain (O'Donnell et al., 2003). Only one study (Asmundson et al., 1998) has attempted to take this overlap into account when interpreting participants' responses on the self-report measures of PTSD. These researchers reported reevaluating the diagnoses of PTSD with the sleep disturbance item of the MPSS deleted to determine the impact of this item on allocation to the PTSD group. They did not attempt to address the other items in the diagnostic criteria of PTSD that overlap to some degree with chronic pain. In this case the deletion of the sleep disturbance item led to only small differences in the proportions of subjects meeting diagnostic criteria for PTSD and Asmundson et al. decided to retain the item for their analyses. Further investigation of the implications of symptom overlap for the interpretation of chronic pain patients' responses on self-report measures of PTSD is

required. This is another issue that could be addressed by thorough psychometric validation of self-report measures of PTSD in samples of chronic pain patients. The issue of symptom overlap was also dealt with in the current investigation of the PCL in Study 1 (see Chapter 6).

4.5. Chapter summary

In summary, there is mounting evidence that chronic pain and PTSD frequently co-occur following traumatic injury. The substantial proportion of patients who present with features of both conditions consistently report higher levels of physical and emotional dysfunction than patients with a single diagnosis. Although several models have been forwarded to explain the co-occurrence of the two conditions, and numerous mechanisms have been identified to account for the interaction between them, research is in its early stages, and many hypotheses remain untested. Furthermore, the literature is characterised by a number of methodological limitations which should be addressed in future research.

From the perspective of the differential impact of types of onset of pain, it is apparent that the potential impact of posttraumatic stress symptoms must be taken into account when comparing adjustment to chronic pain across different onset groups. Distinguishing between the impact of type of onset of pain and the role of posttraumatic stress is important as it may contribute significantly to an understanding of the factors that determine adjustment to chronic pain, and improve approaches to prevention and treatment, particularly for the group of patients who present with both chronic pain and PTSD. The following chapter will discuss potential treatment implications and review the small body of research that has tackled this issue to date.

5. IMPLICATIONS FOR TREATMENT

This chapter focuses on two issues: firstly, the impact of type of onset of pain on treatment outcome; and secondly, the impact of comorbid chronic pain and PTSD on response to treatment for chronic pain. The following discussion reviews the extant literature addressing these issues.

5.1. Impact of type of onset of pain on treatment outcome

The research reviewed in Chapter 2 indicates that onset of pain related to an accident or another specific event is often associated with poor adjustment to chronic pain. This raises the possibility that type of onset of pain also has a negative impact upon treatment outcome. Only three studies have investigated this issue to date.

Firstly, one study has investigated the impact of pain associated with physical trauma on treatment outcome. In a study aimed at detecting possible ethnic group differences in response to biofeedback, Tsushima and Stoddard (1990) divided 238 chronic pain patients into two groups according to the onset of their pain: (1) post-traumatic pain (i.e. the patients reported back pain, neck pain and headaches following a documented incident of physical trauma); and (2) non-traumatic pain (i.e. the patients reported headaches that were not associated with a specific trauma). The investigators reported that patients in the post-traumatic pain group required a greater number of biofeedback sessions and did not respond as well to treatment as patients in the non-traumatic pain group.

Unfortunately, this study has several methodological limitations. Firstly, only self-report of headaches over one week and physiological readings from the biofeedback treatment were used as outcome measures. Consequently, the study does not provide information about treatment outcome in terms of pain-related disability or affective

distress. Secondly, the criteria used to classify patients into the two groups resulted in one group consisting of patients with heterogeneous pain complaints and another consisting only of headache sufferers. Consequently, it is not possible to determine if the results could have been related to the differences in pain site between the two groups. Finally, biofeedback was the only treatment offered, so it is not clear if the results could be generalised to other treatment modalities.

Romanelli, Mock and Tenenbaum (1992) investigated the impact of accident-related pain on treatment outcome. In this study 52 patients who developed temporomandibular disorder after a motor-vehicle accident were compared with another 52 patients who developed the same diagnosis independently of any identifiable physical trauma. The non-accident group was matched to the accident group according to age and gender to eliminate the influence of these demographic variables. A variety of treatment modalities were offered to both groups according to routine clinical management (e.g. application of heat and massage, medication, physiotherapy, trigger point injections, prosthodontic treatment, or biofeedback). Treatment response was assessed with a subjective evaluation by the patient at each appointment. Patients were asked if there was "improvement", "no change", or "worsening of condition", and affective distress was assessed by a series of questions in the clinical interview.

Consistent with the other studies reviewed in Chapter 2, Romanelli et al. reported that 60% of the patients in the accident group were deemed to be experiencing negative affective symptoms, compared to 14% of the non-accident group. Regarding treatment response, only 48% of the accident-onset group reported improvements in their temporomandibular pain symptoms, compared with 75% in the non-accident group. This difference was statistically significant. The investigators noted that the difference in

treatment response occurred despite the fact that all appropriate treatment modalities were used for both groups.

Similarly to Tsushima and Stoddard's (1990) study, Romanelli et al.'s study is limited by a lack of standardised treatment outcome measures. In addition, as the researchers themselves commented, the impact of compensation status on treatment outcome could not be ruled out because this was not taken into account in the analyses.

Finally, Turk, Okifuji, Sinclair and Starz (1998a) evaluated the role of six variables, including onset of pain, in predicting response to a multidisciplinary treatment for fibromyalgia. Discriminant function analysis revealed that idiopathic fibromyalgia (i.e. without an identifiable precipitating event) was predictive of improvements in pain severity, in combination with low pretreatment levels of depression and perceived disability, high pretreatment levels of sense of control and perceived solicitious responses from significant others. Onset of pain was not the focus of Turk et al.'s study, and consequently, this aspect of their analyses was only mentioned briefly. Although it provides preliminary evidence that onset of pain is predictive of response to multidisciplinary pain management programs, given it was a program specifically developed for fibromyalgia and 97% of the sample was female, it is not clear if these results can be generalised to other samples of chronic pain patients.

Overall, the results of these studies suggest that type of onset of pain may influence response to treatment. Specifically, it appears that onset of pain related to a specific event (e.g. accident, injury) is associated with poor response to treatment for chronic pain. However, given the small number of studies that have addressed this issue, and their limitations, further research is clearly warranted. In particular, the impact of type of onset of pain on response to cognitive-behavioural treatments in samples of chronic pain patients that are typical of pain management centres (i.e. heterogeneous groups) has not

been investigated. As discussed in Chapter 1, identifying predictors of treatment outcome may lead to modifications to cognitive-behavioural treatments or to new treatment approaches that could be more effective for sub-groups of chronic pain patients who currently exhibit a limited or poor response to existing treatments (Turk, 2005). Given the impact of type of onset of pain on adjustment-related variables, and the preliminary evidence reviewed above, it is plausible that this variable is also predictive of response to treatment. Consequently, the study presented in Chapter 8 of this thesis examines the impact of type of onset of pain on response to a cognitive-behavioural, multidisciplinary pain management program.

5.2. Comorbid chronic pain and PTSD – Impact on response to treatment

5.2.1. Impact of PTSD on treatment for chronic pain

Early case studies describing patients presenting with both chronic pain and PTSD repeatedly emphasised the importance of identifying posttraumatic stress symptoms when assessing chronic pain patients who have experienced a traumatic event. (e.g. Pilowsky, 1985; Lebovits et al., 1990). Based on their clinical experience, these researchers argued that overlooking posttraumatic stress symptoms could contribute to poor treatment outcome. However, little is known about the impact of the co-occurrence of chronic pain and PTSD on response to treatment for chronic pain because few studies have investigated this issue.

One approach to this issue is to examine the treatment history of patients who present with both chronic pain and PTSD and compare them to patients with chronic pain alone. As part of the study described in the previous chapter, Sherman, Turk and Okifuji (2000) compared the treatment history of fibromyalgia patients with PTSD to a group without PTSD and found no differences between the two groups. Similarly, Chibnall and

Duckro (1994) reported that headache patients with PTSD were no more likely to have had psychological treatment before or after the traumatic event than the patients without PTSD. In contrast, Duckworth and Iezzi (2005) reported that motor-vehicle accident victims with chronic pain and high levels of posttraumatic stress symptoms were more likely than motor-vehicle accident victims with chronic pain alone to have been prescribed medication (particularly anti-depressant medication), and were more likely to have had prior psychological treatment.

Based on these three studies it is difficult to draw any firm conclusions about the impact of comorbid chronic pain and PTSD on treatment history. The limitations of Chibnall and Duckro's and Sherman et al.'s study have already been discussed in the previous chapter and these limitations could have influenced the results. Furthermore, Duckworth and Iezzi's sample consisted only of patients referred to a private practice for medico-legal assessment; consequently, it could be argued that the patients were not representative of chronic pain patients in general.

To date, there are no controlled studies investigating the impact of PTSD on treatment for chronic pain and the literature consists only of case studies. Muse (1986) described three patients with chronic pain and PTSD who had derived limited benefit from multidisciplinary pain management interventions, and who were subsequently treated for PTSD with imaginal exposure (in the form of systematic desensitisation). Muse reported good outcomes for all three PTSD interventions and noted that after the PTSD was addressed the patients' management of their chronic pain also improved.

Similarly, Hickling, Blanchard, Schwarz and Silverman (1992b) reported a case series of 12 patients treated in a private practice for post-traumatic headache related to motor-vehicle accidents. The patients were treated with a range of treatment modalities, including CBT. Although a diagnosis of PTSD did not affect treatment outcome (i.e.

there were no differences in obtained pain relief), the investigators reported that patients with PTSD required significantly more treatment sessions (almost 2.5 times as many) as the patients without PTSD. Consistent with Muse's case studies, Hickling et al. commented that their impression had been that the PTSD needed to be addressed before the patients were able to make progress in managing their headaches more effectively.

5.2.2. Impact of chronic pain on treatment for PTSD

Interest in the impact of chronic pain on response to treatment for PTSD has grown in recent years, and this has been reflected in an increasing number of papers addressing this issue (e.g. the series of papers commenting on a case study presented by Wald & Taylor, 2006a in a recent issue of "Cognitive and Behavioral Practice"). Despite this, few empirical investigations have been conducted.

Shipherd, Beck, Hamblen, Lackner and Freeman (2003) employed a multiple-baseline across-subjects design to a case series of the treatment of six female patients with chronic pain and PTSD following motor-vehicle accidents. They were interested in the impact of treating PTSD on chronic pain symptoms, and consequently, delivered CBT for PTSD to each participant while ensuring that no intervention was delivered for the chronic pain. The treatment for PTSD was delivered over 12 weeks and standardised outcome measures for both PTSD and chronic pain were administered to the participants prior to treatment, before each weekly therapy session, and post-treatment. These measures included the Impact of Events Scale (IES; Horowitz et al., 1979) for posttraumatic stress symptoms and the Owestry Disability Index (ODI; Fairbank et al., 1980) for pain-related function. Participants were also asked to provide ratings of average pain intensity and time spent in bed over the past week.

All six participants reported an improvement in their PTSD symptoms, and all but one patient no longer met diagnostic criteria for PTSD at the end of treatment. Importantly, there was some evidence that the intervention also led to some improvement in pain-related variables. For example, three out of four of the patients who were not working due to pain prior to treatment returned to full-time employment by the end of treatment. Four out of the six patients reported reductions in average pain intensity, and all but one reported spending less time in bed. Five out of the six patients also reported improvements in pain-related function, as assessed by the ODI. Shipherd et al. concluded that treatment of PTSD symptoms in chronic pain patients may help to alleviate chronic pain-related dysfunction even if the pain itself is not addressed.

However, as the investigators noted, the results of the study must be interpreted with caution. Firstly, they noted that the sample was entirely female and excluded patients with current substance abuse problems. Secondly, the study employed a small sample in an uncontrolled design. In addition, although the participants were recruited from a pain treatment centre, two of the participants had only been experiencing pain for 3-4 months. Although this meets the IASP definition of chronic pain, they may not have been representative of the chronic pain patients who typically attend pain management centres.

Wald, Taylor and Federoff (2004) presented two case studies of the treatment of comorbid pain and PTSD. As was the case in Shipherd et al's case series, both patients were female and had been involved in a motor-vehicle accident. The patients received behavioural therapy for PTSD (i.e. imaginal and in vivo exposure) with pain management techniques added as needed (i.e. activity pacing, use of relaxation). Based on the evidence that anxiety sensitivity may play a role in both chronic pain and PTSD, Wald et al. also included interoceptive exposure to reduce anxiety sensitivity. Interoceptive exposure

involves guiding the patient to induce feared physical sensations in order to challenge their beliefs that the sensations are dangerous (Taylor, 2000). A battery of self-report measures (primarily to assess PTSD symptoms and affective distress) were administered pre- and post-treatment. Pain-related variables (i.e. frequency of flare-ups and pain intensity) were assessed by clinical interview for the first patient, who had only been experiencing pain for one month. The other patient presented with chronic pain as the most severe of her problems and consequently, standardised pain self-report measures were utilised (Multidimensional Pain Inventory; Kerns et al., 1985; Pain Disability Index; Tait et al., 1990).

Wald et al. reported that the first patient responded well to the integrated treatment and improvements were noted in both PTSD symptoms and pain (as measured by an average weekly pain severity rating). They noted that although the patient's pain had interfered with her ability to engage in exposure exercises at a number of points during treatment, modifications to the exposure protocol reduced the frequency of exposure-induced flare-ups of pain (e.g. more frequent use of relaxation and breaking down exposure exercises into more manageable tasks). In contrast, despite incorporating a substantial amount of pain management strategies into therapy, the second patient did not report improvements in PTSD symptoms or pain-related variables. Wald et al. pointed out that the second patient presented with a more chronic (duration over a year) and severe pain problem, and speculated that treatment of her PTSD may have been more successful if it had been delivered in the context of treatment in a multidisciplinary pain centre. Wald and Taylor (2006a; b) re-emphasised the need to address chronic pain directly in therapy in the context of presenting another case study in which a patient reported no improvement in pain or pain-related disability at the end of treatment for PTSD, and was unable to maintain his initial PTSD treatment gains at follow-up. They argued that this

outcome was consistent with a mutual maintenance model of comorbid chronic pain and PTSD. That is, if treatment improves PTSD symptoms but does not affect chronic pain, the persistence of the latter may re-ignite the PTSD.

The outcome of Wald et al.'s (2004) second case study is consistent with the findings of Taylor, Federoff, Koch, Thordarson, Fecteau and Nicki (2001), which investigated the impact of pain on PTSD treatment outcome. Fifty patients with PTSD related to motor-vehicle accidents completed a 12-week CBT program for PTSD. Most of the participants reported some form of recurrent pain as a result of the accident. Pain severity and pain-related interference were both assessed with the Multidimensional Pain Inventory. Dynamic cluster analysis was used to identify patterns of treatment outcome. These analyses revealed two patterns of response: partial responders and responders. Taylor et al. reported that compared with responders, partial responders reported significantly higher levels of pain severity prior to treatment. There was also a trend for partial responders to have higher levels of pain-related interference in daily functioning prior to treatment. The investigators hypothesised that treatment outcome might be improved by increasing the duration of therapy, or by including pain management strategies.

Finally, Freidenberg, Hickling, Blanchard and Malta (2006) reported additional results from a randomised controlled comparison of CBT, supportive psychotherapy, and a wait-list control condition for the treatment of PTSD in 78 motor-vehicle accident survivors (Blanchard, Hickling, Devineni, Veazey, Galovski, Mundy, Malta & Buckley, 2003). Freidenberg et al. reported that post-treatment changes in PTSD symptoms were not affected by whether the participants' were suffering from a whiplash injury prior to treatment. In addition, treatment did not affect the pain ratings of the participants with whiplash injuries. Although these results are in contrast to those of Taylor et al. (2001),

Friedenberg et al. acknowledged that given that the impact of whiplash was not the focus of the treatment study the data did not allow them to perform more detailed analyses to investigate the issue.

5.2.3. Research implications

As the above sections illustrate, there is little empirical data regarding the impact of PTSD on response to treatment for chronic pain. Likewise, only a few studies have examined the impact of chronic pain on the treatment of PTSD. Although an integrated CBT treatment protocol that attempts to target both conditions could be effective, such an approach is yet to be evaluated (Otis et al., 2006). As a result, several authors have emphasised that further research is urgently required (Asmundson et al., 2002; Asmundson & Taylor, 2006; Otis et al., 2006). To date, only case studies have examined the impact of PTSD on chronic pain treatment outcomes. In light of this, in addition to investigating the impact of type of onset of pain on treatment outcome, the study presented in Chapter 8 of this thesis also examines the impact of posttraumatic stress symptoms on response to a cognitive-behavioural, multidisciplinary pain management program.

5.3. Treatment implications

Given the absence of empirical data, there is little evidence available to guide clinicians when making judgements about how to approach treatment of individuals with both chronic pain and PTSD (Asmundson et al., 2002; Wald et al., 2004; Asmundson & Taylor, 2006). Although some papers have outlined recommendations for the modification of standard treatments when treating comorbid chronic pain and PTSD, or have offered advice regarding the optimal sequence of treatments, these ideas have been speculative and based predominantly on theory and clinical experience (see e.g. Koch &

Taylor, 1995; Sharp, 2004; Wald et al., 2004; Asmundson & Hadjistavropolous, 2006; Shipherd, 2006; Wald & Taylor, 2006a; b).

Firstly, the importance of helping patients to see the links between their chronic pain and PTSD symptoms, and assisting them to generalise cognitive-behavioural strategies they have learned for one condition to the other has been emphasised (e.g. Sharp & Harvey, 2001; Otis et al., 2006). Secondly, when targeting PTSD symptoms, several authors have suggested modifying standard relaxation techniques to avoid deliberate tensing of muscles that may contribute to pain (e.g. Asmundson & Hadjistavropolous, 2006; Wald & Taylor, 2006a). Modifications to exposure protocols to allow brief relaxation sessions between exposure trials, or shorter exposure trials within session have also been recommended in order to avoid exposure-induced exacerbation of pain (e.g. Wald et al., 2004; Asmundson & Taylor, 2006). Finally, as noted earlier, given the potential importance of anxiety sensitivity, the benefits of incorporating interoceptive exposure have also been highlighted (e.g. Asmundson et al., 2002; Wald et al., 2004; Asmundson & Hadjistavropolous, 2006; Shipherd, 2006). While most researchers have advocated integrating treatments for chronic pain and PTSD in cases of comorbidity (e.g. Asmundson & Hadjistavropolous, 2006; Otis et al., 2006), Bryant and Hopwood (2006) disagreed, arguing that patients can be overwhelmed by attempting to address too many issues at once.

It is worth noting that most of the discussion in the literature has centered on modifications to PTSD treatment protocols; less consideration has been given to potential modifications to CBT for chronic pain when the patient also presents with PTSD. Only Sharp (2004) has commented on this issue by suggesting that a chronic pain patient who is gradually pacing up their tolerance of walking despite pain could be encouraged to gradually walk towards feared trauma-related stimuli.

5.4. Chapter summary

There is little research investigating the impact of type of onset of pain or comorbid PTSD symptoms on treatment for chronic pain, and there is a need for the literature to move beyond case studies to more systematic treatment outcome research. The preliminary nature of the evidence reviewed in this chapter underscores the importance of examining treatment issues in large samples of chronic pain patients undergoing CBT for chronic pain.

6. STUDY 1 - PSYCHOMETRIC PROPERTIES OF A MODIFIED VERSION OF THE PTSD CHECKLIST (PCL)

6.1. Introduction

As discussed in Chapter 4, there is a growing body of research indicating that chronic pain and PTSD frequently co-occur. Previous studies investigating the prevalence of PTSD in chronic pain samples or examining the relationship between chronic pain and PTSD have used self-report measures of posttraumatic stress symptoms initially developed for use in samples other than chronic pain (e.g. combat veterans, rape victims). To date, none of these measures have been adequately validated for use with chronic pain patients, particularly the samples of heterogeneous chronic pain patients characteristic of multidisciplinary pain management centres.

Furthermore, numerous studies have not ensured that participants in chronic pain and PTSD studies respond to items on PTSD self-report measures with specific reference to the event that was associated with the onset of their pain. Consequently, it is not possible to exclude the possibility that at least some of the posttraumatic stress symptoms reported by the participants in these studies were actually related to traumatic events experienced prior, or subsequent, to the onset of pain. As outlined in Chapter 4, this not only has implications for theoretical perspectives on the relationship between chronic pain and PTSD, but is also likely to have led to differences in reported prevalence rates across studies.

Consequently, in this study a widely-used self-report measure of posttraumatic stress symptoms, the PTSD Checklist (PCL; Weathers et al., 1993), was modified to address this issue. The PCL was chosen above other self-report measures of posttraumatic stress symptoms for a number of reasons. Firstly, there are several versions of the PCL

for use in different research and clinical contexts, one of which, the PCL-Specific, allows the identification of a specific stressor. A number of studies have utilised this flexibility to modify the PCL instructions to refer to a specific traumatic event (e.g. Cordova, Studts, Hann, Jacobsen & Andrykowski, 2000; Schnurr et al., 2000; McKenzie, Ikin, McFarlane, Creamer, Forbes, Kelsall, Glass, Ittak & Sim, 2004). This type of modification was made to the PCL for this study to prompt participants to complete the questionnaire with reference to the specific incident or event which led to the onset of their pain (see Section 6.2.3 for details). The ability to modify the instructions in this way was considered essential to address the limitations of previous research. Related to this, given that some chronic pain patients experience a spontaneous or insidious onset of pain, the ability to modify the instructions also ensured that the measure could be completed by these patients. This was necessary in order to examine the psychometric properties of the PCL in a sample of heterogeneous chronic pain patients.

Secondly, based on the research reviewed below, the PCL was judged to have sound psychometric properties across a range of clinical and non-clinical populations, including other medical groups. In addition, the PCL can be used both as a continuous measure of posttraumatic stress symptoms and as a screening tool for PTSD, and this was considered potentially useful in a clinical context when working with chronic pain patients.

Finally, the PCL has been shown to be sensitive to change following treatment for PTSD (Forbes, Creamer & Biddle, 2001) and this was considered necessary for the third study investigating the impact of posttraumatic stress symptoms on chronic pain treatment outcome.

One aspect of the validity of the PCL in chronic pain patient samples pertains to the measure's factor structure. Theoretically, factor structures are considered to be a

reflection of underlying mechanisms (Cattell, 1978); consequently, factor analytic studies of posttraumatic stress in chronic pain patients are important for improving knowledge about posttraumatic stress reactions in this patient group. As far as the author is aware, only one study has explored the factor structure of PTSD symptoms (as measured by the PCL) in individuals with chronic pain. Using confirmatory factor analysis, Asmundson, Wright, McCreary and Pedlar (2003) used the PCL to compare two models of PTSD symptom structure in two groups of male United Nations peacekeepers. One group (n=427) reported experiencing problems with pain and had been diagnosed with chronic back pain, while the other group did not report problems with pain. Seventeen percent of the sample met diagnostic criteria for PTSD, and 13% reported symptoms below the diagnostic threshold. Asmundson et al. reported that both factor models provided a good fit to the data for both groups. The first model consisted of four interrelated factors (i.e. reexperiencing, avoidance, numbing, and hyperarousal), and the second was a hierarchical 2-factor model in which the two lower-order factors were intrusion-avoidance (PCL items 1 to 7) and numbing-hyperarousal (PCL items 8 to 17). As noted in Chapter 4, the final models for the pain and no pain group contained statistically significant different factor loadings for the following symptoms: (1) physical reactions to reminders of the trauma; (2) emotional numbing; (3) a sense of foreshortened future; and (4) hypervigilance (this was different only in the four interrelated factor model).

The four interrelated factor model has also been supported by confirmatory factor analytic studies of the PCL in other trauma groups (e.g. DuHamel, Ostrof, Ashman, Winkel, Mundy, Keane, Morasco, Vickberg, Hurley, Burkhalter, Chhabra, Scigliano, Papadopoulos, Moskowitz & Redd, 2004; Marshall, 2004; Palmieri & Fitzgerald, 2005; Schinka, Brown, Borenstein & Mortimer, 2007). At least two studies have provided support for a slightly different four-factor solution in which the numbing symptoms and

three of the hyperarousal symptoms (i.e. sleep disturbance, irritability and difficulty concentrating) load onto a "dysphoria" or "general distress" factor (Simms, Watson & Doebbelling, 2002; Palmieri, Weathers, Difede & King, 2007). Only one study has provided support for the second-order, three-factor symptom structure reflected in the DSM-IV criteria for PTSD (Cordova et al., 2000).

Fewer studies have applied exploratory factor analytic techniques to PCL data and the results of these studies have also been mixed. For example, in a validation study of the PCL using Gulf War veterans Weathers et al.'s (1993) analysis revealed one large factor consisting mainly of reexperiencing, avoidance and hyperarousal items, and one smaller factor consisting mainly of emotional numbing and hyperarousal items. In contrast, studies of cancer patients have identified a number of different solutions, typically involving four factors (Smith, Redd, DuHamel, Vickberg & Ricketts, 1999; Shelby, Golden-Kreutz & Andersen, 2005). It has been suggested that PTSD symptom structure may vary to some degree across different types of trauma and sample characteristics (such as prevalence of PTSD or treatment-seeking status) and that this may account for the range of factorial models reported both in PCL research and studies using other measures of PTSD (Palmieri et al., 2007).

Given the variability of previous factor analytic studies of the PCL, and the fact that Asmundson et al.'s (2003) study was conducted with a male-only sample of individuals who were not presenting for treatment of chronic pain, exploration of PTSD symptom structure (as measured by the PCL) in samples of treatment-seeking chronic pain patients is warranted. Consequently, the aim of the current study was to investigate the psychometric properties (including the factor structure) of the modified PCL in a large sample of chronic pain patients presenting for treatment at a tertiary referral pain management centre.

6.2. Method

6.2.1. Participants

The participants were 615 individuals, including 263 males (42.8%) and 352 females (57.2%), referred to the University of Sydney Pain Management and Research Centre at Royal North Shore Hospital in Sydney, Australia. These individuals underwent a multidisciplinary assessment at the Centre on their first visit between June 2003 and April 2005. Initially, 643 patients were identified as participants in the study; however, of these, 28 patients were not included in the study. Eight patients were excluded because they were unidentifiable, or their assessments at the Centre had not proceeded, or they were not new patients to the Centre (i.e. they had completed the self-report measures on prior visits). Twenty patients (3.1% of the initial sample) were excluded because they did not provide consent for their responses on the self-report measures to be used in research at the Centre. The demographic characteristics of these 20 individuals were compared to the characteristics of those who did consent, and the outcomes of these analyses are provided in the Results section. A complete description of the sample who participated in the study is also provided in the Results section.

6.2.2. Procedure

Information regarding the demographic and clinical characteristics of the sample was obtained from a questionnaire that is routinely mailed to new patients to complete prior to their assessment at the Pain Management and Research Centre (see Appendix A). The questionnaire covers basic demographic information (e.g. age, gender, marital status, highest level of education, private health insurance and pension status), pain-related information (e.g. pain site, how the pain began), work-related information (e.g. work status), and compensation-related information (when applicable). In the present study,

data that was found to be missing from these questionnaires (e.g. pain site or work status) were obtained from the participant's medical file to ensure that as much information as possible was obtained about each participant.

At the time of their assessment all patients presenting to the Centre are asked to complete a battery of questionnaires. For this study the PCL was administered as part of this standard battery. The standard battery includes the Multidimensional Pain Inventory (MPI; Kerns et al., 1985), a modified version of the Roland and Morris Disability Questionnaire (RMDQ; Roland & Morris, 1983), the Pain Self-Efficacy Questionnaire (Nicholas, 1989), the Tampa Scale for Kinesiophobia (Kori et al., 1990), the Pain-Related Self-Statements Scale (PRSS; Flor & Turk, 1988) and the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995b). Copies of these questionnaires are provided in Appendix A. For the purposes of the current study, the RMDQ and one scale of both the MPI and DASS were used to describe the sample's characteristics and to analyse patterns of missing data. Consequently, although they were not the main focus of the study these three measures are described in detail in the following section. The rest of the questionnaire battery will be introduced in subsequent chapters as relevant.

Ethics approval for the study was provided by the Northern Sydney Health Human Research Ethics Committee. Patients were asked to complete and sign a cover sheet to indicate their consent for information collected about them to be used for research purposes. A copy of the cover sheet is provided in Appendix A.

6.2.3. Measures

The PTSD Checklist (PCL)

The PCL (Weathers et al., 1993) contains 17 items, corresponding to the DSM-IV diagnostic criteria for PTSD, thereby allowing calculation of three subscale scores

corresponding to the DSM-IV PTSD symptom clusters. Respondents indicate how much they have been bothered by each symptom in the past month using a 5-point scale, where 1 = "not at all" and 5 = "extremely". A total score is obtained by summing each item score so that possible scores range from 17 to 85. The Reexperiencing subscale score is calculated by summing the responses to items 1 to 5, the Avoidance subscale score is calculated by summing the responses to items 6 to 12, and the Arousal subscale score is calculated using the responses to items 13 to 17.

The PCL can be used as a continuous measure of PTSD symptom severity by calculating the Total score across all 17 items, and can also be used to determine whether a PTSD diagnosis is likely by following the DSM-IV diagnostic algorithm for PTSD (Weathers et al., 1993). That is, a score of three (or "Moderately" according to the scale) and above for an item can be considered as symptomatic. An individual who scored three or above for at least one Reexperiencing item, at least three Avoidance items, and at least two Arousal items would be considered to potentially meet diagnostic criteria for PTSD

In this study, the PCL instructions were modified as follows:

INSTRUCTIONS: Below is a list of problems and complaints that people sometimes have in response to stressful life experiences. For this questionnaire, the stressful experience we would like you to refer to is the incident or event (e.g. accident or injury) that lead to the onset of your pain.

Participants who did not attribute their pain to a specific incident were prompted to answer the questions with reference to the period in which their pain developed, as follows:

If your pain was not the result of an injury or accident, the stressful experience would be the period when your pain was developing.

The PCL items were also modified to link symptoms to the onset of pain (e.g. "Repeated, disturbing dreams of when your pain began" or "Feeling very upset when something reminded you of when your pain began"). A copy of the modified PCL used in this study is provided in Appendix A.

The PCL has been used to assess PTSD symptoms in a wide range of trauma groups, including World War II, Vietnam, and Persian Gulf war veterans (e.g. Weathers et al., 1993; Schnurr et al., 2000; Forbes et al., 2001; McKenzie et al., 2004), former prisoners of war (e.g. Cook, Thompson, Coyne & Sheikh, 2003), motor-vehicle accident survivors (e.g. Blanchard, Jones-Alexander, Buckley & Forneris, 1996a), sexual assault victims (e.g. Blanchard et al., 1996a), cancer patients or survivors (e.g. Cordova, Andrykowski, Kenady, McGrath, Sloan & Redd, 1995; Andrykowski, Cordova, Studts & Miller, 1998), and HIV/AIDS patients (Smith et al., 2002). The PCL has also been used to determine levels of PTSD symptoms in non-clinical samples, either in primary care settings (e.g. Stein et al., 2000; Monnier, Grubaugh, Knapp, Magruder & Frueh, 2004), or in community studies (e.g. Barnes, Treiber & Ludwig, 2005).

Importantly for the current context, the PCL has been utilised in one study investigating PTSD symptoms in a sample of 141 patients presenting to an orofacial pain centre (Sherman et al., 2005). Sherman et al. reported that the PCL had good psychometric properties in this patient group. In particular, they reported high internal consistency coefficients for the PCL sub-scales and the total score (Reexperiencing = 0.92; Avoidance = 0.90; Arousal = 0.86; and Total score = 0.95).

The research conducted to date has also provided support for the reliability of the PCL in other populations. In the original validation study with 123 Vietnam Veterans, Weathers et al. (1993) reported internal consistency coefficients (Cronbach's alpha) of 0.97 for the total scale, 0.93 for the Reexperiencing subscale, 0.92 for the Avoidance

subscale, and 0.92 for the Arousal subscale. Similarly high reliability coefficients were reported by Weathers and his colleagues (1993) in a larger (N=1006) validation study with Persian Gulf veterans, and by Blanchard et al. (1996a) in a sample of motor-vehicle accident and sexual assault victims. Further evidence supporting the internal consistency of the PCL can be found in studies of breast cancer survivors, bone marrow transplant recipients, female veterans in primary care, and in a university student study (Andrykowski et al., 1998; Cordova et al., 2000; Lang, Laffaye, Satz, Dresselhaus & Stein, 2003; Ruggiero, Del Ben, Scotti & Rabalais, 2003).

Research has also supported the validity of the PCL. Weathers et al. (1993) and Ruggerio et al. (2003) both reported strong correlations between the PCL and other well-established measures of PTSD symptoms (e.g. Impact of Events Scale), indicating good convergent validity. Both studies also reported moderate correlations between the PCL and variables that would be expected to be less related to PTSD symptoms, such as depression and measures of general psychopathology, suggesting the PCL also has good discriminant validity (Weathers et al., 1993; Ruggiero et al., 2003).

The PCL has also been shown to correlate highly with established structured clinical interviews for PTSD and numerous studies have demonstrated the scale's ability to accurately predict PTSD diagnostic status in a range of patient groups. Blanchard et al. (1996a) reported that the overall correlation between the PCL and the CAPS was 0.929 in a mixed motor-vehicle accident and sexual assault victim sample. In the orofacial pain study cited above, Sherman et al. (2005) used the SCID for DSM-IV (Spitzer, Williams, Gibbon & First, 1995) to diagnose PTSD and reported that a cut-off score of 41 on the PCL provided sensitivity (i.e. the chance that a condition that is present will be detected) of 0.82, and specificity (i.e. the chance that a condition that is not present will be found to

be absent) of 0.92. They also reported that this cut-off score correctly classified 89.9% of participants.

Research conducted with other patient groups has indicated that different PCL cut-off scores may be appropriate for different groups (Andrykowski et al., 1998; McKenzie et al., 2004; Norris & Hamblen, 2004). For example, Weathers et al. (1993) reported that a cut-off score of 50 provided sensitivity of 0.82, and specificity of 0.83 in a Vietnam veteran sample. Blanchard et al. (1996a) found that when compared with clinician ratings on the CAPS, a cut-off score of 44 maximised diagnostic efficiency for motor vehicle accident and sexual assault victims. As Norris and Hamblen (2004) have pointed out, when using the PCL as a screening measure, lower cut-off scores may be more appropriate for samples with lower PTSD rates. Walker, Newman, Dobie, Ciechanowski and Katon (2002) identified 30 as an optimal cut-off score for females seen in a primary care setting, while Dobie, Kivlahan, Maynard, Bush, McFall, Epler and Bradley (2002) suggested a cut-off score of 38 was optimal for use with female veterans assessed in primary care. A number of investigators have suggested that rather than choosing between use of a cut-off score or a diagnostic algorithm, using the two methods in combination increases the diagnostic accuracy of the PCL (Blanchard et al., 1996a; Cook et al., 2003; Ruggiero et al., 2003).

The West-Haven Yale Multidimensional Pain Inventory (MPI)

The MPI (Kerns et al., 1985) was developed to provide a comprehensive assessment of chronic pain from a cognitive-behavioural perspective; that is, it focuses on the assessment of the individual's perceptions and beliefs about the pain, the degree to which pain interferes in daily activities, and the responses of significant others. It was

designed to be administered in the context of comprehensive evaluations of chronic pain patients in clinical settings (Kerns et al., 1985).

The MPI contains 52 items divided into three sections (only Sections 1 and 2 are routinely administered at the Centre). Section 1 consists of five scales assessing: (1) Pain Severity (PS) – perceptions of pain severity and suffering; (2) Interference (I) – perceptions of the degree to which pain interferes in household, work-related, social, family and recreational activities; (3) Life Control (LC) – perceptions of the ability to solve problems and exert control over stressors (including pain); (4) Affective Distress (AD) – includes depressed mood, irritability and tension; and (5) Support (S) – perceptions of the support received from significant others. Section 2 evaluates the individual's perception of the responses by significant others to displays of pain (Kerns et al., 1985). It contains 14 items and consists of three scales: (1) Punishing Responses (PR) e.g. ignoring, or expressing irritation, frustration or anger; (2) Solicitous Responses (SR) e.g. giving pain medication or food, taking over chores, asking how he/she can help; and (3) Distracting Responses (DR) e.g. suggesting or encouraging work on a hobby. The version of the MPI used in this study includes an additional eight items in Section 1 but the conceptual basis of the scales does not differ from the original version (Rudy, 1989).

In this study, only the Pain Severity scale was used for the analyses. The Pain Severity scale is calculated using three items enquiring about level of pain at the "present moment" and "during the last week" and about the degree of suffering experienced because of pain. Possible scores range from 0 to 6. Holmes and Stevenson (1990) demonstrated that the Pain Severity scale could be used on its own as a reliable measure of pain intensity in a study involving both chronic pain patients and patients with pain of recent-onset (i.e. less than four weeks). The internal consistency (Cronbach alpha) of the Pain Severity scale has been reported to be 0.72, with a 2-week test-retest reliability

coefficient (Pearson product-moment correlation) of 0.82 (Kerns et al., 1985). In the current study, the Cronbach alpha = 0.756.

The Roland and Morris Disability Questionnaire (RMDQ)

The RMDQ (Roland & Morris, 1983) was developed to measure self-rated disability due to back pain. It consists of 24 statements (e.g. "I only stand up for short periods of time because of my back") and the patient is asked to tick the statements which describe them "today". The respondent's score is calculated by adding the number of ticked statements. Consequently, possible scores range between 0 (no disability) and 24 (severe disability).

A number of studies have demonstrated the reliability and validity of the RMDQ (e.g. Roland & Morris, 1983; Deyo, 1986a; Jensen, Strom, Turner & Romano, 1992). It has also been shown to be sensitive to improvements in back pain over time (Roland & Morris, 1983; Deyo, 1986a) and has been reported to be responsive to changes with treatment (Klein & Eek, 1990; Beurskens, de Vet & Koke, 1996).

The version of the RMDQ routinely administered at the Centre includes a modification to the items so that the questionnaire can be completed by chronic pain patients with pain in various sites. For the 23 statements regarding daily activities the phrase, "because of my back" is replaced with "because of my pain". The statement "My back is painful almost all the time" (item 13) is replaced with "I am in pain almost all the time". The validity of this modified version of the RMDQ as a measure of pain-related disability in a heterogeneous sample of chronic pain patients (i.e. pain in different sites) has been supported by a number of studies (Jensen et al., 1992; Asghari & Nicholas, 2001; Nicholas, Asghari & Blyth, 2008).

The Depression Anxiety Stress Scales (DASS)

The DASS (Lovibond & Lovibond, 1995b) consists of 42 items assessing current symptoms of depression, anxiety and stress. The Depression scale (DASS-D) assesses anhedonia, hopelessness, lack of incentive, loss of self-esteem and devaluation of life. The Anxiety scale (DASS-A) assesses autonomic arousal and situational anxiety, and the Stress scale (DASS-S) measures symptoms of chronic non-specific arousal, including irritability, nervousness, physical tension and agitation (Lovibond & Lovibond, 1995b).

The DASS scales each contain 14 items. Respondents are instructed to rate the degree to which they experienced each symptom "over the past week" according to a 4-point scale, where 0 corresponds to "Did not apply to me at all" and 3 corresponds to "Applied to me very much, or most of the time". Scale scores are calculated by summing the 14 relevant item scores. Possible scores for each scale range from 0 to 42.

Research conducted since the development of the DASS has confirmed the three-factor structure of the questionnaire in both non-clinical and clinical samples (Lovibond & Lovibond, 1995a; Brown, Chorpita, Korotitsch & Barlow, 1997; Antony, Bieling, Cox, Enns & Swinson, 1998; Clara, Cox & Enns, 2001; Crawford & Henry, 2003). Studies utilising non-clinical and clinical samples have provided strong support for the internal consistency of the DASS (Lovibond & Lovibond, 1995a; Brown et al., 1997; Antony et al., 1998; Crawford & Henry, 2003).

A key advantage of the DASS is that when compared to other self-report measures of depression containing numerous somatic items (e.g. disturbed sleep, constipation, fatigue) the DASS is a more accurate measure of depressive symptoms in chronic pain patients (Taylor, Lovibond, Nicholas, Cayley & Wilson, 2005). Taylor et al.'s study also demonstrated that the DASS exhibits excellent internal consistency in a sample of chronic pain patients (DASS-D = 0.96; DASS-A = 0.90 and DASS-S = 0.94). In the current

study, the Cronbach alpha for the three scales were: DASS-D = 0.951; DASS-A = 0.882 and DASS-S = 0.941.

6.2.4. Aims

The aim of this study was to assess the performance of a modified version of the PCL in a sample of chronic pain patients. There were five key objectives associated with the study.

(1) To determine if patients were able to follow the modified instructions described above so that the PCL provided a measure of PTSD symptoms related only to the onset of pain and not to prior or subsequent events.

(2) To compare the participants' responses as a group to those obtained in prior research with the PCL in other populations.

(3) To investigate specific psychometric properties of the modified PCL in this sample; that is, internal consistency, split-half reliability, and the item-total correlations.

(4) To investigate the factor structure of the modified PCL in a chronic pain sample.

(5) To determine the proportion of the sample that would be classified as meeting diagnostic criteria for PTSD if the diagnostic algorithm and cut-off scores suggested previously in the literature were applied to chronic pain patients.

6.2.5. Data analyses

All statistical analyses were conducted using the SPSS for Windows package, Version 12.0, unless otherwise specified. Statistical significant was set at $p < 0.05$.

To determine whether the sample was representative of chronic pain patients typically presenting to the Centre, the participants' demographic and pain-related

characteristics and mean scores on the self-report measures were compared with the Centre's normative data sample (Nicholas et al., 2008).

Secondly, the demographic characteristics of the 20 individuals who did not provide consent to be involved in the research were compared to the characteristics of those who did consent. These comparisons were conducted using Student's *t*-tests for age and pain duration, and Fisher's exact tests for gender and whether the visit to the Centre was related to a claim or legal case.

Missing Values Analyses

In the initial stages of the study, the completed questionnaires of 33 participants were examined for missing values to determine if patients were able to complete the PCL with the modified instructions.

In the absence of empirically derived guidelines for dealing with missing values in the PCL, for the main analyses the PCL was considered incomplete if more than 30% of the items (i.e. more than five items) were missing from the total scale. The Reexperiencing, Arousal and Avoidance subscales were all considered incomplete if more than one item contributing to that subscale was missing (i.e. more than one out of five items for the Reexperiencing and Arousal subscales and more than one out of seven items for the Avoidance subscale). Participants with any incomplete subscales, participants who had more than 30% of items missing from the total scale, and participants who had not completed the PCL at all were all categorised as "non-completers".

Similarly to the PCL, in the absence of empirically derived guidelines for dealing with missing values in the other self-report measures, they were considered incomplete if more than 30% of responses for the total scale or subscales were missing. For the DASS,

the scales were considered incomplete if more than two items were missing (P. Lovibond, personal communication, 30 May 2005).

As recommended by Hair, Black, Babin, Anderson and Tatham (2006), an analysis of the missing PCL values was conducted prior to the main analysis to determine whether the missing values were randomly distributed throughout the data set. The main purpose of examining the randomness of missing values is to ensure that the method selected for dealing with this missing data does not introduce a bias into the data set (Hair et al., 2006). For the purposes of this study, a missing values analysis was not conducted for the MPI, RMDQ and DASS because they were not the focus of the study and were only used to describe the sample.

Potential patterns in the missing PCL values were investigated by comparing participants who had completed the PCL with those who were classified as "non-completers". Seventy-nine participants (or 12.8%) were classified as non-completers. The two groups were compared on the main demographic and clinical variables using Student's *t*-tests, chi-square tests or Fisher's exact tests as appropriate. The demographic variables examined included age, gender, and reason for the visit to the Centre (i.e. was the visit regarding a compensation claim or some other legal case?). The clinical variables examined included pain duration, pain severity (as measured by the MPI Pain Severity Scale), how the pain began, pain-related disability (as measured by the RMDQ), and depression (DASS-D).

Main Analyses

A number of analyses were conducted to examine the performance of the PCL in this sample of chronic pain patients. Firstly, descriptive statistics such as the range of scores, means and standard deviations were calculated for each of the PCL items, the

PCL subscales and the PCL total score. The pattern of responses on the PCL was also examined by determining the percentage of participants endorsing each symptom. Following Weathers et al. (1993), a symptom was considered to be endorsed if the patient rated it as 3 or above (i.e. "moderately" or above). The cut-off scores of 50 and 41, as suggested by Weathers et al. (1993) and Sherman et al. (2005) were also applied to the data to determine the proportion of participants that would be classified as potentially meeting diagnosis for PTSD.

Secondly, the internal consistency of the PCL in this sample was examined by computing Cronbach's alpha coefficient for each subscale and for the total scale. Internal consistency is one way of testing the reliability of a measure and is based on the consistency of responses to all items in that measure (Anastasi, 1988). If the items in a test or measure correlate highly with another, the items are considered to be measuring the same underlying construct (Oppenheim, 1992). Cronbach's alpha is one of the most widely used measures of internal consistency and is based on the individual test item variances (Friedenberg, 1995). Cronbach's alpha coefficients range between 0 and 1, with higher values indicating greater internal consistency.

Another measure of reliability, split-half reliability, was also examined using the Spearman-Brown and Guttman split-half reliability coefficients. Split-half reliability methods involve dividing the measure into two halves and calculating the correlation between the two halves (Oppenheim, 1992). The Spearman-Brown split-half reliability coefficient indicates the degree of the correlation between the two halves, adjusted for the shortened length of the test (Friedenberg, 1995). The Guttman split-half reliability coefficient also measures the correlation between the two halves but does not assume equal variances between the two split halves (Friedenberg, 1995). The Guttman

coefficient is considered suitable if the two variances differ significantly (Friedenberg, 1995).

The inter-item correlations and item-total correlations were also calculated to provide more detail regarding the relationships between items and the total scale. A low correlation between an item and the total scale indicates that the item may not be measuring the same construct as the rest of the scale (Cohen & Swerdlik, 2002). Recalculation of Cronbach's alpha with each item deleted indicates the degree to which the internal consistency of the scale would be improved if that item was deleted, providing further information regarding the relationship between each item and the total scale.

Finally, the PCL items were subjected to exploratory factor analysis. The suitability of the data for factor analysis was assessed using the Bartlett test of sphericity (Bartlett, 1954) and the Kaiser-Meyer-Oklin measure of sampling adequacy (Kaiser, 1970; 1974). The Bartlett test of sphericity is a statistical test for the presence of correlations among the variables and it must be significant ($p<0.05$) for the data to be considered suitable for factor analysis (Tabachnik & Fidell, 2001). However, given that it is a statistical test it is sensitive to increases in sample size (Hair et al., 2006). Consequently, the Kaiser-Meyer-Oklin measure of sampling adequacy was also used. This is an index ranging from 0 to 1, with 0.6 being the minimum value required to consider the data suitable for factor analysis (Tabachnik & Fidell, 2001).

An exploratory factor analytic approach was selected over confirmatory factor analysis given that no other studies have been conducted with a sample of heterogeneous chronic pain patients presenting to a multidisciplinary pain management centre. A principal components analysis (PCA) was initially selected because it has been argued that common factor analysis suffers from factor indeterminacy; that is, more than one set

of factor scores can be calculated from a given factor pattern (Schonemann & Wang, 1972; Mulaik & McDonald, 1978). However, given that the advantages and disadvantages of the two methods have been debated (Hair et al., 2006) both types of analyses were conducted to ensure that the results obtained were not due to the method employed.

Following the recommendations of Thompson and Daniel (1996), the number of factors to retain was determined using multiple criteria: (1) applying the latent root criterion of eigenvalues greater than 1 (Kaiser, 1960); (2) applying the scree-test criterion (Cattell, 1966); and (3) parallel analysis, a statistical technique for determining the appropriate break in the scree-plot (Horn, 1965). Parallel analysis involves comparing the eigenvalues with those obtained from a randomly generated data set of the same size, and retaining only the eigenvalues that are larger than the corresponding values from the random data set. Parallel analysis has been shown to be one of the most accurate methods for determining how many factors to retain (Zwick & Velicer, 1986). Following the recommendations of Longman, Cota, Holden and Fekken (1989), parallel analysis was performed using both the mean eigenvalues and the 95^{th} percentile eigenvalues. The parallel analysis was conducted using the Monte Carlo PCA for Parallel Analysis program (Watkins, 2000).

An oblique (Oblimin) rotation was performed because this method allows for the factors extracted being correlated (Hair et al., 2006). Following Comrey and Lee's (1992) recommendations, the following guidelines for interpretation of factor loadings were applied: (1) factor loadings ≥ 0.32 are poor but can be interpreted; (2) factor loadings ≥ 0.45 are fair; (3) factor loadings ≥ 0.55 are good; (4) factor loadings ≥ 0.63 are very good; (5) factor loadings ≥ 0.71 are excellent.

6.3. Results

6.3.1. Participant characteristics

The demographic and pain-related characteristics of the sample of 615 participants in this study and in the Centre's normative data sample (Nicholas et al., 2008) are presented in Tables 6.1 and 6.2 on the following pages. The mean age of participants was 51 years (SD = 16.7; range = 14 to 90 years of age), and the mean duration of pain was 93.4 months or approximately 7 years (SD = 129.5; range = 1 to 760 months). The most common single pain site was lower back and lower limbs (132 participants or 22.3% of the available data for this characteristic), with 221 participants (or 37.3% of available data) reporting pain in two or more major sites. 155 participants (26.2% of available data) identified the cause of their pain as a work-related accident, while 128 (21.7%) reported that their pain "just began, no clear reason". 233 participants (39.8%) were visiting the Centre about a pain condition related to a compensation claim or other legal case. As can be seen in Tables 6.1 and 6.2, the sample in the current study was typical of the Centre's patients across all demographic and pain-related variables.

Table 6.1: Participants' demographic characteristics compared with the normative sample (Nicholas et al., 2008)

Variable	Current Study	Nicholas et al. (2008)
	N = 615	**N = 5, 941**
Age (years)	n = 615	n = 5, 941
M (SD)	51 (16.7)	48 (16.2)
Range	14 - 90	[+]
Gender	n = 615	n = 5, 941
Male	263 (42.76%)	2,528 (42.6%)
Female	352 (57.24%)	3,413 (57.4%)
Marital Status	n = 586	n = 4, 508
Married/De facto	356 (60.75%)	2886 (64.0%)
Divorced/Separated	68 (11.6%)	544 (12.1%)
Single/Never Married	119 (20.31%)	800 (17.7%)
Widowed	43 (7.34%)	278 (6.2%)
Highest Level of Education	n = 560	n = 4, 377
Post high school qualification	231 (41.25%)	1529 (34.9%)
Completed high school	57 (10.12%)	453 (10.4%)
Between 9 and 11 years	193 (34.46%)	1678 (38.3%)
Less than 9 years	57 (10.18%)	717 (16.4)
Other	22 (3.93%)	[+]
Work Status	n = 574	n = 4, 438
Full-time/Part-time work	169 (29.44%)	1348 (30.4%)
Home Duties	41 (7.14%)	462 (10.4%)
Unemployed due to pain	182 (31.71%)	1430 (32.2%)
Retired	132 (23.0%)	804 (18.1%)
Other	50 (8.71%)	394 (8.9%)
Is this visit related to:	n = 585	n = 4, 467
A Workers Compensation Claim	196 (33.5%)	1429 (32.0%)
A Third Party Accident Compensation Claim	33 (5.64%)	318 (7.1%)
Some other legal case	4 (.68%)	78 (1.8%)
None of the above	352 (60.17%)	2642 (59.1%)

[+] Data not reported in Nicholas et al. (2008)

Table 6.2: Participants' pain-related characteristics compared with the normative sample (Nicholas et al., 2008)

Variable	Current Study N = 615	Nicholas et al. (2008) N = 5, 941
Pain Duration (months)	n = 612	n = 5, 285
M (SD)	93.44 (129.47)	80.2 (111.2)
Range	3 - 760	6 - >300
Pain Site	n = 593	n = 4, 932
Head, face and mouth	40 (6.75%)	364 (7.4%)
Cervical region	13 (2.19%)	146 (3.0%)
Upper shoulder and upper limbs	69 (11.64%)	566 (11.5%)
Thoracic region	20 (3.37%)	102 (2.1%)
Abdominal	7 (1.18%)	92 (1.9%)
Lower back, lumbar spine and sacrum	37 (6.24%)	641 (13.0%)
Lower limbs	44 (7.42%)	391 (7.9%)
Pelvic region	6 (1.01%)	53 (1.1%)
Anal, peri-anal and genital	4 (.67%)	60 (1.2%)
Lower back and lower limbs	132 (22.26%)	701 (14.2%)
More than 2 major sites	221 (37.27%)	1816 (36.8%)
How did your pain begin?	n = 591	n = 4, 635
Accident at work	155 (26.23%)	1245 (26.9%)
At work, but not involving an accident	41 (6.94%)	318 (6.9%)
Accident at home	14 (2.37%)	166 (3.6%)
Motor vehicle accident	65 (11.0%)	589 (12.7%)
After surgery	74 (12.52%)	546 (11.8%)
After illness	24 (4.07%)	185 (4.0%)
Pain just began, no clear reason	128 (21.66%)	1028 (22.2%)
Other	70 (11.84%)	558 (12.0%)
Multiple cause	20 (3.38%)	[+]

[+] Data not reported in Nicholas et al. (2008)

The means and standard deviations of the self-report measures analysed in this study are presented with those from the normative sample in Table 6.3. As was the case with the demographic and pain-related variables, the participants in the study obtained comparable scores on the self-report measures to the Centre's normative sample.

Table 6.3: Means and standard deviations for self-report measures in the current study and in the normative sample (Nicholas et al., 2008)

Self-report Measure	Current Study	Nicholas et al. (2008)
	N = 615	N = 5, 941
MPI – PS	n = 592	n = 4, 846
M (SD)	4.1 (1.0)	4.2 (1.1)
RMDQ	n = 599	n = 4, 897
M (SD)	12.3 (5.6)	12.3 (5.7)
DASS – D	n = 571	n = 2, 445
M (SD)	13.9 (11.6)	14.3 (11.9)

Note: MPI – PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS – D = Depression Anxiety Stress Scales – Depression scale.

6.3.2. Preliminary analyses

Consent to participate in research

Student *t*-test and Fisher's exact test comparisons revealed that there were no significant differences in age, duration of pain, or gender between patients who provided consent for their self-report measures to be used in research and those who did not consent (see Table 6.4). In addition, patients who did not provide consent did not significantly differ from those who did consent with respect to whether they were visiting the Centre regarding a compensation claim or legal case (Fisher's Exact Test, $p = 0.19$).

Missing Values Analysis

As outlined in the Method section, in the initial stages of the study the completed questionnaires of 33 participants were examined for missing values to determine if patients were able to complete the PCL with the modified instructions. Only five missing items were found in the 33 questionnaires (i.e. less than 1% of the total number of items) indicating that participants did not have any notable difficulties completing the PCL with the modified instructions.

Table 6.4: Comparison of patients who consented and did not consent to participate in research

Variable	Provided Consent (n = 615)	Did not Consent (n = 20)	*t* (df) or Fisher's exact	*p*
Age (years)	n = 615	n = 20		
M (SD)	51.00 (16.72)	49.75 (12.27)	.44 (21)	.663
Pain Duration (months)	n = 612	n = 19		
M (SD)	93.44 (129.47)	82.95 (81.82)	.35 (629)	.726
Gender	n = 615	n = 20		
Male	263 (42.76%)	6 (30%)	6.0	.183
Female	352 (57.24%)	14 (70%)		
Visit related to claim or legal case?	n = 585	n = 19		
Yes	233 (39.83%)	10 (52.63%)	9.0	.188
No	352 (60.17%)	9 (47.37%)		

The results of the Student *t*-tests, chi-square tests and Fisher's exact tests comparing PCL completers and non-completers are presented in Appendix B (see Table 1). For the demographic variables, there was a significant difference in age between completers (M = 49.55 years, SD = 16.21) and non-completers [M = 60.84 years, SD = 16.91; $t(613) = -5.75$, $p = 0.000$]. A greater proportion of participants involved in a claim or legal case completed the PCL compared with those who were not involved in a claim (42.66% compared with 20.27%; Fisher's exact test, $p = 0.000$). An independent samples *t*-test confirmed that this was related to the age difference between the two groups; that is, the participants not involved in a claim or legal case were older (M = 57.07 years, SD = 17.50) than those who were involved in one [M = 41.85 years, SD = 10.46; $t(578) = -13.15$, $p = 0.000$]. There were no significant differences between completers and non-completers in terms of gender (Fisher's exact test, $p = 0.429$).

No significant differences were found between completers and non-completers in terms of pain duration [$t(90) = -1.19$, $p = 0.236$], pain severity [$t(590) = -1.36$, $p = 0.173$], pain-related disability [$t(597) = -0.58$, $p = 0.565$] or depression [$t(569) = 0.48$, $p = 0.634$].

However, the chi-square test comparing the two groups on the basis of how their pain began was significant (X^2 = 23.06, p = 0.005). An examination of the adjusted residuals for this analysis indicated that non-completers were less likely than completers to choose one of the specific categories provided (e.g. work-related accident, motor-vehicle accident, pain began after an illness, pain just began with no clear reason) and instead were more likely to choose the "other" category. Unlike the differences detected between the two groups on the demographic variables, this result did not appear to be due to the difference in age between completers and non-completers as a *t*-test comparing participants who chose the "other" category with those who did not revealed no significant age differences [$t(589)$ = -0.85, p = 0.394]. However, this result could be explained by the finding that non-completers were less likely to be involved in a claim or legal case. Since compensation claim and legal suits tend to be the result of an injury or accident, those not involved in such a claim may have found it more difficult to choose one of the specific categories to describe how their pain began.

Given the age difference between PCL completers and non-completers, another series of *t*-tests were conducted to determine if the same age pattern could be found for the other self-report measures. These analyses indicated no significant age differences between participants who had completed the MPI scales and the RMDQ and those who had not (all *p*-values were greater than or equal to 0.06). However, there were significant age differences between those who had completed the other self-report measures and those who had not, with the non-completers for each questionnaire found to be significantly older as a group than the completers (all *p*-values were equal to 0.000). The MPI and RMDQ were the first two questionnaires in the battery, suggesting that the older participants tended not to complete the entire battery compared with the younger participants. It is important to note that the PCL was positioned last in the battery.

Overall, the missing values analyses suggested that the missing values were not randomly distributed throughout the data set, and were concentrated in the older age groups. Tabachnik and Fidell (2001) recommend retaining all cases when missing data is not randomly distributed, as deleting cases with missing data from the analysis could result in a biased sample. In this situation they recommend using a method for estimating the missing data. For this study, this would have involved estimating a large number of PCL items, as 79 participants (most of whom had not completed the PCL at all) had been classified as non-completers. Given that one of the main objectives of the study was to evaluate the reliability of the PCL (particularly internal consistency), it was decided that estimating all of the missing values could influence the reliability of the scale by artificially inflating the correlations between items. However, before deciding to delete the non-completers, another set of analyses was conducted to ascertain the degree to which the sample would be biased if the non-completers were deleted.

For these analyses completers and non-completers were compared within their own age group to determine whether the non-completers were different to the completers in their peer group. The age groups were: 21-30 years; 31-40 years; 41-50 years; 51-60 years; 61-70 years; 71-80 years; and 81 years and over. Participants 20 years old and younger were not included in these analyses because there were only seven patients who were in this age group. The variables examined in the analyses were pain duration, pain severity, pain-related disability, depression and gender. It was not possible to examine most of the other demographic variables across the age groups due to small numbers of non-completers in some of the groups.

The results of these analyses indicated that within each age group completers and non-completers did not differ significantly on any of the variables tested (see Appendix B, Table 2). The only exception to this was depression in the oldest age group. However,

this was not considered significant because there were only four participants in the non-completers group for this analysis.

Furthermore, an examination of the sample by age group indicated that the proportion of the sample in each age group would not change significantly if the non-completers were deleted (see Appendix B, Table 3). For these reasons it was concluded that deleting the non-completers from the main analysis would not bias the sample significantly, and that this was preferable to affecting the psychometric properties of the scale given that examining these properties was one of the main aims of the study.

Deletion of the PCL non-completers from the sample resulted in a sample size of 536 participants for the main analyses.

In this final sample there were 50 missing PCL values (i.e. 0.5% of the total number of PCL items). These values occurred across 44 participants. Again, following Hair et al.'s (2006) advice, the randomness of these missing values was investigated. *t*-test and chi-square analyses were conducted to compare participants with missing PCL items and those without missing items on the demographic, pain-related and clinical variables. These analyses indicated that there were no significant differences between the two groups in age [$t(534) = -0.94$, $p = 0.348$], gender (Fisher's exact test, $p = 0.352$), involvement in a claim or legal case (Fisher's exact test, $p = 0.368$), and whether the participants chose a specific category to describe how their pain began, or chose "other" (Fisher's exact test, $p = 0.598$). Furthermore, there were also no significant differences between the two groups in pain duration [$t(531) = 0.81$, $p = 0.418$], pain severity [$t(519) = -2.34$, $p = 0.02$], pain-related disability [$t(530) = -0.84$, $p = 0.402$] or depression [$t(44) = -2.29$, $p = 0.027$].

In summary, there were no significant differences found between participants with missing PCL items and those with no missing items, indicating that there were no

patterns to these missing values. Given that these missing values constituted a very small proportion of the sample (half a percent) and were randomly distributed, a mean substitution method was employed to estimate the missing values rather than delete another 44 participants. Tabachnik and Fidell (2001) note that mean substitution is acceptable when a very small proportion of the data is missing. Consequently, the missing values for each participant were replaced by their mean item value on the PCL, rather than the mean item value for the sample as a whole.

6.3.3. Main analyses

PCL descriptive statistics

The range of scores, means and standard deviations obtained on the PCL are provided in Table 6.5. PCL total scores ranged from 17 to 80, with a mean PCL total score of 38.24 (SD = 13.97). This mean is considerably lower than the means reported for samples of Vietnam veterans (e.g. M=50.58, SD=24.24; Weathers et al., 1993) and is also lower than the mean reported in a mixed sample of motor-vehicle accident and sexual assault survivors (M= 45.8, SD=16.1; Blanchard et al., 1996a). At the same time, the PCL mean is higher than those reported in studies of breast cancer survivors (e.g. M=29.7, SD=13.0; Cordova et al., 2000) and the means reported in non-clinical samples (e.g. M=29.4, SD=12.9; Ruggiero et al., 2003).

Table 6.5: Descriptive statistics for the PCL Scales

PCL Score	Range	Mean (SD)	95% CI
Total Score	17 – 80	38.24 (13.97)	37.05 – 39.42
Reexperiencing	5 – 24	8.92 (4.55)	8.53 – 9.3
Avoidance	7 – 35	15.97 (6.3)	15.44 – 16.51
Arousal	5 – 25	13.35 (4.8)	12.94 – 13.75

In the case of the PCL subscales, PCL Reexperiencing scores ranged from 5 to 24 (M = 8.92, SD = 4.55), PCL Avoidance scores ranged from 7 to 35 (M = 15.97, SD = 6.3) and PCL Arousal scores ranged from 5 to 25 (M = 13.35, SD = 4.8). In contrast to the PCL total score, the mean obtained for the Reexperiencing subscale was similar to those reported in studies of breast cancer survivors (e.g. M=8.5, SD=4.1; Cordova et al., 2000), patients who have undergone bone marrow procedures (e.g. M=9.61, SD=4.56; Andrykowski et al., 1998), and even non-clinical samples (e.g. M=9.2, SD=4.2; Ruggiero et al., 2003). The means obtained for the Avoidance and Arousal subscales in the current study are higher than those reported in these other PCL studies, and this is particularly the case for the Arousal subscale (c.f. means of 9.2, 9.7 and 8.2 respectively as reported in the three studies cited for the Reexperiencing subscale).

As shown in Table 6.6 three of the lowest item means were found in the Reexperiencing subscale on item 2 (M = 1.57; "Repeated, disturbing dreams of when your pain began?"), item 5 (M = 1.6; "Having physical reactions e.g. heart pounding, trouble breathing, sweating when reminded of when your pain began?") and item 3 (M = 1.62; "Suddenly acting or feeling as if the incident or period when your pain began was happening again, as if you were reliving it?"). The highest mean item scores were for item 13 (M = 3.36; "Trouble falling or staying asleep?"), item 9 (M = 3.1; "Loss of interest in activities that you used to enjoy?") and item 15 (M = 2.92; "Having difficulty concentrating?"). Consistent with this, when an item score of 3 (i.e. "moderately" on the scale provided) or higher is considered symptomatic, as Weathers et al. (1993) suggested, symptom endorsement was highest for items 13 (endorsed by 70% of participants) and 9 (endorsed by 65.5% of participants). The least-frequently endorsed items were items 2 (endorsed by 15.9% of participants) and 5 (endorsed by 17.5% of participants). The mean number of PCL items endorsed was 6.3 (SD = 4.6, range = 0 – 17).

Table 6.6: PCL Item means, standard deviations and percentage of participants rating each item as "3" or above

PCL Subscale	Item	Mean	Standard Deviation	Percentage of participants rating item as "3" or above
Reexperiencing	1	2.10	1.21	33.2
	2	1.57	.99	15.9
	3	1.62	1.01	18.8
	4	2.03	1.27	28.9
	5	1.60	1.03	17.5
Avoidance	6	1.92	1.21	27.4
	7	1.84	1.18	24.4
	8	1.81	1.20	23.7
	9	3.10	1.35	65.5
	10	2.84	1.39	54.9
	11	2.10	1.28	31.7
	12	2.37	1.35	41.4
Arousal	13	3.36	1.38	70.0
	14	2.65	1.28	49.3
	15	2.92	1.29	58.4
	16	2.18	1.25	36.2
	17	2.23	1.26	34.3

Inter-item, Item-total and Subscale Correlations

All inter-item correlation coefficients are presented in Appendix C. PCL inter-item correlation coefficients ranged from 0.20 (the correlation coefficient for items 8 and 12) to 0.66 (for items 1 and 3, and items 2 and 3) on the total scale. For items on the Reexperiencing subscale inter-item correlation coefficients ranged from 0.53 (for items 2 and 4) to 0.66 (for items 1 and 3, and items 2 and 3). Inter-item correlation coefficients for the Avoidance subscale ranged from 0.20 (for items 8 and 12) to 0.64 (for items 9 and 10). Inter-item correlation coefficients for the Arousal subscale ranged from 0.28 (for items 13 and 16) to 0.59 (for items 14 and 15).

Corrected item-total correlation coefficients (i.e. correlations which exclude the relevant item from the total for each correlation) for the full PCL scale ranged from 0.39 (Item 8: "Trouble remembering important parts of the incident or period when your pain

began?") to 0.74 (Item 4: "Feeling very upset when something reminded you of when your pain began?"). Corrected item-total coefficients for the PCL subscales scores ranged from 0.66 to 0.76 for the Reexperiencing subscale, from 0.35 to 0.69 for the Avoidance subscale, and 0.45 to 0.66 for the Arousal subscale (see Table 6.7). Overall, the corrected item-total correlations for the total scale were strong, indicating that these items are all likely to be measuring the same construct. Items 8 ("Trouble remembering important parts of the incident or period when your pain began?") and 13 ("Trouble falling or staying asleep?") were the least correlated with the total score, and were the only two items with item-total correlations below 0.50, indicating that these items may be different to the other items in the scale.

Table 6.7: Corrected item-total correlations and Cronbach's alpha correlation coefficient if item is deleted

Item	**Total Scale**		**Reexperiencing**		**Avoidance**		**Arousal**	
	Item-total	α	Item-total	α	Item-total	α	Item-total	α
1	.665	.915	.756	.843				
2	.587	.917	.705	.857				
3	.630	.916	.740	.849				
4	.740	.913	.723	.854				
5	.646	.916	.664	.865				
6	.592	.917			.547	.810		
7	.602	.916			.537	.811		
8	.391	.922			.355	.838		
9	.601	.917			.628	.796		
10	.686	.914			.690	.785		
11	.659	.915			.648	.793		
12	.659	.915			.612	.799		
13	.462	.921					.450	.800
14	.629	.916					.624	.742
15	.657	.915					.643	.736
16	.570	.917					.525	.773
17	.649	.915					.656	.732

The correlations between the subscale scores indicate a strong correlation between the Avoidance and Arousal subscales (correlation coefficient = 0.74) and the Reexperiencing and Avoidance subscales (correlation coefficient = 0.70). The correlation between the Reexperiencing and Arousal subscales was moderate (correlation coefficient = 0.60).

Internal Consistency

Cronbach's alpha coefficient for the PCL Total score was 0.921, indicating excellent internal consistency. Cronbach's alpha coefficients for the Reexperiencing, Avoidance and Arousal subscales were also high at 0.880, 0.828 and 0.796 respectively.

In the case of the total scale, the Cronbach alpha does not change significantly if any of the items from the scale are deleted (see Table 6.7). The internal consistency of the total scale is not improved to a notable degree by deletion of items 8 or 13, which are the two items with the lowest item-total correlations. Deletion of these two items from the Avoidance and Arousal subscales respectively leads to only a slight improvement in the internal consistency of these two subscales. That is, although items 8 and 13 do not correlate as strongly with the total score as other items, they do not appear to be influencing the internal consistency of the scale to a significant degree.

Both the equal-length and unequal-length Spearman-Brown split-half reliability coefficients for the total scale were 0.83, providing further support for the PCL's reliability. The Guttman split-half correlation coefficient was generated even though the variances of the two split-halves were not markedly different (Part 1: M = 17.58, variance = 55.5; Part 2: M = 20.66, variance = 58.7). Consistent with this, the Guttman reliability coefficient for the total scale also equaled 0.83.

Factor analysis

The Bartlett test of sphericity reached statistical significance (X^2 = 4568.95, p = 000), and the Kaiser-Meyer-Oklin value was 0.94, exceeding the recommended value of 0.6, indicating that the data was suitable for factor analysis.

Using both principal components analysis (PCA) and a principal-axis factor analysis (PAF) only the first two factors had eigenvalues greater than 1, explaining 45% and 9.5% of the variance respectively. Catell's (1966) scree test also suggested that only the first two factors be retained. This was further supported by the results of the parallel analysis, which showed only two factors with eigenvalues exceeding the corresponding criterion values (see Table 6.8). Consequently, two factors were extracted. The initial eigenvalues, scree plot, and the unrotated factor solution for both the PCA and the PAF are provided in Appendix D.

Table 6.8: Comparison of eigenvalues from PCA and PAF and the corresponding criterion values obtained from parallel analysis

Component number	Eigenvalue	Criterion value Mean (SD)	Criterion value 95th percentile
1	7.658	1.3218 (.0331)	1.3707
2	1.619	1.2550 (.0240)	1.2926
3	.987	1.2079 (.0220)	1.2425

The factors were rotated using an oblique (Oblimin) transformation for both the PCA and PAF. The two-factor solution accounted for 54.6% of the variance in the PCA and 49.1% of the variance in the PAF. In both analyses the factors were correlated (PCA = -0.617; PAF = -0.686), justifying the application of an oblique rotation. Table 6.9 provides the loadings and communalities for the two rotated factors for the PCA and PAF. As the table shows, Factor 1 had 9 loadings ≥0.32 in both types of analysis. Following Comrey and Lee's (1992) guidelines, the loadings on Factor 1 ranged from fair

to excellent in both the PCA and PAF, with more than half of the loadings considered very good or excellent. In the PCA, 7 out of the 8 interpretable loadings on Factor 2 were in the "very good" range, and 5 out of 8 were in the "excellent" range.

The loading for item 8 was only just above 0.32 and was well below the loadings for the other items. Consistent with this, item 8 did not load onto either factor in the PAF. Apart from this discrepancy in the factor loading for item 8, the pattern and magnitude of loadings on Factor 2 in the PAF was similar to that obtained in the PCA. An examination of the communalities also indicated that the communality for item 8 in both types of analyses was very low (PCA = 0.197, PAF = 0.163), confirming that the two-factor solution did not adequately account for this item (Tabachnik & Fidell, 2001). The communalities for items 13 (difficulty sleeping) and 16 (hypervigilance) were also lower than the other items in both the PCA and PAF.

The two factors in this solution were labelled, "Numbing/Hyperarousal" (Factor 1) and "Intrusion/Avoidance" (Factor 2). The internal consistency of each of the factors was also calculated. Cronbach's alpha coefficient for the Numbing/Hyperarousal factor was high at 0.886. The internal consistency of this factor did not improve markedly if Items 13 or 16 were deleted (see Table 6.10). However, consistent with the relatively low communalities for these two items, their correlations with the total factor score were lower than the other items. Cronbach's alpha coefficient for the Intrusion/Avoidance factor was 0.879. This improved to 0.892 if Item 8 was deleted. Consistent with this, the correlation of Item 8 with the total factor score was noticeably lower than the other items which loaded onto this factor. This is also consistent with the low loading of Item 8 on this factor in the PCA, and its failure to load onto either factor in the PAF.

Table 6.9: Pattern matrices (loadings)* and communalities (h2) for the rotated two-factor solution using PCA and PAF

PCA				PAF			
PCL Item	**1**	**2**	**h^2**	**PCL Item**	**1**	**2**	**h^2**
10 (feeling distant/ cut-off)	**.869**	.069	.686	10 (feeling distant/ cut-off)	**.879**	.107	.654
14 (irritability/ anger)	**.790**	.046	.582	11 (emotionally numb)	**.759**	.020	.513
11 (emotionally numb)	**.783**	.007	.607	14 (irritability/ anger)	**.741**	.037	.556
15 (difficulty concentrating)	**.764**	-.008	.591	15 (difficulty concentrating)	**.730**	-.002	.535
9 (loss of interest)	**.724**	.011	.514	9 (loss of interest)	**.668**	-.001	.447
12 (future cut short)	**.661**	-.123	.553	12 (future cut short)	**.626**	-.117	.506
17 (easily startled)	**.631**	-.139	.526	17 (easily startled)	**.589**	-.135	.475
13 (trouble with sleep)	**.598**	.040	.330	13 (trouble with sleep)	**.489**	-.021	.254
16 (super-alert)	**.466**	-.224	.397	16 (super-alert)	**.428**	-.210	.352
3 (reliving the event)	-.107	**-.890**	.686	3 (reliving the event)	-.119	**-.875**	.637
1 (intrusive memories or thoughts)	-.001	**-.823**	.675	1 (intrusive memories or thoughts)	-.015	**-.808**	.637
2 (dreams)	-.077	**-.814**	.591	2 (dreams)	-.060	**-.760**	.519
5 (physical reactions to reminders)	.049	**-.743**	.599	5 (physical reactions to reminders)	.070	**-.682**	.536
7 (avoidance of situations)	.008	**-.737**	.550	4 (distress when reminded)	.181	**-.680**	.472
4 (distress when reminded)	.186	**-.703**	.691	7 (avoidance of situations)	.050	**-.652**	.664
6 (avoidance of thoughts)	.059	**-.670**	.501	6 (avoidance of thoughts)	.107	**-.575**	.426
8 (amnesia)	.165	**-.323**	.197	8 (amnesia)	.180	-.258	.163

*Factor loadings ≥0.32 are listed in bold.

Table 6.10: Corrected item-total factor correlations and Cronbach's alpha correlation coefficient for factors if item is deleted

Factor	Item	Factor Total	
		Corrected Item-total correlation	α if Item deleted
Numbing/Hyperarousal	9	.621	.875
	10	.744	.864
	11	.692	.869
	12	.663	.871
	13	.474	.887
	14	.669	.871
	15	.681	.870
	16	.541	.881
	17	.652	.872
Intrusion/Avoidance	1	.714	.856
	2	.643	.865
	3	.721	.857
	4	.750	.852
	5	.693	.860
	6	.641	.864
	7	.653	.863
	8	.373	.892

Although the multiple criteria applied indicated that extraction of two factors was appropriate, problems with all methods (especially the eigenvalue criterion and the scree test, but also parallel analysis) have been identified, (Zwick & Velicer, 1986; Turner, 1998; Hayton, Allen & Scarpello, 2004). Given that the next initial eigenvalue was 0.987 a three-factor solution was also extracted. A three-factor solution was also worth investigating given the three symptom cluster conceptualisation of PTSD in the DSM-IV. This analysis was rejected because it resulted in a poor factor solution for both the PCA and the PAF, with several cross-loadings and lower factor loadings than the two-factor solution. The results of this three-factor solution are provided in Appendix D.

Application of a diagnostic algorithm and cut-off scores

Following previous research, two cut-off scores were applied to the PCL data: Weathers et al.'s (1993) original cut-off score of 50 (derived from a Vietnam veteran sample) and Sherman et al.'s (2005) cut-off of 41 (derived from a sample of orofacial pain patients). When the cut-off score of 50 was applied, 111 patients (20.7% of the sample) could be classified as potentially meeting criteria for a diagnosis of PTSD. When the cut-off score of 41 was applied, 195 patients (36.4% of the sample) could be classified as potentially meeting diagnostic criteria.

The diagnostic algorithm suggested by Weathers et al. (1993) was also tested in this sample. As described in the Method, this algorithm follows the DSM-IV criteria for PTSD and defines endorsement of a symptom as a score of 3 or above. When applied to this sample, 162 patients (30.2% of the sample) could be classified as meeting diagnostic criteria for PTSD.

6.4. Discussion

The aim of this study was to assess the performance of a modified version of the PCL in a sample of chronic pain patients. The PCL was modified to allow participants to respond with reference to the event associated with the onset of their pain, or the period during which their pain began if the onset was gradual or spontaneous. The key objectives of the study were: (1) to assess patients' ability to complete the PCL with the modified instructions; (2) to compare the sample's responses on the PCL to studies using the PCL in other populations; (3) to examine the psychometric properties of the PCL in a chronic pain sample; (4) to investigate the factor structure of the modified PCL in a chronic pain sample; and (5) to apply a diagnostic algorithm and cut-off scores as suggested in the literature to determine the proportion of the sample that could be classified as meeting

diagnostic criteria for PTSD based on their PCL responses. Each of these objectives will be discussed in the following sections.

6.4.1. The modified PCL

In terms of the first objective, the vast majority of the participants in the study completed the PCL with the modified instructions, indicating that it is possible to ask chronic pain patients to respond to a PTSD self-report measure with specific reference to the onset of their pain. Although the missing values analysis was initially intended to be part of the preliminary examination of the data and not the main analyses, it actually assisted in achieving the study's first objective by revealing the groups of participants who may have had difficulties completing the modified PCL. In particular, the analysis indicated that participants who did not complete the PCL were older as a group and were less likely to be involved in a claim or legal case than those who completed the PCL. In turn, participants not involved in such a claim were older than those who were. Patients who did not complete the PCL were also more likely to choose the "other" category when describing how their pain began (as opposed to choosing one of the specific categories provided).

There are a number of possible explanations for these results. Firstly, this latter finding may be accounted for by the observation that since compensation claims and legal suits tend to be the result of an injury or accident, those not involved in such a claim may have found it more difficult to choose one of the specific categories to describe how their pain began. Importantly, it is possible that these patients also found it more difficult to complete the PCL because they did not have a specific event or incident to refer to when answering the items. However, the finding that older participants were less likely to finish the battery of questionnaires also suggests that the PCL may not have been completed by

these patients because it was positioned last in the battery, rather than it being due to difficulties answering the items with reference to the onset of their pain.

The limitations of the study, being an initial examination of the performance of the modified PCL in a chronic pain sample, render it impossible to determine which of the above explanations fully accounts for the participants who did not complete the PCL. The first limitation involves the categories used in the demographic questionnaire to collect data about onset of pain. The categories did not clearly distinguish between pain that began with a specific event and pain that developed spontaneously or gradually. For example, responses to the categories "other" and "multiple cause" could be classified as both sudden and gradual/spontaneous onset. This means that some of the patients who chose these options could have experienced a sudden onset of pain that they did not think fit into one of the other categories. Consequently, it is not possible to determine if the patients who chose these categories found it more difficult to complete the PCL because they had experienced a spontaneous or gradual onset of pain. Secondly, the PCL's position in the battery was not varied in the study, so the impact of position on completion rates also remains unclear.

Future research can investigate this issue further by ensuring that participants can easily be classified into the different types of onset of pain. Classifying patients into groups according to the different types of onset of pain could also have allowed a comparison of PCL responses across this variable. For example, it could be hypothesised that patients who have experienced a sudden and/or traumatic onset of pain report higher levels of prototypic PTSD symptoms (e.g. the reexperiencing symptoms) than patients who experienced a gradual or spontaneous onset of pain.

6.4.2. Comparisons with PCL studies in other populations

The PCL scores obtained in this study are consistent with previous research reporting substantial levels of PTSD symptomatology in chronic pain samples (e.g. Sherman et al., 2005; Von Korff et al., 2005; Sterling & Kenardy, 2006). Participants in this study reported being at least moderately bothered by an average of six to seven symptoms, and 14 out of the 17 items were endorsed at the moderate level or above by at least 20% of the sample. Six symptoms were endorsed at the moderate level or above by at least 40% of the sample. Even the least endorsed symptoms (i.e. recurrent nightmares and physiological reactivity to reminders of the onset of pain) were reported at the moderate level or above by more than 15% of patients.

The PCL total score also provided support for noteworthy levels of PTSD symptoms in this sample compared with other studies that have utilised the PCL. As would be expected given prior research reporting elevated rates of PTSD symptomatology in chronic pain samples, the mean PCL total score was higher than that reported in non-clinical samples (e.g. Ruggiero et al., 2003). The mean obtained in this study was also higher than that reported in studies of breast cancer survivors (e.g. Cordova et al., 2000), which is a medical population recognised as being at risk of developing PTSD. However, given that not all chronic pain patients have experienced a traumatic event, it is not surprising that the mean was not as high as those reported in studies of typical PTSD populations such as sexual assault victims or Vietnam veterans (e.g. Weathers et al., 1993; Blanchard et al., 1996a).

Examination of the pattern of responses on the PCL raises a number of important issues. It is important to note that the most commonly endorsed symptoms on the PCL were problems widely reported by chronic pain patients that are not exclusive to PTSD. These symptoms were disturbed sleep, loss of interest in previously enjoyable activities,

and difficulty concentrating. These three symptoms and a number of others (particularly those included in the Arousal subscale), were endorsed at a moderate level or above by at least 30% of patients. Item 13 ("Trouble falling or staying asleep") was endorsed at a moderate level or above by 70% of the sample. Difficulty with feelings of irritability or anger was reported at a moderate level or above by almost 50% of patients. Given the widespread nature of these types of problems in chronic pain patients, it is likely that the high rates of these symptoms reported in the current study are at least partly attributable to chronic pain as opposed to PTSD. Additional evidence that symptom endorsement may have been related to chronic pain and not PTSD comes from comparisons of the sub-scale scores obtained in this study and those obtained in studies of other groups. Although the mean score on the Reexperiencing sub-scale was similar in this study to those reported in breast cancer and non-clinical samples, scores on the Avoidance and Arousal sub-scales (which contain most of the problems that are arguably common in chronic pain samples) were higher.

This is a similar problem to that which has been noted when assessing depression in chronic pain patients since some self-report measures of depression contain somatic symptoms that can be attributable to chronic pain and its sequelae rather than depression (e.g. fatigue, sleeping problems, reduced appetite; Williams & Richardson, 1993; Taylor et al., 2005). As these authors and several others (e.g. Romano & Turner, 1985; Benjamin, Lennon & Gardner, 1991; Wilson, Mikail, D'Eon & Minns, 2001) have noted, the overlap between symptoms of depression and problems associated with chronic pain, and the inclusion of somatic items in self-report measures of depression can lead to an inflation of reported rates of depression in chronic pain patients. Consequently, investigators have attempted to identify self-report measures of depression that do not include somatic items (such as the DASS) and the evidence to date confirms that such

measures are more accurate in chronic pain patient samples (Taylor et al., 2005). Similar avenues of investigation may need to be pursued in studies of chronic pain and PTSD in order to find a self-report measure of posttraumatic stress symptoms that takes into account the issue of symptom overlap. Alternatively, it may be possible to address this issue by identifying a diagnostic algorithm or cut-off score that maximises sensitivity and specificity in a chronic pain sample. This will be discussed in further detail in Section 6.4.5.

Although it is possible that the PCL scores in this study were inflated by the symptom overlap between chronic pain and PTSD, it is important to note that symptoms considered hallmarks of post-traumatic stress (e.g. intrusive recollections of the traumatic event, nightmares, distress when faced with reminders of the event) were endorsed at a moderate level or above by a considerable number of patients. For example, 33.2% of the sample reported being at least moderately bothered by intrusive recollections of the onset of their pain, 15.9% reported being at least moderately bothered by dreams about the onset of their pain, and 28.9% reported being at least moderately bothered by distress when faced with reminders. These figures indicate that even if symptom overlap is a problem in studies of chronic pain and PTSD, a significant proportion of chronic pain patients report PTSD symptoms that cannot be attributed to their pain.

At the same time, however, as O'Donnell, Creamer, Bryant, Schnyder and Shalev (2003) have pointed out, individuals who have experienced severe injuries also ruminate about the injury, its consequences, and associated physical symptoms (including pain), and it is important to distinguish between voluntary rumination over these issues and the intrusive, involuntary reexperiencing symptoms associated with PTSD. The wording of items 1 ("Repeated, disturbing memories, thoughts, or images of when your pain began") and 4 ("Feeling very upset when something reminded you of when your pain began") in

particular may not adequately capture the difference between the reexperiencing symptoms of PTSD and the rumination that is common to a range of other clinical conditions. Consistent with this, these two items were endorsed more frequently in this sample than other items in the Reexperiencing sub-scale.

Further evidence that the PCL does not differentiate adequately between the symptomatology of PTSD and chronic pain could also come from comparisons of responses on the PCL with the results of structured clinical interviews since interviews allow the clinician or researcher to ensure that the individual understands the exact nature of the symptoms being enquired about. In addition to providing a more accurate assessment of PTSD symptomatology, structured clinical interviews also assess the DSM-IV stressor criterion. In the current study, posttraumatic stress symptoms were assessed, but no attempt was made to ensure that the events associated with the participants' onset of pain actually satisfied the definition of a traumatic event. Although Sherman et al. (2005) reported that the PCL exhibited good predictive validity when compared with the SCID-IV in a sample of orofacial pain patients, comparisons between the PCL and structured clinical interviews need to be conducted in samples of heterogeneous chronic pain patients. The lack of interview data (or any other corroborating data) to assess the diagnostic utility of the PCL in this sample is an important limitation of the current study.

6.4.3. Psychometric properties of the PCL

Another objective of this study was to examine the psychometric properties of the PCL in a chronic pain sample. The Cronbach's alpha coefficients and split-half reliability coefficients indicated that the PCL exhibited excellent reliability in this patient group. The item-total correlation coefficients also provided support for the internal consistency

of the PCL. Only two item-total correlations were below 0.50; those for items 13 and 8. It is possible that the low item-total correlation for item 13 is related to the symptom overlap issue discussed above; that is, since disturbed sleep is a common aspect of the chronic pain experience and is not exclusive to PTSD this item may perform differently to other items on the PCL. However, given that the other items that also assess overlapping symptoms of the two conditions exhibited higher item-total correlations this explanation does not adequately account for the performance of item 13. The low item-total correlation for item 8 may be related to onset of pain. That is, some patients may have found it difficult to answer a question asking them if they have trouble remembering aspects of the onset of their pain if their pain developed gradually. Other PTSD research has also identified problems with the amnesia item and this research will be discussed further in the following section.

The correlations between the subscale scores also supported the psychometric properties of the PCL, indicating a moderate to strong relationship between the subscales. This is consistent with previous investigations of the PCL (e.g. Ventureyra, Yao, Cottraux, Note & De Mey-Guillard, 2002; Ruggiero et al., 2003).

Although the results of this study indicated that the PCL exhibited good psychometric properties in a chronic pain sample, the conclusions are limited by the fact that convergent validity was not examined. High correlations between the PCL and other established self-report measures of PTSD could have provided evidence that the PCL measures the same construct that other PTSD questionnaires are purported to be measuring. Although there is support from previous studies for the convergent validity of the PCL (e.g. Weathers et al., 1993; Ruggiero et al., 2003), this question has not been examined in the area of chronic pain. Future studies should evaluate the validity of the PCL using both structured clinical interviews and other measures of PTSD.

6.4.4. Factor structure of the PCL

Exploratory factor analysis of the PCL data in this study supported a factor structure previously reported in the literature. Both the prinicipal components analysis and prinicipal axis factor analysis revealed a two-factor solution. The first, labelled "Numbing/Hyperarousal", consisted of nine out of the ten numbing and hyperarousal symptoms as described in DSM-IV (i.e. items 9 to 17). The second factor, labelled "Intrusion/Avoidance", consisted of the five reexperiencing symptoms (items 1 to 5) and the two avoidance symptoms (items 6 and 7).

The remaining item, item 8, loaded weakly onto Factor 2 in the PCA, and did not load onto either factor in the PAF. This has been reported previously in factor analytic studies of PTSD symptoms, with several studies finding that item 8 loads weakly, or not at all, onto identified factors (e.g. Foa, Riggs & Gershuny, 1995; Buckley, Blanchard & Hickling, 1998; King, Leskin, King & Weathers, 1998a; Taylor, Kuch, Koch, Crockett & Passey, 1998). This has been attributed to the relatively low prevalence of amnesia for traumatic events and difficulties assessing memory deficits, particularly based on self-report (King et al., 1998a; Palmieri et al., 2007). In addition, it has also been suggested that amnesia for traumatic events may not be a central feature of PTSD (Palmieri & Fitzgerald, 2005). Consistent with this, in the current study the correlations between item 8 and both the Total score and the Factor 2 total were low, suggesting that this item is different to the other items on the PCL.

The two-factor model identified in this study is consistent with the only other study to investigate the factor structure of the PCL in a sample of chronic pain patients. Asmundson et al. (2003) applied confirmatory factor analysis in a large sample of United Nations peacekekeepers with chronic pain and reported that this two-factor model provided a good fit to the data. The main difference between the solution obtained in this

study and the model tested by Asmundson and colleagues is that the latter included Item 8 on the Numbing/Hyperarousal factor. A two-factor model has also been supported in other explanatory and confirmatory factor analytic studies of DSM-IV PTSD symptoms (Buckley et al., 1998; Taylor et al., 1998); however, neither of these studies used the PCL and both reported that the hypervigilance and exaggerated startle response items loaded onto the "Intrusion/Avoidance" factor, contrary to the findings of the current study. From a theoretical perspective, it has been noted that although a two-factor model is inconsistent with the DSM-IV conceptualisation of PTSD, it is consistent with other conceptual models, for example, Foa, Zinbarg and Rothbaum's (1992) model in which avoidance and numbing are two distinct mechanisms that occur in response to intrusive and hyperarousal symptoms respectively (Asmundson et al., 2003). Given the implications for understanding the mechanisms underlying chronic pain and PTSD when they co-occur, studies testing the validity and replicability of the two-factor models described above in other sample of chronic pain patients are warranted.

6.4.5. Application of the diagnostic algorithm and cut-off scores

The application of cut-off scores and the diagnostic algorithm derived from previous research in the current sample of chronic pain patients suggested that a significant proportion of the sample (up to 36%) could potentially be diagnosed with PTSD. While this is consistent with reports of high levels of PTSD symptoms in chronic pain patient groups presenting for treatment following a traumatic event (e.g. MVA; Hickling & Blanchard, 1992), this proportion is much higher than the rates typically reported in chronic pain clinic settings (Muse, 1985; Aghabeigi et al., 1992; Sherman et al., 2005). There are a number of possible explanations for the lower rates reported in these studies. Firstly, the three studies cited here utilised structured clinical interviews

(Aghabeigi et al., 1992; Sherman et al., 2005) or an interview conducted in a clinical setting (Muse, 1985) to diagnose PTSD, rather than a self-report measure. As noted earlier, structured clinical interviews may provide more accurate estimates of prevalence by clarifying the nature of the symptoms being reported, and by ensuring that the symptoms are related to PTSD and not to other conditions (such as chronic pain). Secondly, the two studies that utilised structured clinical interviews were conducted with orofacial pain patients. It is possible that the prevalence of PTSD is higher in samples of heterogeneous chronic pain patients as such a sample may reflect exposure to a wider range of traumatic events. Finally, the Aghabeigi et al. and Muse studies both employed small samples (N = 34 and 64, respectively). All of these factors may have contributed to the lower prevalence rates reported in these studies.

These methodological differences aside, it is not possible to ascertain the accuracy of the diagnostic algorithm or cut-off scores in the current study given the lack of corroborating information from other sources. At the same time, the high rate of possible PTSD diagnoses identified by the diagnostic algorithm and cut-off scores (particularly when compared to studies utilising structured clinical interviews) calls into question the validity of the diagnostic algorithm and cut-off scores when applied in chronic pain settings. As noted earlier, the overlap between the symptoms of PTSD and problems associated with chronic pain may have led to inflated PCL scores. However, the fact that the factor structure identified in the exploratory factor analysis is similar to those reported in other studies suggests that the PCL was measuring the same construct in this chronic pain sample as it does in other groups. This could be considered as evidence against the notion that symptom overlap impacted negatively upon the scale's reliability in this sample.

6.5. Summary

The aim of this study was to examine the psychometric properties of the PCL in a large sample of heterogeneous chronic pain patients. The limitations of previous research in the field of chronic pain and PTSD were addressed by modifying the PCL so that participants were able to respond to the self-report measure with specific reference to the event associated with the onset of their pain.

Overall, the results provided preliminary support for the suitability of the PCL as a self-report measure of PTSD symptoms in chronic pain patients. Participants were able to complete the PCL with reference to the onset of their pain and the PCL exhibited good psychometric properties in this patient group. Exploratory factor analyses identified a two-factor solution similar to others reported in previous factor analytic studies of PTSD symptomatology, providing support for the construct validity of the PCL in a chronic pain setting.

However, as already noted, there were a number of limitations to the current study. Firstly, it was limited by the absence of PTSD symptom data from other sources. Consequently, it was not possible to: (1) examine the diagnostic utility of the PCL in a chronic pain patient sample; (2) assess the impact of symptom overlap on PCL scores; and (3) fully ascertain the accuracy of the diagnostic algorithms or cut-off scores suggested in previous research. Secondly, participants were treated as one group despite the fact that only some would have experienced an onset of pain consistent with the DSM-IV definition of a traumatic event. This could have been reflected in difficulties completing the PCL (e.g. for patients who had experienced a gradual/spontaneous onset of pain). In addition, classifying participants according to the onset of their pain could have allowed group comparisons in PCL responses.

The factor analyses conducted in the current study may be particularly important for an improved understanding of the mechanisms underlying the relationship between chronic pain and PTSD. As far as the author is aware, this study represents the first attempt to investigate the factor structure of the PCL in a sample representative of patients who typically present to multidisciplinary pain management centres. Further studies are needed to determine if the two-factor structure identified in this study can be replicated in other samples of heterogeneous chronic pain patients.

7. STUDY 2 - IMPACT OF TYPE OF ONSET OF PAIN ON PAIN-RELATED ADJUSTMENT

7.1. Introduction

Study 1, presented in the previous chapter, provided preliminary support for the suitability of a modified version of the PCL as a self-report measure of PTSD symptoms in a chronic pain clinic setting. An examination of the PCL data in Study 1 revealed a high rate of endorsement of PTSD symptomatology in the sample of heterogeneous chronic pain patients. Application of a diagnostic algorithm and cut-off scores recommended in earlier studies suggested that up to 36% of the sample could potentially meet DSM-IV diagnostic criteria for PTSD.

Unfortunately, it was not possible to determine the accuracy of this figure since the PCL was the only measure of PTSD symptoms in the study. Detailed information about the participants' onset of pain was also not collected, rendering it impossible to determine accurately what proportion of the sample actually experienced a traumatic onset of their pain condition. Consequently, in Study 2, in order to examine the diagnostic utility of the PCL in a chronic pain clinic setting, information about both onset of pain and posttraumatic stress symptoms was collected from the participants' medical files.

Study 2 is primarily an investigation of the impact of type of onset of pain on pain severity and pain-related adjustment. As argued in Chapter 2, previous studies in this area have typically grouped together patients with accident-related pain and patients who attribute their pain to other specific events and have compared them to patients who have experienced an insidious or spontaneous onset of pain. As a result, it is not clear whether findings that the former group of patients exhibit poor adjustment to chronic pain when compared with the latter group can be interpreted as support for the importance of

developing pain following an accident, or the importance of developing pain following any specific event. This question is addressed in this study by comparing three groups of patients: (1) individuals who attribute their pain to an accident (Accident); (2) individuals who attribute their pain to a specific incident that is not an accident (Specific Incident); and (3) individuals who report that they experienced an insidious or spontaneous onset of pain (Insidious/Spontaneous).

A second question arising from this area of research is the degree to which the disability and distress reported by chronic pain patients with accident-related pain is attributable to these individuals having experienced the onset of pain in the context of an event that could potentially be experienced as traumatic. Consequently, this study also compared the following groups on pain severity and pain-related adjustment: (1) patients who experienced a traumatic onset of pain (Traumatic); (2) patients who experienced a non-traumatic (but sudden) onset of pain (Non-traumatic); and (3) patients who reported an insidious or spontaneous onset of pain (Insidious/Spontaneous).

The psychometric properties of the PCL were also examined further in Study 2. Correlations between the PCL and other self-report measures administered in the study were examined to evaluate the construct validity of the PCL. Additional evidence that the PCL was a valid measure of posttraumatic stress symptoms in a chronic pain sample was sought by comparing the PCL scores of the Traumatic, Non-traumatic, and Insidious/Spontaneous groups.

In addition to allowing examination of the diagnostic utility of the PCL, obtaining information about onset of pain also ensured that two of the issues discussed in Chapter 4 regarding the literature pertaining to chronic pain and PTSD were addressed. Specifically, by evaluating the traumatic nature of the events associated with the participants' onset of pain it was possible to determine whether the posttraumatic stress symptoms that

participants reported on the PCL were actually related to a traumatic event. In turn, the modification made to the PCL in Study 1 ensured that the posttraumatic stress symptoms the participants reported were related to the onset of pain and not prior or subsequent traumatic events.

In summary, the aims of Study 2 were to investigate questions pertaining to the impact of type of onset of pain on pain-related adjustment, to further evaluate the psychometric properties of the PCL, and to address some of the methodological shortcomings of Study 1 in order to examine the diagnostic utility of the modified PCL in a chronic pain clinic setting.

7.2. Method

7.2.1. Participants

The sample consisted of 206 individuals, including 77 males (37.4%) and 129 females (62.6%), referred to the University of Sydney Pain Management and Research Centre at Royal North Shore Hospital. These individuals underwent a multidisciplinary assessment at the Centre on their first visit between October 2004 and April 2005. Initially, 238 patients were identified as participants in Study 2; however, 32 patients were excluded for the following reasons. One patient was excluded because he or she had not provided any identifying details. Another 15 patients (6.3% of the initial sample) were excluded because they had not completed the demographic questionnaire. Sixteen patients (6.7% of the initial sample) were excluded because they did not provide consent for information collected about them to be used in research at the Centre. The demographic characteristics of these 16 individuals were compared to the characteristics of those who did consent, and the outcomes of these analyses are provided in the Results section. A

complete description of the sample who participated in the study is also provided in the Results section.

7.2.2. Procedure

Information regarding the demographic and clinical characteristics of the sample was obtained from the same demographic questionnaire described in the Method section for Study 1. Ethics approval for Study 2 was provided by the Northern Sydney Health Human Research Ethics Committee. Consent to participate in the study was obtained in the same manner as Study 1.

7.2.3. Classification of participants into onset groups

The demographic questionnaire contained one question enquiring about onset of pain with the following question: "How did your pain begin? *(tick ONE – if more than one applies, select the one which applies BEST).*" Participants had a choice of the following options: (1) Accident at work; (2) At work but not involving an accident; (3) Accident at home; (4) Motor vehicle accident; (5) After surgery; (6) After an illness; (7) Pain just began, no clear reason; (8) Other *(specify).*

For the first group comparison (i.e. the comparison of Accident, Specific Incident and Insidious/Spontaneous groups) options 1, 3, and 4 above were classified as "Accident", options 5 and 6 were classified as "Specific Incident", and options 2 and 7 were classified as "Insidious/Spontaneous". The "Other" option was classified into one of the three groups according to the description provided. For example, the response "Fall from a horse" was classified as "Accident".

For the second group comparison (i.e. Traumatic, Non-traumatic, and Insidious/Spontaneous) the responses to the above question were used to identify participants who had experienced an onset of pain attributed to any specific incident (i.e.

options 1, 3, 4, 5 and 6, and some responses from the "Other" category as appropriate). For these participants detailed information about the specific incident was obtained from their medical files and provided to two independent experts in the area of PTSD to code. The information included descriptions of the incident taken from file notes and medical reports. The two experts were PhD-level clinical psychologists with clinical and research experience in the area of PTSD, including extensive experience in the administration of structured clinical interviews for PTSD. The full set of instructions provided to the coders is provided below. A copy of the coding sheet is provided in Appendix E.

> Based on the information provided for each participant please indicate if the event that triggered the onset of pain meets the following criteria:
>
> 1. DSM-IV criterion A1 - "The person witnessed, experienced or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others".
>
> 2. DSM-IV criterion A2 - "The person's response involved intense fear, helplessness, or horror".
>
> 3. In the final column please indicate if, in your opinion, the event could be considered potentially traumatic i.e. even if it is not clear from the available information if the event was traumatic in this particular case, it is possible (based on your knowledge of the events typically considered to be potentially traumatic).

Initially, it had been decided that an event would be classified as traumatic if both coders agreed that it met DSM-IV Criteria A1 and A2. However, when collecting information from the participants' medical files it became apparent that for some cases there was insufficient information to make a judgement about the traumatic nature of the event. In particular, although the event itself was typically described in detail, the participant's response at the time was not always documented. Consequently, coders were given the option of noting that there was insufficient information to make a judgement about Criterion A2. In addition, for every event they were asked to make a judgement about whether the event could be considered potentially traumatic based on their clinical

and research experience (Item 3 in the above instructions). If both coders agreed that an event met both Criteria A1 and A2 or judged the event to be potentially traumatic the participant was added to the traumatic onset group. All other participants who had experienced onset of pain related to a specific incident were placed in the Non-traumatic group. Participants who had experienced an insidious/spontaneous onset of pain were placed in the Insidious/Spontaneous group.

7.2.4. Measures

The same questionnaire battery used in Study 1 was administered in this study. The questionnaires used in this study included the MPI Pain Severity scale, the modified RMDQ, and the DASS. Since these measures were described in detail in the previous chapter they will not be described again here. The modified PCL was also administered as part of this questionnaire battery as it was in Study 1.

7.2.5. Aims and hypotheses

The aims of this study were to investigate the impact of type of onset of pain on pain severity and adjustment-related variables, and to examine the psychometric properties of the PCL in a chronic pain clinic setting. There were four key objectives associated with the study. Two of these objectives were concerned with the impact of type of onset of pain on pain and pain-related adjustment. The remaining objectives focused on the validity and diagnostic utility of the PCL in a chronic pain patient sample. The objectives were: -

(1) To determine if patients in the Accident group report higher levels of pain severity and exhibit poor adjustment to chronic pain in comparison to patients in the Specific Incident and Insidious/Spontaneous groups.

It was predicted that, compared with the other two onset groups, patients in the Accident group would report higher levels of pain severity, pain-related disability, and symptoms of depression, anxiety, and stress. No significant differences were expected between the Specific Incident and Insidious/Spontaneous groups.

(2) To determine if patients in the Traumatic onset group report higher levels of pain severity and exhibit poor adjustment to chronic pain in comparison to patients in the Non-traumatic and Insidious/Spontaneous groups.

It was predicted that, compared with the other two onset groups, patients in the Traumatic onset group would report higher levels of pain severity, pain-related disability, and symptoms of depression, anxiety, and stress. Based on the evidence from the literature that onset of pain associated with accidents is associated with poor adjustment, it was also predicted that the Non-traumatic group would report higher levels of pain severity and exhibit poor adjustment to chronic pain when compared with the Insidious/Spontaneous onset group (because it was expected that a large proportion of the Non-traumatic group would have accident-related pain).

(3) To evaluate the construct validity of the PCL by examining the relationship between the PCL and other self-report measures administered in the study, and by comparing the levels of PTSD symptoms endorsed by the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. It was predicted that the Traumatic onset group would report significantly higher levels of PTSD symptoms compared with the other two groups, providing support for the validity of the PCL in a chronic pain sample. It was also expected that because these measures assess similar constructs, correlations between the PCL and the three DASS scales would be higher than the correlation between the PCL and a measure that assesses a distinct construct (i.e. pain-related disability as measured by the RMDQ).

(4) To examine the diagnostic utility of the PCL in a chronic pain clinic setting by comparing PCL responses with details of PTSD symptoms obtained from participants' medical files. Based on PTSD prevalence rates from previous studies of individuals with chronic pain and the results of Study 1, it was predicted that a diagnostic algorithm and cut-off scores suggested in previous research would overestimate the proportion of participants in the sample who meet DSM-IV diagnostic criteria for PTSD.

7.2.6. Data analyses

All analyses were conducted using the SPSS v. 16.0 for Windows. Statistical significance was set at $p < 0.05$.

To determine if the sample was representative of chronic pain patients typically presenting to the Centre the participants' demographic and pain-related characteristics and mean scores on the self-report measures were compared with the Centre's normative data sample (Nicholas et al., 2008).

Secondly, the demographic characteristics of the 16 individuals who did not provide consent to be involved in the research were compared to the characteristics of those who did consent. These comparisons were conducted using Student's *t*-tests for age and pain duration, and Fisher's exact tests for gender and whether the visit to the Centre was related to a compensation claim or legal case.

Missing values analyses

The results of the missing values analyses are described in the Results section.

The criteria used in Study 1 to classify the self-report measures as incomplete were applied in this study. Twelve patients (or 5.8% of participants) were classified as non-completers. In order to determine if these participants were different to those who completed the questionnaires, the two groups were compared on a number of key

demographic and clinical variables using Student's *t*-tests, chi-square tests or Fisher's exact tests as appropriate. Potential patterns in missing values were also investigated by comparing participants who had completed the PCL with those who had not on the main demographic and clinical variables.

To address the question raised by Study 1 regarding the ability of patients with an insidious/spontaneous onset of pain to complete the modified PCL, a chi-square analysis comparing the completion rates across onset groups (i.e. Accident, Specific Incident, and Insidious/Spontaneous) was conducted.

The missing values analysis also involved examining the types and extent of missing data amongst completers. Following Tabachnik and Fidell (2001), it was not considered a significant problem if less than 5% of data points were missing from a particular variable in a random pattern. Variables with more than 5% of data points missing were examined in further detail by testing for patterns in the missing data.

Main analyses

The first two study objectives were addressed with two groups of analyses. The first set of analyses compared the Accident, Specific Incident, and Insidious/Spontaneous groups. The second set of analyses compared the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. Both of these group comparisons were made by fitting the data to separate General Linear Models (GLMs) with onset group as the predictor variable and continuous measures of pain severity, pain-related disability, and affective distress (i.e. MPI – Pain Severity, RMDQ, and the DASS Depression, Anxiety and Stress scales) as outcome variables.

As the results of previous studies (Geisser, Roth, Bachman & Eckert, 1996; Turk, Okifuji, Starz & Sinclair, 1996) have reported differences between onset groups, cross-

sectional comparisons of the groups were made using ANOVA and chi-squared tests as appropriate. Variables identified as being significantly different ($p < 0.05$) between the onset groups were used in the GLMs to control for imbalance. The variables investigated included age, pain duration, compensation status, and gender.

The third study objective was addressed by investigating the relationship between the PCL and the three DASS scales using Spearman's Rank Order Correlation coefficients. This non-parametric statistic was selected because the PCL Total score and the DASS scales were not normally distributed. The Pearson Product-Moment Correlation coefficient was calculated to measure the relationship between the PCL and the RMDQ. The third objective was also addressed by fitting the data to separate GLMs with Traumatic onset group status as the predictor variable, the PCL Total score and subscales as outcome variables, and the aforementioned demographic variables as covariates (if there were significant differences between the groups on those variables).

Testing of assumptions underlying the GLM (e.g. normality, homoscedasticity of errors) was conducted and included a combination of examination of normal probability plots and scatterplots of studentized residuals against predicted values, and the Shapiro-Wilks test for normality (Shapiro & Wilk, 1965) applied to the distribution of studentized residuals. Cases with Cook's distance values greater than one (Cook & Weisberg, 1982) and studentized residuals greater than |2.75| were examined further in order to identify potentially influential cases and possible outliers. Non-parametric statistics were used when assumptions for parametric methods were violated. Specifically, the Kruskal-Wallis test and the median test were used. Residual plots and the other tests used to evaluate the assumptions of the GLMs (including statistics for transformed outcome variables) are provided in Appendix F. All tests were two-tailed. Statistical significance was set at $p < 0.05$.

7.3. Results

7.3.1. Participant characteristics

The demographic and pain-related characteristics of the participants in this study and the Centre's normative data sample (Nicholas et al., 2008) are presented in Table 7.1. The mean age of participants was 53 years (SD = 16; range = 21 to 88 years of age), and the mean duration of pain was 90.2 months or approximately 7½ years (SD = 112.8; range = 3 to 568 months). The most common single pain site was lower back and lower limbs (53 individuals or 25.7% of the available data for this characteristic), with 64 patients (or 31.1% of available data) reporting pain in two or more major sites. Seventy-two participants (34.9%) were visiting the Centre about a pain condition related to a compensation claim or other legal case. As can be seen in Table 7.1, the sample was typical of the Centre's patients across demographic and pain-related variables.

7.3.2. Preliminary analyses

Consent to participate in research

Student *t*-test and Fisher's exact test comparisons revealed that there were no significant differences in age, duration of pain, or gender between patients who provided consent for their self-report measures to be used in research and those who did not consent (see Table 7.2). In addition, patients who did not provide consent did not differ significantly from those who did consent with respect to whether they were visiting the Centre regarding a compensation claim or legal case (Fisher's Exact Test, $p = 0.287$).

Table 7.1: Participants' demographic and pain-related characteristics compared with the normative sample (Nicholas et al., 2008)

Variable	Current Study N = 206	Nicholas et al. (2008) N = 5, 941
Age (years)	n = 206	n = 5, 941
M (SD)	53 (16)	48 (16.2)
Range	21 - 88	[+]
Gender	n = 206	n = 5, 941
Male	77 (37.4%)	2,528 (42.6%)
Female	129 (62.6%)	3,413 (57.4%)
Marital status	n = 206	n = 4, 508
Married/De facto	114 (55.3%)	2886 (64.0%)
Divorced/Separated	29 (14.1%)	544 (12.1%)
Single/Never Married	44 (21.4%)	800 (17.7%)
Widowed	19 (9.2%)	278 (6.2%)
Work status	n = 204	n = 4, 438
Full-time/Part-time work	51 (25%)	1348 (30.4%)
Home Duties	17 (8.3%)	462 (10.4%)
Unemployed due to pain	57 (27.9%)	1430 (32.2%)
Retired	59 (28.9%)	804 (18.1%)
Other	20 (9.9%)	394 (8.9%)
Is this visit related to:	n = 206	n = 4, 467
A Workers Compensation Claim	59 (28.6%)	1429 (32.0%)
A Third Party Accident Compensation Claim	11 (5.3%)	318 (7.1%)
Some other legal case	2 (1%)	78 (1.8%)
None of the above	134 (65.1%)	2642 (59.1%)
Pain duration (months)	n = 205	n = 5, 285
M (SD)	90.07 (112.57)	80.2 (111.2)
Range	3 - 568	6 - >300
Pain site	n = 206	n = 4, 932
Head, face and mouth	12 (5.8%)	364 (7.4%)
Cervical region	4 (1.9%)	146 (3.0%)
Upper shoulder and upper limbs	30 (14.6%)	566 (11.5%)
Thoracic region	9 (4.4%)	102 (2.1%)
Abdominal	2 (1.0%)	92 (1.9%)
Lower back, lumbar spine and sacrum	17 (8.3%)	641 (13.0%)
Lower limbs	13 (6.3%)	391 (7.9%)
Pelvic region	0 (0.0%)	53 (1.1%)
Anal, peri-anal and genital	2 (1.0%)	60 (1.2%)
Lower back and lower limbs	53 (25.7%)	701 (14.2%)
More than 2 major sites	64 (31.1%)	1816 (36.8%)

[+] Data not reported in Nicholas et al. (2008)

Table 7.2: Comparison of patients who consented and did not consent to participate in research

Variable	Provided consent	Did not consent	*t* (df) or Fisher's exact	*p*
Age (years)	n = 221	n = 15		
M (SD)	53.11 (16.02)	49.2 (15.75)	0.916 (234)	0.361
Pain duration (months)	n = 220	n = 13		
M (SD)	87.64 (110.22)	66.23 (48.91)	0.695 (231)	0.488
Gender	n = 221	n = 16		
Male	84 (38.0%)	8 (50.0%)	8	0.244
Female	137 (62.0%)	8 (50.0%)		
Visit related to claim or legal case?	n = 205	n = 13		
Yes	72 (35.1%)	3 (23.1%)	3.0	0.287
No	133 (64.9%)	10 (76.9%)		

Missing values analyses

Details of the comparisons between completers and non-completers are provided in Appendix F. For the demographic variables, there was a significant difference in age between completers (M = 52.27 years, SD = 15.64) and non-completers [M = 67.42 years, SD = 14.94; $t(204) = 3.263$, $p = 0.001$]. There were no significant differences between completers and non-completers on any other demographic or pain-related variables. A significant difference between completers and non-completers was found on depression [M = 14.35, SD = 12.61 vs M = 2, SD = 2.31; $t(8) = -8.332$, $p < 0.001$]. However, the non-completers group for this analysis only contained four participants so this result was not considered valid, and it was concluded that age was the only notable difference between the two groups. This was consistent with the missing data analysis conducted for Study 1. Consequently, the same decision was taken to delete 12 non-completers. This resulted in a final sample size of N=194 for the main analyses comparing different onset groups.

Once the non-completers were deleted, the type and pattern of missing values in the remaining data set were examined. The only variables with more than 5% of values missing were the three DASS scales (DASS-S = 5.7%, DASS-A = 6.7%, DASS-D = 5.2%). All other variables had very few missing values (typically <1%) and so a mean substitution method was employed to estimate the missing values on these variables (Tabachnik & Fidell, 2001).

To investigate the missing DASS values further, participants with incomplete DASS scales were compared with participants with complete DASS scales. The results of these comparisons are presented in Appendix F. In brief, when compared with participants who had completed the DASS-A, participants with missing values reported significantly higher levels of depression [M = 13.93, SD = 12.28 vs M = 39.67, SD = 3.21; $t(3.083) = -12.442$, $p = 0.001$]. However, as there were only three participants in the non-completers group, this was not considered a valid comparison. This was the only significant difference between DASS completers and non-completers; thus, the missing DASS values were considered to be indicative of a random pattern and mean substitution was used as the method of imputation (Hair et al., 2006).

Twenty-one patients (10.8%) did not complete the PCL. There were no significant differences between these patients and those who had completed the PCL on a range of demographic and clinical variables (see Appendix F for details). The only difference between the groups was age, with the completers being significantly younger (M = 51.47, SD = 15.43) than non-completers [M = 58.86, SD = 16.21; $t(192) = -2.06$, $p = .041$]. The comparison of PCL completion rates between the Accident, Specific Incident, and Insidious/Spontaneous groups revealed no significant differences, indicating that patients with an insidious/spontaneous onset of pain were just as able to complete the modified PCL as the other onset groups ($\chi^2 = .552$, $p = .759$).

7.3.3. Main Analyses

Accident onset comparisons

This set of analyses compared the Accident, Specific Incident, and Insidious/Spontaneous groups. Information about the onset of pain for each group is presented in Table 7.3. The majority of participants in the Accident group attributed their pain to a work-related accident (49 participants or 56.3% of the Accident group). In the Specific Incident group, the onset event was almost equally distributed between surgery and illness.

Table 7.3: Onset of pain data for the Accident onset comparisons

Variable	Accident	Specific incident	Insidious / Spontaneous
How did your pain begin?	n = 87	n = 20	n = 87
Accident at work	49 (56.3%)	-	-
At work, not involving an accident	-	-	11 (12.6%)
Accident at home	5 (5.7%)	-	-
Motor vehicle accident	22 (25.3%)	-	-
After surgery	-	9 (45.0%)	-
After illness	-	11 (55.0%)	-
Pain just began, no clear reason	-	-	67 (77.0%)
Other	11 (12.6%)	0 (0.0%)	9 (10.3%)

Data for other demographic and pain-related variables for each group are presented in Table 7.4. Between-groups comparisons revealed that the groups differed significantly in terms of age [$F(2, 191) = 26.662$, $p < 0.001$] and pain duration [$F(2, 179) = 18.705$, $p < 0.001$]. For age, post-hoc analyses revealed that participants in the Accident onset group were significantly younger than the participants in the other two groups. There was no age difference between participants in the Specific Incident and Insidious/Spontaneous groups. In the case of pain duration, participants in the Insidious/Spontaneous group had

experienced pain for longer than the participants in the other two groups. There was no difference in pain duration between the Accident and Specific Incident groups. The chi-square analysis indicated that compared with the other two groups, significantly more participants in the Accident group were involved in a compensation claim or legal case [$\chi^2(2) = 81.818$, $p < 0.001$]. As noted earlier, given these differences and similar findings in previous studies, age, pain duration, and compensation status were included as covariates in the main analyses.

Table 7.4: Demographic and pain-related variables by group (Accident onset comparisons)

Variable	Accident	Specific incident	Insidious / Spontaneous	*F*-ratio or X^2	*p*
Age (years)	n = 87	n = 20	n = 87	26.662	<.001
M (SD) [a]	44.23 (10.97)	60.8 (16.81)	58.36 (15.69)		
Pain duration (mths)	n = 82	n = 19	n = 81	18.705	<.001
M (SD) [b]	43.61 (40.43)	32.79 (30.38)	103.62 (92.71)		
Attendance related to claim or legal case? [a]	n = 87	n = 20	n = 87		
Yes	62 (71.3%)	1 (5.0%)	8 (9.2%)	81.818	<.001
No	25 (28.7%)	19 (95.0%)	79 (90.8%)		

[a] Accident differs from Specific Incident and Insidious/Spontaneous
[b] Accident and Specific Incident differ from Insidious/Spontaneous

Tables 7.5 and 7.6 provide details of each of the GLMs investigating group differences on the dependent variables, controlling for the effects of compensation status, age, and pain duration. For pain severity, a sensitivity analysis was conducted by removing one case with an out of range studentized residual. This did not change the overall model so all cases were retained and this is the model reported in Table 7.5 (details of the alternative analysis are provided in Appendix F).

Type of pain onset was not a significant predictor of pain severity ($F = .256$, $p = .774$), with no significant differences in pain severity across the onset groups after adjusting for the effects of the covariates. Age and pain duration were also not significant

predictors of pain severity (p = .887 and p = .405, respectively). Compensation status was a significant predictor in the model, with participants involved in a compensation claim or legal case reporting higher levels of pain severity compared with those not involved [M = 4.519, CI = 4.145 – 4.893 vs M = 3.863, CI = 3.628 – 4.099; F = 8.453, p = .004].

Type of pain onset was not a significant predictor of pain-related disability (F = .492, p = .612). Age and compensation status were also not significant predictors (p = .068 and p = .060, respectively). However, pain duration was a significant predictor in the model, with longer pain duration associated with higher levels of pain-related disability (β = .010, p = .021). The variables included in these models accounted for only a small proportion of the variance in pain severity and pain-related disability.

Table 7.5: Accident group comparison for pain severity and pain-related disability (variables not transformed)

	Accident	**Specific Incident**	**Insidious / Spontaneous**	**Total**	***F***	***p***
MPI-PS	n = 87	n = 20	n = 87	N = 194	.256	.774[a]
Raw mean	4.39	3.87	3.88	4.12		
Adjusted Mean	4.28	4.18	4.12	4.19		
(95% CI)*	(4.01-4.54)	(3.64-4.72)	(3.82-4.41)	(3.97-4.41)		
RMDQ	n = 86	n = 20	n = 87	N = 193	.492	.612[b]
Raw mean	12.80	11.25	11.75	12.17		
Adjusted Mean	13.07	12.14	11.95	12.38		
(95% CI)*	(11.73-14.40)	(9.41-14.87)	(10.45-13.44)	(11.27-13.5)		

*Adjusted means are estimated marginal means from the GLM controlling for age, pain duration, and compensation status.

[a]R^2 = .092 (Adjusted R^2 = .068). Age and pain duration = ns. Comp status = sig (p = .004).

[b]R^2 = .060 (Adjusted R^2 = .035). Compensation status and age = ns. Pain duration = sig (p = .021).

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire.

The distribution of the DASS Depression scale residuals did not meet the necessary assumptions after transformation. Consequently, the non-parametric tests were employed for this variable. The DASS Anxiety and Stress scales were square-root transformed. For

the anxiety variable, a sensitivity analysis was conducted by removing one case with an out of range studentized residual. Although this did not change the overall model, the result was a better fit to the assumptions of the GLM and this is the model reported here (details of the alternative analysis are provided in Appendix F). Type of onset of pain was not a significant predictor in both DASS models, with comparable levels of anxiety and stress reported across onset groups after adjustment for the covariates (see Table 7.6 for details). Similarly, none of the covariates were significant predictors in the models. The greatest proportion of variance in symptomatology was explained in the stress model (R^2 = .135, adjusted R^2 = .112). For depression, there was no significant difference between the groups according to the median test (χ^2 = 4.777, p = .092). The Kruskal-Wallis test approached significance (χ^2 = 5.809, p = .055; see Table 7.7).

Table 7.6: Accident group comparison for anxiety and stress symptoms (transformed variables)

	Accident	**Specific Incident**	**Insidious/ Spontaneous**	**Total**	***F***	***p***
DASS-A	n = 87	n = 20	n = 86	N = 193	.842	.432[a]
Raw mean	11.92	7.36	8.46	9.9		
Adjusted Mean	9.52	7.54	7.42	9.14		
(95% CI)*	(7.58-11.67)	(4.19-11.72)	(5.49-9.60)	(6.61-9.80)		
DASS-S	n = 87	n = 20	n = 87	N = 194	1.69	.187[b]
Raw mean	19.84	12.38	12.47	15.77		
Adjusted Mean	16.39	13.26	12.47	13.99		
(95% CI)*	(13.81-19.16)	(8.74-18.63)	(9.96-16.23)	(12.0-16.14)		

*Adjusted means and 95% CI are reverse-transformed to original scale. Adjusted means are estimated marginal means from the GLM controlling for age, pain duration, and compensation status.

[a]R^2 = .065 (Adjusted R^2 = .040). Age, pain duration and compensation status = ns.

[b]R^2 = .135 (Adjusted R^2 = .112). Age, pain duration and compensation status = ns.

Note: DASS-A = Depression Anxiety Stress Scales – Anxiety; DASS-S = Depression Anxiety Stress Scales – Stress.

Table 7.7: Accident group comparison for depression symptoms (non-parametric tests)

DASS - D		Accident (n = 87)	Specific Incident (n = 20)	Insidious / Spontaneous (n = 87)	Total (N=194)	χ^2	*p*
Mean (SD)		16.98 (13.33)	11.84 (11.7)	12.42 (11.53)	14.4 (12.54)		
Median (min-max)		14 (0-42)	8 (0-36)	9 (0-41)	10.5 (0-42)		
Median Test	>Median	51	8	38		4.777	.092
	<=Median	36	12	49			
Kruskal-Wallis	Mean Rank	108.17	85.22	89.65		5.809	.055

Note: DASS-D = Depression Anxiety Stress Scales – Depression.

Traumatic onset comparisons

This group of analyses compared the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. As described in the Method section, allocation to the Traumatic and Non-traumatic groups was based on independent evaluations of the potentially traumatic nature of the onset of pain. Inter-rater reliability between the two experts was excellent (Kappa value = 0.958, $p < 0.001$). The raters disagreed on only two events, so these two cases were deleted from the analyses. One of these participants had attributed the onset of her pain to an accident at home in which a pole and a heavy stone fell on her back from a car port. The other participant had reported being involved in a motor-vehicle accident in which her car was hit from the rear by another vehicle. Another two cases were excluded because their clinic file was missing during the period of data collection. This resulted in a sample size of 190 for the traumatic onset comparisons.

Table 7.8 indicates that the raters judged the majority of work-related accidents to be non-traumatic and all the motor-vehicle accidents as traumatic or potentially traumatic. Accidents that occurred at home were equally distributed between the Traumatic and

Non-traumatic groups. Pain that was associated with surgery or illness was classified as non-traumatic.

Table 7.8: Onset of pain data for the traumatic onset comparisons

Variable	Traumatic onset	Non-traumatic onset	Insidious / Spontaneous
How did your pain begin?	n = 34	n = 69	n = 87
Accident at work	7 (20.6%)	41 (59.4%)	-
At work, not involving an accident	-	-	11 (12.6%)
Accident at home	2 (5.9%)	2 (2.9%)	-
Motor vehicle accident	21 (61.8%)	0 (0%)	-
After surgery	0 (0%)	9 (13.0%)	-
After illness	0 (0%)	10 (14.5%)	-
Pain just began, no clear reason	-	-	67 (77.0%)
Other	4 (11.8%)	7 (10.1%)	9 (10.3%)

Group comparisons revealed significant differences between the three groups in age ($p < 0.001$) and pain duration ($p < 0.001$). For age, participants in the Traumatic and Non-traumatic onset group were significantly younger than the participants who reported a spontaneous or insidious onset of pain (see Table 7.9 for details). There was no age difference between participants reporting a Traumatic and Non-traumatic onset of pain. Similarly, in the case of pain duration, participants in the Insidious/Spontaneous group had experienced pain for longer than the participants in the other two groups. There was no difference in pain duration between the Traumatic and Non-traumatic onset groups. Chi-square analysis indicated that compared with the Insidious/Spontaneous onset group, significantly more participants in the Traumatic and Non-traumatic onset groups were involved in a compensation claim or legal case [$\chi^2(2) = 52.771$, $p < 0.001$]. Consequently, these demographic variables were included as covariates in the traumatic onset group comparisons.

Table 7.9: Demographic and pain-related variables by group (Traumatic onset comparisons)

Variable	Traumatic	Non--traumatic	Insidious / Spontaneous	*F*-ratio or X^2	*p*
Age (years)	n = 34	n = 69	n = 87	17.331	< .001
M (SD) [a]	42.24 (12.64)	49.36 (13.59)	58.36 (15.69)		
Pain Duration (mths)	n = 34	n = 69	n = 87	10.721	< .001
M (SD) [a]	66.68 (96.33)	52.0 (67.53)	125.33 (125.56)		
Attendance related to claim or legal case? [a]	n = 34	n = 69	n = 87		
Yes	21 (61.8%)	41 (59.4%)	8 (9.2%)	52.771	< .001
No	13 (38.2%)	28 (40.6%)	79 (90.8%)		

[a] Traumatic and Non-traumatic differ from Insidious/Spontaneous

As was the case with the accident onset comparisons, the MPI-PS and RMDQ did not require transformation in the traumatic onset comparisons. A sensitivity analysis was conducted for the pain severity model to investigate the impact of three cases with out of range studentized residuals. Deletion of two of these cases improved the fit of the distribution to the assumptions of the GLM and this is the model reported here (see Appendix F for details).

As presented in Table 7.10, type of pain onset was not a significant predictor in either the pain severity or disability models after adjustment for the effects of the covariates, with the three groups reporting comparable levels of pain severity and pain-related disability. None of the covariates were significant predictors in the pain severity model, but compensation status and pain duration were both significant in the disability model. In particular, longer pain duration was associated with higher levels of disability (β =.010, p= .017). Compared with participants who were not involved in a compensation claim or legal case, those who were involved in such a claim reported higher levels of disability (compensation: M = 13.88, CI = 12.36 – 15.39; no compensation: M = 11.38,

CI = 10.15 – 12.6; F = 5.564, p = .019). Only a small proportion of the variance was explained in both models.

Table 7.10: Traumatic group comparison for pain severity and pain-related disability (variables not transformed)

	Traumatic	Non-traumatic	Insidious/ Spontaneous	Total	*F*	*p*
MPI-PS	n = 33	n = 69	n = 86	N = 188	.886	.414[a]
Raw mean	4.5	4.27	3.92	4.15		
Adjusted Mean	4.46	4.23	4.13	4.28		
(95% CI)*	(4.08-4.84)	(3.98-4.49)	(3.86-4.40)	(4.10-4.45)		
RMDQ	n = 34	n = 68	n = 87	N = 189	.303	.739[b]
Raw mean	12.53	12.56	11.75	12.18		
Adjusted Mean	12.94	12.84	12.10	12.63		
(95% CI)*	(10.95-14.92)	(11.46-14.23)	(10.66-13.54)	(11.73-13.53)		

*Adjusted means are estimated marginal means from the GLM controlling for age, pain duration, and compensation status.

[a]R^2 = .090 (Adjusted R^2 = .065). Age and pain duration = ns. Comp status = sig (p = .003).

[b]R^2 = .060 (Adjusted R^2 = .034). Age = ns. Pain duration = sig (p = .017); Comp status = sig (p = .019).

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire.

Distributions of the DASS Depression and Anxiety scale residuals did not meet the necessary assumptions after transformation. Consequently, non-parametric tests were conducted. As presented in Table 7.11, there were no significant differences between the three onset groups on both DASS scales. Similarly, type of pain onset was not a significant predictor of stress symptoms after adjustment for the effects of the covariates (see Table 7.12 for details of the DASS-S model, square-root transformation). Age and pain duration were also not significant predictors in the stress model. Compensation status was found to be a significant predictor of stress symptoms, with participants involved in a compensation claim reporting higher levels of stress symptoms than those not involved in such a claim (M = 17.5, CI = 14.5 – 20.76 vs M = 12.15, CI = 10.09 – 14.38; F = 7.011, p = .009).

Table 7.11: Traumatic group comparison for depression and anxiety symptoms (non-parametric)

		Traumatic (n = 34)	Non-traumatic (n = 69)	Insidious / Spontaneous (n = 87)	Total (N=190)	χ^2	*p*
			DASS-D				
Mean		15.67	16.4	12.42	14.4		
(SD)		(12.16)	(13.97)	(11.53)	(12.54)		
Median		13	12	9	10.5		
(min-max)		(0-40)	(0-42)	(0-41)	(0-42)		
Median	>Median	20	36	38		2.559	.278
Test	<=Median	14	33	49			
Kruskal-Wallis	Mean Rank	103.09	101.26	87.97		3.042	.218
			DASS-A				
Mean		10.81	11.37	8.46	9.9		
(SD)		(9.15)	(10.4)	(8.37)	(9.27)		
Median		7.85	8	6	7		
(min-max)		(0-36)	(0-42)	(0-38)	(0-42)		
Median	>Median	18	35	38		1.188	.552
Test	<=Median	6	34	49			
Kruskal-Wallis	Mean Rank	102.38	102.41	87.33		3.548	.170

Note: DASS-D = Depression Anxiety Stress Scales – Depression; DASS-A = Depression Anxiety Stress Scales – Anxiety.

Table 7.12: Traumatic group comparison for stress symptoms (transformed)

	Traumatic n = 34	Non-traumatic n = 69	Insidious / Spontaneous n = 87	Total N = 190	*F*	*p*
DASS-S					.830	.438[a]
Raw mean	19.43	18.0	12.47	15.72		
Adjusted Mean (95% CI)*	15.75 (12.07-19.89)	15.36 (12.79-18.15)	13.09 (10.60-15.82)	14.71 (13.04-16.47)		

*Adjusted means and 95% CI are reverse-transformed to original scale. Adjusted means are estimated marginal means from the GLM controlling for age, pain duration, and compensation status.

[a]R^2 = .137 (Adjusted R^2 = .114). Age and pain duration = ns. Comp status = sig (*p* = .009).

Note: DASS-S = Depression Anxiety Stress Scales – Stress.

PCL validity analyses

Descriptive statistics for the PCL across the sample are provided in Table 7.13. These scores are virtually identical to the PCL scores obtained in the first study. The PCL exhibited excellent internal consistency, with Cronbach's alpha coefficients similar to those obtained in Study 1 (PCL total = 0.915; Reexperiencing = 0.873; Avoidance = 0.810; and Arousal = 0.82).

Table 7.13: Descriptive statistics for the PCL scales

PCL scale	Range	Mean (SD)	95%CI
Total Score (n = 173)	17 – 79	37.24 (13.86)	35.16 – 39.32
Reexperiencing (n = 172)	5 – 25	8.84 (4.72)	8.13 – 9.55
Avoidance (n = 172)	7 – 35	15.26 (6.08)	14.35 – 16.18
Arousal (n = 173)	5 – 25	13.20 (4.99)	12.45 – 13.95

The PCL Total score was strongly positively correlated with all three DASS scales (DASS – D: $\rho = 0.712$, $p < 0.001$; DASS – A: $\rho = 0.670$, $p < 0.001$; DASS – S: $\rho = 0.759$, $p < 0.001$). As would be expected, the relationship between the PCL Total score and the RMDQ was moderate, but not as strong as the association with the DASS scales ($\rho = 0.412$, $p < 0.001$).

For the comparison of levels of PTSD symptoms endorsed by the Traumatic, Non-traumatic, and Insidious/Spontaneous groups the PCL Total scale and the Avoidance and Arousal subscales were subjected to log transformation and these models are reported here. The Reexperiencing subscale was not able to be transformed adequately and non-parametric tests were conducted.

As presented in Tables 7.14 and 7.15, type of onset was not a significant predictor of the PCL Total score or scores on the Avoidance and Arousal subscales after

adjustment for the covariates in the model. Age was a significant predictor of the PCL Total score (β = -.005; F = 6.590, p = .011) and the PCL Avoidance subscale (β = -.006; F = 6.995, p = .009), with younger age associated with higher PCL scores. Compensation status was a significant predictor of the PCL Arousal subscale, with participants involved in a claim reporting higher levels of symptoms (M = 14.04, CI = 12.69 – 15.55), compared with those not involved in a claim (M = 11.61, CI = 10.7 – 12.62; F = 7.282, p = .008). In contrast to the other analyses, the nonparametric tests comparing the groups' responses on the PCL Reexperiencing subscale revealed significant differences between the groups (see Table 7.15 for details). However, it is important to note that these tests do not control for the effects of the covariates.

Table 7.14: Traumatic group comparison for PCL scales (transformed variables)

	Traumatic	**Non-traumatic**	**Insidious / Spontaneous**	**Total**	***F***	***p***
PCL Total	n = 32	n = 63	n = 76	N = 171	1.162	.316[a]
Raw mean	43.31	39.29	32.92	37.21		
Adjusted Mean	38.02	36.05	33.68	35.87		
(95% CI)*	(33.62-42.95)	(33.08-39.29)	(30.75-36.89)	(33.92-37.94)		
PCL Avoid	n = 32	n = 63	n = 75	N = 170	.412	.663[b]
Raw mean	17.44	15.75	13.95	15.27		
Adjusted Mean	15.06	13.97	14.18	14.40		
(95% CI)*	(13.11-17.31)	(12.68-15.39)	(12.79-15.72)	(13.52-15.33)		
PCL Arou	n = 32	n = 63	n = 78	N = 173	1.235	.293[c]
Raw mean	15.16	14.24	11.6	13.22		
Adjusted Mean	13.38	13.11	11.88	12.77		
(95% CI)*	(11.72-15.30)	(11.94-14.38)	(10.77-13.11)	(12.03-13.57)		

*Adjusted means and 95% CI are reverse-transformed to original scale. Adjusted means are estimated marginal means from the GLM controlling for age, pain duration, and compensation status.

[a]R^2 = .155 (Adjusted R^2 = .129). Pain duration and compensation status = ns. Age = sig (p = .011).

[b]R^2 = .105 (Adjusted R^2 = .078). Pain duration and compensation status = ns. Age = sig (p = .009).

[c]R^2 = .162 (Adjusted R^2 = .137). Age and pain duration = ns. Compensation status = sig (p = .008).

Note: PCL Total = PTSD Checklist Total score; PCL Avoid = PTSD Checklist Avoidance subscale; PCL Arou = PTSD Checklist Arousal subscale.

Table 7.15: Traumatic group comparison for the PCL Reexperiencing subscale (non-parametric tests)

PCL Reexp		Traumatic (n = 30)	Non-traumatic (n = 64)	Insidious / Spontaneous (n = 76)	Total (N=170)	χ^2	*p*
Mean		10.63	9.47	7.54	8.84		
(SD)		(5.75)	(4.33)	(4.29)	(4.72)		
Median		10	8	6	7		
(min-max)		(5-25)	(5-21)	(5-24)	(5-25)		
Median Test	>Median	17	35	21		13.181	**.001**
	<=Median	13	29	55			
Kruskal-Wallis	Mean Rank	102.2	98.15	68.26		17.635	**.000**

Note: PCL Reexp = PTSD Checklist Reexperiencing subscale.

Application of a diagnostic algorithm and cut-off scores

Weathers et al.'s (1993) original cut-off score of 50 on the PCL indicated that 36 participants (20.8% of the 173 participants who completed the PCL) could be classified as potentially meeting criteria for a diagnosis of PTSD. Sherman et al.'s (2005) cut-off score of 41 placed 50 participants (28.9%) in this group. According to the diagnostic algorithm suggested by Weathers et al. 54 patients (31.2%) could be classified as meeting diagnostic criteria for PTSD. The outcome of applying the diagnostic algorithm was more consistent with the cut-off score of 41 than the cut-off score of 50.

However, information obtained from the participants' medical files indicated that only 12 participants (7%) had notes in their medical file indicating that they were experiencing post-traumatic stress symptoms. Only one of these patients was actually diagnosed with PTSD at the time of their assessment at the Centre. Table 7.17 provides the PCL Total and subscale scores for each of these 12 participants. Participant "3" in the table was the only one who was given a PTSD diagnosis. All but three were classified as "High PCL" according to a median split of PCL Total scores. All but one participant had

been placed in the Traumatic onset group by the independent experts. This participant was a 42-year old female who had presented with right foot and coccyx pain of 14 months duration. According to the medical notes, she had been injured at work when a trolley ran over her right foot. However, inspection of the file notes indicated that she was experiencing panic attacks and flashbacks in relation to a home invasion which had occurred two years prior to the assessment at the Centre. That is, although she had experienced a non-traumatic onset of pain (and was therefore allocated to the Non-traumatic group), she had endorsed symptoms on the PCL in relation to a prior traumatic event.

Table 7.16: PCL scores and type of onset of pain of participants noted as reporting PTSD symptoms in their medical file

Participant	PCL Re-experiencing	PCL Avoidance	PCL Arousal	PCL Total	PCL median split*	Onset group
1	17	25	22	64	High	Traumatic
2	6	10	10	26	Low	Traumatic
3	17	20	22	59	High	Traumatic
4	20	23	16	59	High	Traumatic
5	9	11	13	33	Low	Traumatic
6	13	17	10	40	High	Traumatic
7	5	10	13	28	Low	Traumatic
8	7	14	19	40	High	Traumatic
9	24	30	25	79	High	Traumatic
10	10	23	18	51	High	Traumatic
11	13	20	12	45	High	Non-traumatic
12	12	16	11	39	High	Traumatic

* PCL Total score median = 33. High PCL group = scores > 33, low PCL group = scores ≤ 33.

Consistent with the above discrepancies between the results of the diagnostic algorithm and clinical data, a comparison of participants who endorsed sufficient symptoms on the PCL to meet diagnosis according to the algorithm with the Traumatic onset group categories indicated that 17% of the Insidious/Spontaneous group would have been identified as potential PTSD cases despite the fact that their pain had not developed

in the context of a specific event. At the same time, there was no difference between the Traumatic and Non-traumatic groups in the proportion of participants that would have been identified as potential PTSD cases. However, there was a significant difference between these two groups and the Insidious/Spontaneous group.

Table 7.17: The results of the diagnostic algorithm across traumatic onset groups

Variable	Traumatic n = 32	Non-traumatic n = 63	Insidious / Spontaneous n = 76	χ^2	*p*
Symptoms met criteria for PTSD according to diagnostic algorithm	15 (46.88%)	25 (39.68%)	13 (17.11%)	12.852	.002
Symptoms did not meet criteria for PTSD according to diagnostic algorithm	17 (53.13%	38 (60.32%)	63 (82.89%)		

Additional analyses

Given the lack of significant differences between the onset groups in the main analyses, additional analyses were conducted to assist in explaining this result. Firstly, in order to ascertain whether the above results were due to the way in which participants were grouped, the participants were re-grouped so that the comparison groups corresponded to those applied by Turk et al. (1996). This resulted in two groups: (1) Accident: pain associated with all accidents; and (2) Insidious/Spontaneous: pain that was not associated with any identifiable event. Following Turk et al., participants who attributed their pain to illness or surgery were excluded from these analyses. Comparisons were only conducted for pain severity, pain-related disability, and depression.

The results of these analyses are not described in detail here because they were the same as those obtained in the Accident onset group comparisons described above (the results are, however, included in Appendix F). That is, type of pain onset was not a

significant predictor of pain severity or pain-related disability after adjustment for age, pain duration, and compensation status. Non-parametric tests for the DASS Depression scale revealed significant differences between the two onset groups; however, as already noted, this result should be interpreted with caution because the covariates are not accounted for in these statistical tests.

Secondly, the participants were re-grouped a second time so that the comparison groups corresponded to those applied by Turk and Okifuji (1996). This resulted in two groups: (1) Specific Incident: pain associated with all incidents; that is, accidents, surgery, illness; and (2) Insidious/Spontaneous: pain that was not associated with any identifiable event. Following Turk and Okifuji, only participants who were not involved in a compensation claim or legal case were included in the analyses.

Again, type of pain onset was not a significant predictor of pain severity or pain-related disability after adjustment for age and pain duration (see Appendix F). Age and pain duration were both significant predictors of pain-related disability, with older age and longer pain duration associated with higher levels of disability. Non-parametric tests for the DASS Depression scale revealed no significant differences between the groups.

Finally, to identify variables that may have been better predictors of pain-severity and pain-related adjustment than onset of pain in the current sample, three cognitive variables were examined using the same statistical approach as the main analyses. These measures were mentioned in Study 1 as part of the Centre's standard battery, but were not fully described. A complete description of these cognitive measures is provided in Chapter 8. They were utilised in the present study as part of a post-hoc analysis because it was thought they might shed further light on the analysis. The three cognitive variables were self-efficacy (Pain Self Efficacy Questionnaire, PSEQ; Nicholas, 1989), fear-avoidance beliefs (Tampa Scale for Kinesiophobia, TSK; Kori et al., 1990), and

catastrophising (Pain Related Self-Statements, PRSS; Flor & Turk, 1988). Type of onset was not included in these models; however, age, pain duration, and compensation status were included as covariates.

The results of these analyses are presented in Table 7.18 and are consistent with previous research (as reviewed in Chapter 1). Self-efficacy, catastrophising, and fear-avoidance beliefs were all significant predictors of pain severity. Lower levels of self-efficacy, and higher levels of catastrophising and fear-avoidance beliefs were associated with higher levels of pain severity. Involvement in a compensation case was also a significant predictor of pain severity (compensation: M = 4.54, SD = 0.84, no compensation: M = 3.84, SD = 1.23; F = 10.102, $p = 0.002$). Self-efficacy, fear-avoidance beliefs, and age were significant predictors of pain-related disability. Older age, lower levels of self-efficacy, and higher levels of fear-avoidance beliefs were associated with higher levels of pain-related disability. Self-efficacy, catastrophising, and pain duration were predictors of depression. Longer pain duration, lower self-efficacy, and higher levels of catastrophising were associated with higher levels of depression. The relationship between depression and catastrophising was particularly strong ($\beta = 5.081$). Inclusion of the cognitive variables in the models increased the proportion of explained variance to at least 35%.

Table 7.18: Results of GLMs including cognitive variables

	PSEQ	TSK	PRSS - Cat	Age	Pain duration
MPI-PS[a]	β = -.027	β = .02	β = .198	β = .005	β = .000
(N = 190)	F = 18.45	F = 5.59	F = 7.07	F = .867	F = .054
	p = **.000***	*p* = **.019**	*p* = **.009**	*p* = .353	*p* = .817
RMDQ[b]	β = -.157	β = .183	β = -.007	β = .058	β = .006
(N = 189)	F = 23.85	F = 19.05	F = .000	F = 5.506	F = 3.625
	p = **.000**	*p* = **.000**	*p* = .985	*p* = **.02**	*p* = .058
DASS-D[c]	β = -.272	β = .012	β = 5.081	β = -.004	β = -.014
(N = 190)	F = 17.582	F = .021	F = 44.753	F = .008	F = 4.204
	p = **.000**	*p* = .886	*p* = **.000**	*p* = .929	*p* = **.042**

[a]R^2 = .375 (Adjusted R^2 = .354). Comp status = sig (*p* = .002).

[b]R^2 = .358 (Adjusted R^2 = .337). Compensation status = ns.

[c]R^2 = .461 (Adjusted R^2 = .443). Compensation status = ns.

* *p*-values in bold are significant $p < .05$.

Note: PSEQ = Pain Self-Efficacy Questionnaire; TSK = Tampa Scale for Kinesiophobia; PRSS – Cat = Pain Related Self-Statements – Catastrophising scale; MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire.

7.4. Discussion

The aims of this study were to investigate questions pertaining to the impact of type of onset of pain on pain-related adjustment, to further evaluate the psychometric properties of the PCL, and to examine the diagnostic utility of the modified PCL in a chronic pain clinic setting. The key objectives of the study were: (1) to determine if patients with accident-related pain report higher levels of pain severity and exhibit poor adjustment to chronic pain in comparison to patients who have experienced a spontaneous or insidious onset of pain, or the onset of pain following other incidents; (2) to determine if patients who have experienced a potentially traumatic onset of pain report higher levels of pain severity and exhibit poor adjustment to chronic pain in comparison to patients who have experienced non-traumatic and spontaneous or insidious onset of pain; (3) to

evaluate the construct validity of the PCL in a chronic pain clinic setting; and (4) to examine the diagnostic utility of the PCL in this setting. These objectives will be discussed in the following sections.

7.4.1. Impact of type of onset of pain

In summary, the analyses comparing Accident, Specific Incident, and Insidious/ Spontaneous onset groups and Traumatic, Non-traumatic, and Insidious/Spontaneous onset groups revealed no significant differences between the groups on measures of pain severity, pain-related disability, and symptoms of affective distress after adjustment for age, pain duration, and compensation status. However, participants involved in a compensation claim or legal case reported significantly higher levels of pain severity in the Accident onset comparisons, and significantly higher levels of pain-related disability and symptoms of stress in the Traumatic onset comparisons, compared with participants who were not involved in a claim. Longer pain duration was associated with higher levels of pain-related disability in both group comparisons. Age was not a significant predictor of any of the outcome variables in either set of analyses. Overall, the combination of the predictor variable and the covariates did not account for a sizeable proportion of the variance in pain severity, pain-related disability, or affective distress.

Consistent with previous research (e.g. Geisser, Roth, Bachman & Eckert, 1996; Turk et al., 1996), there were significant differences between the onset groups on age, pain duration, and compensation status. As expected, participants who had experienced the onset of pain following an accident or injury (whether or not it was experienced as traumatic) were younger than participants who had experienced a spontaneous or insidious onset of pain, or pain associated with illness and surgery. Compared with the latter groups, patients presenting with accident- and injury-related pain were also more

likely to be involved in a compensation claim or legal case. On the other hand, patients who had experienced a spontaneous or insidious onset of pain had experienced pain for longer than patients in the other groups. As Geisser at al. (1996) noted, these group differences can probably be accounted for by the fact that older people tend to develop chronic pain conditions that are typically not associated with accidents or injuries (e.g. arthritis), and that are, therefore, not compensable. Related to this, the group differences in pain duration may be due to compensation claimants being referred for assessment earlier in the course of their condition than might otherwise be the case (Nicholas, 2005).

The lack of significant differences between the Accident, Specific Incident and Insidious/Spontaneous groups on measures of pain severity and pain-related adjustment is not consistent with existing research. As discussed in Chapter 2, previous studies have reported differences in these outcomes between patients with pain related to accidents or other specific incidents, and patients with pain that is unrelated to a specific precipitating event (e.g. Greenfield, Fitzcharles & Esdaile, 1992; Himmelstein, Feuerstein, Stanek, Koyamatsu, Pransky, Morgan & Anderson, 1995; Geisser et al., 1996; Turk & Okifuji, 1996; Turk et al., 1996; Nicholas, 2005). Although there has been some variation across studies in the exact onset categories which have been compared (e.g. accident-related pain versus pain related to other events), the current study failed to detect differences between groups even when participants had been categorised in exactly the same manner as two other studies (i.e. Turk & Okifuji, 1996; Turk et al., 1996).

Inspection of the raw means in both the Accident onset and Traumatic onset group comparisons indicated that before adjustment for the effects of the covariates, the Accident and Traumatic onset groups generally did report higher pain severity and poor adjustment in comparison to the other groups, but these differences were typically small and/or disappeared once the covariates were taken into account. It is possible that this was

due to inadequate statistical power, and that a larger sample (particularly larger numbers in the Specific Incident group) would have revealed significant differences. Consistent with this, Nicholas (2005) found differences between patients with pain related to work and motor- vehicle accidents and those with pain of a spontaneous or insidious nature in a significantly larger sample derived from the same centre as the current study (N > 2,800). However, it is important to note that covariates were not included in these analyses despite significant differences in disability and distress between compensation and non-compensation MVA victims in the sample. It is not clear if the differences between onset groups would have remained had compensation status been taken into account. On the other hand, the studies conducted by Turk and colleagues (Turk & Okifuji, 1996; Turk et al., 1996) controlled for the same covariates and detected differences between onset groups with smaller sample sizes to the current study (i.e. N = 92 in Turk et al., 1996 and N = 63 in Turk & Okifuji, 1996).

Given that approximately half of the studies which have reported differences between onset groups have been conducted with specific groups of chronic pain patients, it is possible that the importance of onset of pain varies across diagnostic groups. Three out of the eight studies reviewed in Chapter 2 employed samples of fibromyalgia patients consisting almost exclusively of females (Greenfield et al., 1992; Waylonis & Perkins, 1994; Turk et al., 1996). A fourth study was conducted in a clinic specialising in the treatment of work-related upper-extremity disorders (Himmelstein, Feuerstein, Stanek, Koyamatsu, Pransky, Morgan & Anderson, 1995). Consequently, it may not be appropriate to generalise the findings of these studies to the type of heterogeneous sample employed in the current study. Furthermore, as discussed in further detail below, although Turk and Okifuji's (1996) sample was heterogeneous in terms of diagnosis and site of

pain, it consisted only of participants who were not involved in a compensation claim. Again, this may limit the generalisability of their findings.

One of the aims of this study was to ascertain if there is a relationship between adjustment to pain and the potentially traumatic nature of the event associated with the onset of the pain. As already noted, the study did not find any significant differences between patients who were exposed to a potentially traumatic event, and those who were not. Incorrect categorisation of patients into the Traumatic and Non-traumatic onset groups could have contributed to the lack of differences observed in this set of comparisons. Allocation to the groups was based on information obtained from the participants' medical files. Lack of detail in the file about the event associated with the onset of pain, particularly about the patient's reaction to the event, could have prevented accurate coding of the events into the appropriate onset group. Although both raters were highly experienced in judging the traumatic nature of stressful events (both in clinical and research contexts) and the degree of agreement between them was very high, making a judgement about the event without information about the DSM-IV's Criterion A2 could have impacted upon the nature of the comparisons being made. Studies examining the role of Criterion A2 have revealed that it reduces estimates of trauma exposure (Breslau & Kessler, 2001; Schnurr, Spiro, Vielhauer, Findler & Hamblen, 2002). That is, rates of trauma exposure are lower if both Criterion A1 and A2 are required to satisfy the definition of a traumatic event. In this study, because raters were asked to judge the potential of an event to elicit PTSD when there was insufficient information about Criterion A2, a combination of events could have been rated as traumatic; that is, events that were actually experienced as traumatic by the patient and events that theoretically could have been, but that actually were not. This could have obscured group differences.

These issues could have been overcome by obtaining a more detailed assessment of the event (and potentially exposure to other traumatic events) through the administration of a validated assessment measure of exposure to traumatic events, such as the Potential Stressful Events Interview (Kilpatrick, Resnick & Freedy, 1991). However, a comprehensive and lengthy interview such as this is not feasible to administer to all patients who present to the Centre given that the multidisciplinary assessment process takes up a considerable part of the day. Given the typical degree of disability exhibited by the Centre's patients (Nicholas, 2005), those who were willing to participate in research that required them to remain at the Centre for a longer period may not have been representative of the Centre's patients overall. Consequently, brief self-report measures of exposure to trauma that could be included in a standard assessment battery (e.g. The Traumatic Life Events Questionnaire; Kubany, Leisen, Kaplan, Watson, Haynes, Owens & Burns, 2000) were also considered. However, these instruments are typically developed as a method of screening for exposure to a broad range of potentially traumatic events, rather than obtaining specific details about one event. A modified combination of the two assessment approaches (i.e. a questionnaire and brief follow-up interview when required), or incorporation of a brief evaluation of the event into standard assessment protocols could be considered for future studies of posttraumatic stress conducted in chronic pain clinic settings when structured interviews are not practical or appropriate.

The literature reviewed in Chapter 4 suggests that it is not the experience of a traumatic event per se that contributes to poor adjustment to chronic pain, but that it is the development of posttraumatic stress symptoms following the event that interacts with pain to lead to increased dysfunction. This hypothesis could have been investigated in the current study by dividing patients who had experienced a traumatic onset of pain into groups according to the degree of posttraumatic stress symptoms they endorsed on the

PCL. Geisser et al. (1996) conducted a similar analysis to this by comparing Accident/High PTSD, Accident/Low PTSD, and No Accident groups of chronic pain patients. However, because Geisser et al. did not collect information about the traumatic nature of the onset of the pain it is not possible to determine the proportion of patients in the Accident groups who had actually experienced a traumatic event, or whether the PTSD symptoms they reported were related to the onset of pain. The problem with this methodology can be demonstrated with data from the current study. A comparison of patients categorised according to Geisser et al.'s approach and the approach employed in the current study reveals that 23 patients who would have been placed in Geisser et al.'s Accident/High PTSD group had not experienced a traumatic onset of pain. Notwithstanding issues with the current study's methodology (as discussed in the previous paragraph), these figures still highlight the importance of assessing the traumatic nature of the onset of pain in studies of chronic pain and PTSD.

Similarly, the literature reviewed in Chapter 2 indicates that it may not be the experience of a particular type of event per se that contributes to poor adjustment, but that other variables associated with the type of onset are also significant. For example, studies of individuals who have experienced a range of stressful events have shown that attributing responsibility for the event to others is associated with poor adjustment (e.g. Delahanty et al., 1997; Lambert et al., 2004). This relationship has been reported in chronic pain and PTSD research (e.g. McParland et al., 2005; Schnyder et al., 2008). This potentially important influence on adjustment was not measured in the current study and may have contributed to the lack of differences observed between the onset groups.

Another variable associated with type of onset is involvement in a compensation claim or legal case. The results of this study repeatedly revealed compensation status to be an important variable, and comparison of the raw and adjusted means indicated that

compensation status typically accounted for any differences between the onset groups. This result is not consistent with previous investigations of the impact of type of onset of pain; however, most of the studies reviewed in Chapter 2 did not control for compensation status (e.g. Greenfield et al., 1992; Himmelstein et al., 1995).

There are a number of possible explanations for why the three studies that did control for compensation status still found differences between onset groups. Firstly, as already argued above, Turk et al.'s (1996) sample consisted entirely of fibromyalgia patients and was predominantly female. It is possible that the influence of compensation is different in this patient group to other groups of chronic pain patients.

Secondly, compared with the current study, the proportion of the accident group involved in compensation was smaller in the Turk et al. (1996) and Geisser et al. (1996) studies (over 70% in the current study cf. approximately 50% in the other studies). At the same time, a greater proportion of the spontaneous/insidious group was involved in compensation in the other studies (25% cf 9% in the current study). It is possible that these differences resulted in variations in the impact of compensation on the outcome variables. Furthermore, both Turk et al. and Geisser et al.'s compensation groups included patients who were receiving long-term disability payments, patients who had a disability claim pending, and patients who were involved in litigation to receive compensation or disability payments. As Turk and Okifuji (1996) have pointed out, there may be differences between patients who are already receiving financial compensation and those who are seeking it. These potential differences in participants within studies, and differences in the proportions of patients in these situations across studies, could also contribute to variations in the overall impact of compensation.

Finally, Turk and Okifuji excluded patients who were involved in compensation from their onset group comparisons; however, given that the majority of their sample was

involved in compensation the generalisability of their results to the sample as a whole could be questioned. Turk and Okifuji did not provide details of the demographic characteristics for the non-compensation group, so it is not possible to determine how representative this group was of the original sample. Similarly, given the strong association between accidents and compensation or litigation, patients with accident-related pain who are not involved in compensation or litigation may be very different to those who are involved. Again, this would limit the generalisability of Turk and Okifuji's findings. In the current study, the minority of patients with pain related to accidents or other events who were not involved in compensation were significantly older and reported longer duration of pain than those who were. Overall, the former group is not typical of the Centre's patients who present with accident-related pain.

These issues aside, there is growing evidence that compensation status is an important predictor of outcome. For example, two recent prospective studies of large cohorts of Australian patients with acute injuries (specifically acute low back pain and orthopaedic trauma patients) have revealed that compensation is associated with higher levels of pain and disability at 12 months post-injury, and is one of the strongest predictors of poor prognosis (Gabbe, Cameron, Williamson, Edwards, Graves & Richardson, 2007; Henschke, Maher, Refshauge, Herbert, Cumming, Bleasel, York, Das & McAuley, 2008). Likewise, meta-analysis of the surgical literature has revealed a strong association between compensation status and poor outcome after surgery (Harris et al., 2005). In studies of individuals with chronic pain, there is evidence that those involved in compensation or litigation report higher levels of pain, pain-related disability, and psychological symptoms, and are less likely to benefit from treatments or rehabilitation (Carron, DeGood & Tait, 1985; Rohling, Binder & Langhinrichsen-

Rohling, 1995; Turk & Okifuji, 1996; Rainville, Sobel, Hartigan & Wright, 1997; Blyth, March, Nicholas & Cousins, 2003).

The current finding that longer pain duration was associated with higher levels of pain-related disability has also been reported previously (e.g. Boersma & Linton, 2005; Dunn & Croft, 2006). Investigations of the roles of fear-avoidance beliefs and catastrophising in the development of chronic pain and disability at different stages of chronicity have revealed that pain duration may be an important moderator between these cognitive variables and disability (Sullivan, Sullivan & Adams, 2002; Boersma & Linton, 2005). Similarly, earlier studies reported that the relationship between coping strategies or beliefs about pain (e.g. the belief that pain equals harm) and adjustment varied as a function of pain duration (e.g. Jensen, Turner & Romano, 1992; Jensen, Turner, Romano & Lawler, 1994). That is, the relationships between pain and psychological variables appear to change as a function of duration of pain so that the importance of a particular variable in the development of pain-related dysfunction may vary depending on the stage being investigated (Jensen, Turner, Romano & Lawler, 1994; Sullivan, Sullivan & Adams, 2002; Boersma & Linton, 2005).

This perspective may have implications for the current context. The two recent studies cited above suggest that fear-avoidance beliefs and catastrophising become increasingly important the longer pain persists (Sullivan et al., 2002; Boersma & Linton, 2005). Thus, it is possible that type of onset of pain plays a greater role in influencing adjustment in the earlier stages post-injury, and that as chronicity increases other variables acquire greater significance in the development of disability and distress (Von Korff, Glasgow & Sharpe, 2002). Consistent with this and with the literature reviewed in Chapter 1, the additional analyses conducted in the current study indicated that fear-avoidance beliefs, catastrophising, and self-efficacy were significant predictors of pain

severity, pain-related disability, and depression (in different combinations and to different degrees for each of the outcome variables). Furthermore, the models that included these variables accounted for a greater proportion of the variance in the outcome measures than the models that included type of onset of pain (and compensation status). This raises the question of the clinical utility of existing research which has investigated type of onset of pain. Although previous studies have found statistically significant differences between onset groups, it is not clear if these differences are clinically meaningful, particularly in relation to the prediction of treatment outcome.

7.4.2. Construct validity of the PCL

As predicted, the PCL Total score was strongly correlated with all three DASS subscales, while the relationship between the PCL Total score and the RMDQ was moderate. This provided support for the construct validity of the PCL in this sample.

The construct validity of the PCL was also examined by comparing the levels of PTSD symptoms endorsed by the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. Although there was a trend in the expected direction, with the Traumatic group scoring higher on the PCL than the other two groups, this trend was not statistically significant on the PCL Total score, and the PCL Avoidance and Arousal subscales. There are a number of potential explanations for this result. Firstly, the scores of the Traumatic group may have been lower than would be expected given the low rate of PTSD in the sample (see the following section for a discussion of this issue). Secondly, the scores of the Non-traumatic group and Insidious/Spontaneous group may have been higher than would be expected due to the PCL items which assess symptoms that overlap with the features of chronic pain or that could be conceptualised as general distress. If this was the case, significant differences would have been expected between the Traumatic group and

the other two groups on the PCL Reexperiencing subscale, but not the other subscales, given that the Reexperiencing subscale measures symptoms which are arguably unique to PTSD. Consistent with this, the non-parametric tests comparing the three groups' responses on this subscale were significant, although the difference was between the two groups who had experienced a sudden onset of pain (Traumatic and Non-traumatic) and the Insidious/Spontaneous group. It is important to emphasise that the non-parametric tests do not control for the effects of covariates, and so must be interpreted with caution in this context given the significance of the covariates in the other analyses.

Additional support for the argument that some aspects of the PCL may have been measuring general distress in this sample came from the finding that younger age was significantly associated with higher scores on the PCL Total score and the PCL Avoidance subscale. The Avoidance subscale contains several items that could arguably be conceptualised as manifestations of general distress or dysphoria, for example, loss of interest in activities and restricted affect (Simms et al., 2002). Four factor analytic studies of PTSD symptoms (i.e. Simms et al., 2002; Baschnagel, O'Connor, Colder & Hawk, 2005; Elklit & Shevlin, 2007; Palmieri et al., 2007) have provided support for this argument with the identification of a four-factor structure of PTSD symptoms in which the four numbing symptoms from the DSM-IV criteria for PTSD (i.e. 4 out of the 7 items that constitute the Avoidance subscale) loaded onto a factor with the three hyperarousal symptoms that are also arguably symptoms of distress (i.e. impaired sleep, difficulty concentrating, irritability). Two of these studies used the PCL (i.e. Simms et al., 2002; Palmieri et al., 2007). Previous research has indicated that older chronic pain patients tend to be less distressed by their pain (e.g. Corran, Farrell, Helme & Gibson, 1997; Riley, Wade, Robinson & Price, 2000; Cook & Chastain, 2001; Wittink, Rogers, Lipman, McCarberg, Ashburn, Oderda & Carr, 2006). Accordingly, the higher scores obtained by

younger participants on the PCL Avoidance subscale could be attributable to some of those items being related to general distress.

Thus, the correlational analyses aside, examination of the PCL's validity in this sample by a comparison of the PCL scores of the traumatic onset groups provided some justification for the concerns about overlapping symptoms expressed in previous chapters. However, given the limitations of the method used to allocate participants to the Traumatic onset groups, and the lack of significant results between these groups in the main analyses, it is difficult to draw any firm conclusions about the PCL's apparent difficulty distinguishing between the Traumatic, Non-traumatic, and Insidious/ Spontaneous groups.

7.4.3. Diagnostic utility of the PCL

The application of cut-off scores and the diagnostic algorithm derived from previous research suggested that up to 31% of the participants in this study could potentially be diagnosed with PTSD. This is similar to the figure reported in Study 1. According to the diagnostic algorithm, 47% of the Traumatic group and 40% of the Non-traumatic group would have potentially met diagnostic criteria for PTSD. Furthermore, 17% of the Insidious/Spontaneous group would have potentially met diagnostic criteria, despite the fact that these participants had not identified a specific precipitating event for their pain.

Information about PTSD symptoms retrieved from the participants' medical files suggested that only 7% of the sample had reported symptoms of PTSD at the time of their initial assessment at the Centre. Most of these patients endorsed posttraumatic stress symptoms above the median for the sample, and all but one had been placed in the

Traumatic onset group. This provided support for the validity of the PCL in the sample, and for the accuracy of the method used to categorise participants into onset groups.

In contrast, the discrepancy between the PTSD symptom data obtained from the medical files and the estimates obtained from the cut-off scores and diagnostic algorithm casts doubt over the accuracy of the cut-off scores and diagnostic algorithm in this sample. PTSD prevalence rates reported in the literature provide support for the view that the cut-off scores and diagnostic algorithm probably overestimate the prevalence of PTSD in samples such as the one employed in this study.

Firstly, general population surveys that have used structured clinical interviews to diagnose PTSD in individuals with chronic pain (i.e. McWilliams et al., 2003; Von Korff et al., 2005) have reported 12-month prevalence rates of 7.3% and 10.7%, respectively. Similar figures have been reported in treatment-seeking samples of orofacial pain patients, with studies that used structured clinical interviews (i.e. Aghabeigi et al., 1992; Sherman et al., 2005) reporting current PTSD rates of 6% and 11.3%, respectively. Higher rates of PTSD have been reported in other studies of chronic pain patients (e.g. approximately 30% - 75%; Hickling et al., 1992a; Chibnall & Duckro, 1994); however, these higher rates have tended to come from samples of patients who have all been exposed to a potentially traumatic event (e.g. groups of motor-vehicle accident victims) and this is not the case in the current sample. Taking these studies into account with evidence that tertiary pain management centre samples tend to be more disabled and distressed than community samples of individuals with chronic pain (Crook, Tunks, Kalaher & Roberts, 1988; Kung, Gibson & Helme, 2000), PTSD rates in samples like the one employed in the current study may be higher than the general community surveys would suggest, but not as high as studies of chronic pain patients who have all been exposed to a potentially traumatic event.

Further evidence that the diagnostic algorithm overestimates the prevalence of PTSD in chronic pain patient samples can be found in studies conducted with other trauma groups. In a study of motor-vehicle accident survivors, Ehlers et al. (1998) reported that the avoidance symptom cluster largely determined whether an individual met diagnostic criteria for PTSD. That is, approximately half of their sample met criteria on the basis of the reexperiencing and hyperarousal symptom clusters, but then did not report sufficient numbing symptoms to meet full diagnostic criteria. Other studies have reported similar findings (Solomon & Canino, 1990; Norris, 1992). This is problematic for diagnosing PTSD in chronic pain patient samples due to the nature of the symptoms in the avoidance/numbing symptom cluster, as strict application of the diagnostic algorithm could lead to patients being diagnosed with PTSD because they have endorsed symptoms from that cluster that are related to their chronic pain experience.

At the same time, when considering these criticisms of the accuracy of the diagnostic algorithm in this sample, it is important to keep the limitations of the current study in mind. Firstly, the reliance on information obtained from medical files to indicate levels of PTSD symptomatology in the sample is a methodological weakness. As would be expected, problems related to chronic pain are the focus of the multidisciplinary assessments at the Centre, and a detailed assessment of comorbid conditions is not always possible. In addition, screening for PTSD symptoms may be overlooked if the event associated with the onset of pain does not immediately appear to have been a potentially traumatic event. These factors may have led to the file notes being a limited indicator of the true prevalence of PTSD symptoms in the sample.

In addition, it is possible that the Non-traumatic and Insidious/Spontaneous groups actually did contain participants who would have met diagnostic criteria for PTSD had this been assessed. Firstly, as already discussed, the method used to allocate participants

into the onset groups was not without its limitations and some participants who had experienced a traumatic onset of pain could have been placed in the incorrect onset group. However, this is unlikely given the high degree of agreement between the two raters, and the high degree of correspondence between group membership and the PTSD symptoms retrieved from the medical files.

Alternatively, as some authors have argued (Solomon & Canino, 1990; Avina & O'Donohue, 2002; Gold et al., 2005; Mol et al., 2005), the range of events that elicit symptoms consistent with PTSD may be broader than the DSM-IV conceptualisation allows. Again, if this is the case, participants who had experienced a traumatic onset of their pain and were experiencing PTSD symptoms related to this event may have been allocated to the incorrect group by the raters, who were following DSM-IV diagnostic criteria.

Finally, the case described in the results section underscores the fact that participants could have endorsed symptoms related to prior or subsequent events despite the modifications made to the PCL. In other words, the attempt to link onset of pain with posttraumatic stress symptoms may not have been entirely successful, and at least some responses on the PCL could have been related to other traumatic events. As argued in Chapter 4, any co-occurrence of chronic pain and PTSD may help to shed light on important interactions between the two conditions, and as such, PCL responses to non-pain related traumatic events are not unimportant. However, research that focuses specifically on the relationship between onset of pain and PTSD is important for elucidating the mechanisms underlying the development of dysfunction and distress when the two conditions emerge from the one event.

Clinically, it is important to diagnose PTSD in chronic pain patients regardless of whether it predated the pain, was associated with its onset, or developed afterwards

because regardless of the temporal connection PTSD is likely to complicate the patient's presentation and have implications for treatment. This is the focus of the study presented in the following chapter.

7.5. Summary

The primary aim of this study was to investigate the impact of onset of pain on pain severity and pain-related adjustment. Comparisons between patients who had experienced different types of onset of pain revealed few significant differences between them. The results did support existing evidence that compensation status and pain duration are predictors of pain severity and pain-related adjustment.

The main strength of this study was the linkage of posttraumatic stress symptoms to the event associated with the onset of pain, and the use of clinical data to evaluate the accuracy of diagnostic methods recommended in the literature in a sample typical of the heterogeneous patients who present to tertiary referral multidisciplinary pain management centres. Although the study provided some evidence for the validity of the PCL in this patient group, the results also substantiated concerns about the role of overlapping symptoms in inflating estimates of the prevalence of PTSD in chronic pain patient samples. The obvious implication for interpretation of the existing chronic pain/PTSD literature is that studies employing self-report measures that are not adequately validated for use in chronic pain samples, and that do not attempt to establish connections between posttraumatic stress symptoms and exposure to trauma, could be overstating the rate of co-occurrence of chronic pain and PTSD. Further research employing standardised measures of trauma exposure and interview approaches to assessing posttraumatic stress symptoms should provide more reliable estimates of the prevalence of PTSD in chronic pain.

Finally, the results of this study also raise questions about the importance of onset of pain, particularly from a clinical perspective. The aim of the final study in this thesis is to investigate the potential impact of onset of pain on treatment outcome.

8. STUDY 3 - IMPACT OF TYPE OF ONSET OF PAIN AND POSTTRAUMATIC STRESS SYMPTOMS ON TREATMENT OUTCOME

8.1. Introduction

As outlined in Chapter 5, few studies have investigated the impact of onset of pain on treatment outcome. The studies which have addressed this issue suggest that pain related to an injury or accident is associated with poor response to treatment for chronic pain (Tsushima & Stoddard, 1990; Romanelli et al., 1992; Turk et al., 1998a). However, there have been no investigations of the impact of type of onset of pain on response to cognitive-behavioural treatments for chronic pain in groups of heterogeneous chronic pain patients. In Study 2, the importance of type of onset of pain in influencing adjustment to chronic pain was brought into question. The study found few differences between patients who presented with pain related to accidents and other specific incidents and patients who had experienced an insidious or spontaneous onset of pain. Despite the findings of previous research, it is not clear to what extent onset of pain is a clinically useful variable, particularly as a predictor of response to treatment.

Accordingly, the first aim of the final study in this thesis was to investigate the impact of type of onset of pain on response to a multidisciplinary, cognitive-behavioural pain management program. One modification was made to the two sets of group comparisons conducted in Study 2. Based on evidence from previous research that pain related to any specific event is associated with poor adjustment to chronic pain, patients with pain related to accidents and other specific incidents were combined and were compared to patients who had experienced an insidious or spontaneous onset of pain. The second group comparison (i.e. Traumatic, Non-traumatic, and Insidious/Spontaneous) was not changed.

Another variable which is related to type of onset of pain and which may be predictive of treatment outcome is posttraumatic stress symptoms. Although there is little research examining the relationship between posttraumatic stress symptoms and response to treatments for chronic pain, there is evidence that patients who present with both chronic pain and PTSD report higher levels of pain, pain-related disability, and affective distress than patients who present with chronic pain alone (see Chapter 4). Therefore, it is quite possible that the presence of posttraumatic stress symptoms could complicate treatment for chronic pain, and a small number of case studies support this hypothesis (Muse, 1986; Hickling et al., 1992b). Consequently, the second aim of this study was to determine if posttraumatic stress symptoms are predictive of response to a multidisciplinary, cognitive-behavioural pain management program.

Study 2 also indicated that cognitive variables (i.e. self-efficacy, catastrophising, and fear-avoidance beliefs) were significant predictors of pain severity and pain-related adjustment across all of the onset groups. As noted in Chapter 1, these variables have also been shown to be predictive of, or mediate, changes in disability and distress in cognitive-behavioural programs (e.g. Jensen, Turner & Romano, 1994b; Burns, Kubilus, Bruehl, Harden & Lofland, 2003; Spinhoven, Ter Kuile, Kole-Snijders, Hutten Mansfeld, Den Ouden & Vlaeyen, 2004). In light of the results of Study 2 and evidence from the literature, these process variables were also included in the analyses of treatment outcome in the current study.

Based on Turk et al.'s (1996) findings that fibromyalgia patients with accident-related pain were more likely to have been prescribed opioid medication, and had trialled more treatments, treatment history was also included in the comparisons of onset groups.

Finally, as compensation status was identified as a potential predictor of response to injury-related pain in Study 2 this variable was also included in the analyses. This may

be particularly important in the present study as a large proportion of participants in the Accident/Specific Incident, Traumatic and Non-traumatic groups reported being involved in compensation claims.

8.2. Method

8.2.1. Participants

The participants were patients who attended the ADAPT pain management program at the University of Sydney Pain Management and Research Centre between June 2004 and January 2006. The participants were originally recruited from 35 different treatment groups delivered in the Centre during this period; however, due to administrative error within the clinic (where the data for 14 groups were mislaid, at least initially) only the data for 21 of these groups was available for analysis. The data for another 12 groups were later located and were used as a comparison data-set to ensure that the 21 groups analysed in the current study were not significantly different to the larger sample. Cross-sectional comparisons of the two data-sets are provided in Appendix G. These analyses indicated that there were no significant differences between the study groups and the comparison groups prior to treatment.

Thus, the participants in Study 3 were 128 patients with heterogeneous pain conditions and sites, including 48 males (37.5%) and 80 females (62.5%), who attended the ADAPT pain management program during the period noted above. Only two patients were excluded because they did not provide consent to be involved in research at the Centre. Details of the participants' characteristics are provided in the Results section.

8.2.2. Procedure

Procedures for obtaining demographic and clinical information and consent to participate in the study were the same as those described for the previous studies.

Participants were allocated to onset groups using the same procedure as Study 2. One of the experts who coded the data for Study 2 was not available, so another independent clinical psychologist with extensive clinical and research experience in the field of PTSD coded the data in addition to one of the original experts from Study 2.

ADAPT pain management program

The ADAPT pain management program is a multidisciplinary program based on cognitive-behavioural principles. Patients attend the program as day patients Monday to Friday from 9am to 5pm for three weeks in groups of 8-10. The content of the program is based on that described in the randomised controlled trial by Williams, Richardson, Nicholas et al. (1996a) and formalised in the manual derived from the UK program but modified for use within Australia (Nicholas, Molloy, Tonkin & Beeston, 2000). The program includes education about chronic pain, individual and group exercises aimed at gradually increasing performance of both exercises and specific functional activities despite pain (e.g. walking, sitting), instruction in pain coping strategies (e.g. goal setting, activity pacing, applied relaxation, interoceptive exposure to pain experience, management of flare-ups and set-backs, problem solving, and cognitive restructuring), and gradual withdrawal of pain-related medication. The program is staffed by a clinical psychologist, physiotherapist, and nurse, with input from medical staff. After the three-week phase of the program (Stage 1), patients are encouraged to work on applying the strategies they have learned in their daily lives for one month (Stage 2), before returning for a follow-up session to review their progress. At the end of either stage of the program,

patients who are in need of further intervention (for pain management or for co-morbid conditions) may be seen by the staff clinical psychologist or referred to local psychologists for individual treatment sessions. Program outcomes are assessed using measures of physical tolerances and responses to self-report psychometric questionnaires administered prior to treatment, at the end of Stage 1 of the program, and at the one-month follow-up.

Following the multidisciplinary assessment at the Centre, patients are referred to the ADAPT program if they present with a history of persisting pain (more than six months), high levels of pain-related disability and/or distress, excessive reliance on medications and unnecessary aids, or if they have ceased or significantly reduced work activities due to pain. Exclusion criteria include: not being willing or motivated to participate in the program; inadequate English language skills; poorly controlled or acute psychiatric symptoms (e.g. psychosis, mania, or acute suicidality); and suitability for further medical treatments.

8.2.3. Measures

The same questionnaire battery used in the two previous studies was administered in Study 3. The self-report measures which were not the focus of the other studies are described in detail below.

The Pain Self-Efficacy Questionnaire (PSEQ)

The PSEQ (Nicholas, 1989) assesses an individual's degree of confidence that they can perform specific tasks despite their pain. The PSEQ consists of 10 items enquiring about activities that are commonly reported as being difficult by chronic pain patients, for example, household chores, working, and socialising. The PSEQ asks patients to rate how confident they are that they can perform the particular task or engage in the activity at

present despite the pain, for example, “I can do most of the household chores (e.g. tidying-up, washing dishes, etc.) despite the pain”. Each item is rated by the respondent on a 7-point scale (0 = “Not at all confident” and 6 = “Completely confident”). The PSEQ is scored by summing the scores obtained for each of the 10 items, resulting in a total score that can range between 0 (low self-efficacy) and 60 (high self-efficacy).

The PSEQ has been shown to have excellent reliability and validity across a number of studies (Nicholas, 1989; Gibson & Strong, 1996; Nicholas, 2007). In the current study, the Cronbach alpha = 0.920. The PSEQ has also been shown to have good predictive utility, with scores on the PSEQ reported to be a significant predictor of pain behaviours (Asghari & Nicholas, 2001) and self-rated disability (Ayre & Tyson, 2001). The PSEQ is also sensitive to treatment changes in cognitive-behavioural pain management programs (Nicholas, Wilson & Goyen, 1992; Williams, Nicholas, Richardson, Pither, Justins, Chamberlain, Harding, Ralphs, Jones, Dieudonne & et al., 1993; Williams et al., 1996a), and is predictive of both treatment outcome and drop-out (Coughlan, Ridout, Williams & Richardson, 1995; Strong, Westbury, Smith, McKenzie & Ryan, 2002).

The Tampa Scale for Kinesiophobia (TSK)

The TSK (Kori et al., 1990) measures fear of movement and (re)injury (e.g. “I’m afraid that I might injure myself if I exercise”, “Pain lets me know when to stop exercising so that I don’t injure myself”). The TSK consists of 17 items scored on a 4-point scale. Respondents are instructed to circle the number for each item that corresponds to how they feel about each statement. The rating scale is: 1 = “Strongly agree”; 2 = “Somewhat disagree”; 3 = “Somewhat agree”; and 4 = “Strongly agree”.

Items numbered 4, 8, 12 and 16 are reverse-scored. Item scores are summed to obtain a total score ranging from 17 to 68.

Previous studies have reported that in chronic back pain samples scores on the TSK are positively associated with catastrophising cognitions (Vlaeyen et al., 1995a), and dysphoric mood (Vlaeyen et al., 1995a; Crombez et al., 1999). Crombez et al. reported that the TSK was a better predictor of disability in chronic back pain patients than pain intensity, pain duration, and negative affect. Studies have also demonstrated that the TSK is a significant predictor of behavioural performance in chronic back pain patients (Vlaeyen et al., 1995a) and is in fact a better predictor than self-report disability or catastrophising (Crombez et al., 1999).

The TSK has been reported to have good internal consistency in samples of chronic back pain patients with reported Cronbach's alpha coefficients ranging from 0.68 to 0.8 (Vlaeyen et al., 1995a; Crombez et al., 1999). In the current study, the Cronbach alpha = 0.797. A number of studies have examined the factor structure of the TSK in chronic pain samples, with the earliest study (Vlaeyen et al., 1995b) obtaining a four-factor solution and more recent studies arguing in favour of a two-factor solution (labelled "activity avoidance" and "somatic focus") which omits the four reverse-scored items (Goubert, Crombez, Van Damme, Vlaeyen, Bijttebier & Roelofs, 2004; Roelofs, Goubert, Peters, Vlaeyen & Crombez, 2004). In the current study only the total TSK score was used following recommendations that the total score is the most reliable and valid (Vlaeyen et al., 1995b; Roelofs et al., 2004).

Pain-Related Self-Statements Scale (PRSS)

The Pain-Related Self-Statements Scale (Flor & Turk, 1988) assesses automatic thoughts experienced by chronic pain patients during episodes of pain. It includes 18

items consisting of two, 9-item subscales. The first subscale, "Catastrophising", refers to self-statements focusing exclusively on the aversive aspects of the pain experience (Flor & Turk, 1988). Items in this subscale include, "I cannot stand this pain any longer", "This pain is killing me" and "I can't go on anymore". The second subscale, named "Coping", includes self-statements that reflect a focus on ways of coping with the pain (e.g. "If I stay calm and relaxed things will be better", "Distraction helps best"). Patients are asked to rate on a 6-point scale how often they experience each thought when their pain is severe (0 = "almost never" to 5 = "almost always"). Each subscale score is derived by calculating the mean rating for the nine items in that subscale. Higher scores indicate higher levels of catastrophising and coping self-statements.

The PRSS has been shown to have good psychometric properties in patients with back pain, rheumatoid arthritis, and heterogeneous pain patients (Flor & Turk, 1988; Flor, Behle & Birbaumer, 1993). In a comparison of cognitive measures in a sample of patients with chronic low back pain Main and Waddell (Main & Waddell, 1991) replicated the factor structure of the PRSS and reported a strong relationship between the Catastrophising subscale and depressive symptoms. Providing further support for the validity of the PRSS, Main and Waddell also reported a high correlation between the PRSS and another widely used cognitive measure (The Coping Strategies Questionnaire; Rosenstiel & Keefe, 1983). In this study, only the Catastrophising subscale was included in the analyses. The Cronbach alpha = 0.856.

8.2.4. Aims and hypotheses

The aims of this study were to investigate the impact of type of onset of pain on response to a multidisciplinary, cognitive-behavioural pain management program, and to

determine if posttraumatic stress symptoms are predictive of response to such a program. The objectives and hypotheses were: -

(1) To determine if patients in the Accident/Specific Incident group report on average significantly smaller changes in the core outcome variables of pain severity, pain-related disability, and depressed mood, compared with patients in the Insidious/Spontaneous group.

(2) To determine if patients in the Traumatic group report on average significantly less change in the core outcome variables, compared with the Non-traumatic and Insidious/Spontaneous groups. It was also predicted that the Non-traumatic group would report on average significantly less change in the treatment outcome variables compared with the Insidious/Spontaneous group.

(3) To determine if posttraumatic stress symptoms (as measured by the PCL) are predictive of changes in the core treatment outcome variables. It was predicted that the PCL would not be a significant predictor of changes in pain severity or pain-related disability, but that it would predict changes in depressive symptoms. In particular, it was predicted that because of the items included in these subscales, the PCL Avoidance and Arousal subscales would be significant predictors of changes in depressive symptoms. Related to this objective, it was also predicted that the cognitive process variables (i.e. catastrophising, self-efficacy, and fear-avoidance beliefs) would be significant predictors of changes in the three core treatment outcome variables for all of the onset groups. Specifically, it was predicted that higher scores on catastrophising and fear-avoidance beliefs would be associated with smaller changes on the outcome variables, while higher scores on self-efficacy beliefs would be associated with larger changes on the outcome variables.

(4) To determine if onset of pain is associated with higher levels of medication use and having trialled a greater number of treatments prior to referral to the Centre.

8.2.5. Data analyses

All analyses were conducted using the SPSS v. 16.0 and SAS Version 8.2 for Windows (SAS Institute, Cary, N.C.). Statistical significance was set at $p < 0.05$.

Preliminary analyses

Questionnaires were considered incomplete if more than 30% of responses for the total scale or subscales were missing. For the DASS, the scales were considered incomplete if more than two items were missing (P. Lovibond, personal communication, 30 May 2005). In order to maximise the amount of data available for the analysis of treatment effects, participants were not excluded if they had not completed all of the questionnaires. Following Tabachnik and Fidell (2001), it was not considered a significant problem if less than 5% of data points were missing from a particular variable in a random pattern. Variables with more than 5% of data points missing were examined in further detail by testing for patterns in the missing data.

Main analyses

Two sets of cross-sectional comparisons were made between onset groups on a number of demographic and clinical variables. The first set involved comparing the Accident/Specific Incident group with the Insidious/Spontaneous group. The second set involved comparing the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. The groups were compared on age, pain duration, gender, compensation status, number of medications being used at pre-treatment, and the number of treatments trialled before

ADAPT. Comparisons were conducted using one-way ANOVAs, chi-square tests, or Fisher's exact tests, as appropriate.

To analyse the effects of treatment over time, differences between onset groups were examined using group differences in mean changes in the outcome variables from baseline to post-treatment (i.e. the end of Stage 1 of the program) and to the one-month follow-up (i.e. the end of Stage 2 of the program). A likelihood-based, mixed-effects model repeated measures (MMRM) approach was selected because of the advantages it confers over traditional repeated measures ANOVAs (Gueorguieva & Krystal, 2004). In particular, it allows estimation of both group and individual trends over time, it can manage both time-dependent and time-independent covariates, and does not require that the data meet assumptions of sphericity and normality. It also deals with missing data and treatment drop-outs more effectively than traditional approaches by using all available data for each participant, and it is not affected by randomly missing data. In addition, it results in more accurate estimates of treatment effect (and thus requires fewer patients to achieve certain levels of power) by allowing the investigator to choose between alternative patterns of covariance through the application of goodness-of-fit indices (such as the Akaike Information criterion).

In this study, the longitudinal model included: the core outcome variables of pain severity, pain-related disability, and depression; the fixed categorical effects of onset group, time point, onset group by time point interaction, day, day by onset group interaction; the fixed continuous effects of age, pain duration, and the baseline score on the outcome variables; and the fixed categorical effects of compensation status and gender. Pain self-efficacy (PSEQ), fear-avoidance beliefs (TSK), catastrophising (PRSS – Catastrophising subscale), and posttraumatic stress symptoms (PCL – Total score) were included in the model as predictors of the three outcome variables. It was decided *a priori*

that if the PCL Total score was a significant predictor of any of the outcome variables another model would be generated that separated the PCL Total score into the three PCL subscales in order to determine which subscale(s) were predictive of treatment outcome.

The MMRM analysis assessed overall average adjusted (LS Means) change from baseline to endpoint group differences. The longitudinal MMRM analysis first examined the effectiveness of the ADAPT program for the entire sample over all time periods. The models were then used to assess between group differences over time for the two sets of onset group comparisons (i.e. Accident/Specific Incident vs. Insidious/Spontaneous group and Traumatic vs. Non-traumatic vs. Insidious/Spontaneous groups). A sub-groups analysis of the process variables (i.e. PRSS-Catastrophising, PSEQ, TSK) by onset group over time was explored. Each of the process variables were dichotomised according to the median. The interaction between the process variables split at the median and the onset groups was examined. All outcomes were assessed over all time points to allow comparisons of group differences in treatment response at post-treatment, at the follow-up, and overall.

Selection of the unstructured covariance structure or the compound symmetry covariance structure to estimate within-patient errors was based on convergence to the best fit as determined by the lowest value obtained of Akaike's information criterion. The KENWARD-ROGER method was used to estimate denominator degrees of freedom. Type III sum-of-squares for the least-squares means was used.

Effect sizes were computed for clinical interpretation of the differences in treatment response between the onset groups. They were calculated using the overall least square mean change from pre-treatment to follow-up for all outcome measures for both sets of group comparisons taken from the MMRM. The MMRM was adjusted for the variables previously listed. The difference between the scores was divided by the square

root of the pooled estimate of the residual error from the MMRM. Higher effect sizes indicate a greater difference in treatment response between the onset groups. Cohen's (1988) guidelines were applied to interpret effect sizes; that is, an effect size of 0.2 = small; 0.5 = moderate; and 0.8 and above = large.

8.3. Results

8.3.1. Participant characteristics

The demographic and clinical characteristics of the participants in this study are presented in Table 8.1. The mean age of participants was 44 years (SD = 11.24; range = 14-65 years of age), and the mean duration of pain was 87.75 months or approximately 7.3 years (SD = 99.45; 7-573). The most common single pain site was lower back and lower limbs (46 individuals or 35.9%), with 34 patients (or 26.6%) reporting pain in two or more major sites. Seventy-four participants (58.3%) were involved in a compensation claim or legal case associated with their injury.

8.3.2. Preliminary analyses

Ninety-five participants (74.2% of the sample) completed Stages 1 and 2 of the program. That is, these patients completed the 3-week day patient phase and attended the one month follow-up. Seven patients (5.5%) did not complete Stage 1. Twenty-six patients (20.3%) completed Stage 1, but did not attend the one month follow-up. These figures are consistent with existing data regarding ADAPT program completion rates (M. K. Nicholas, personal communication, 26 August 2008).

Not including data that was incomplete because patients did not complete Stage 1, or did not attend the follow-up, there were very few incomplete questionnaires (pre-treatment = 6, post-treatment = 10, follow-up = 10); therefore, no variables were missing

more than 5% of data points. Accordingly, a missing values analysis was not required. Incomplete questionnaires were not included in the main analyses, but as noted in the Method section, participants were not excluded if they had not completed all of the questionnaires.

Table 8.1: Participants' demographic and clinical characteristics

Variable	
Age (years)	n = 128
M (SD)	44.55 (11.24)
Range	14-65
Gender	n = 128
Male	48 (37.5%)
Female	80 (62.5%)
Marital status	n = 126
Married/De facto	77 (61.1%)
Divorced/Separated	20 (15.9%)
Widowed	6 (4.8%)
Single	23 (18.3%)
Highest Level of Education	n = 120
Post high school qualification	29 (24.2%)
Up to and including high school	91 (75.8%)
Work status	n = 125
Working	43 (34.4%)
Not working	82 (65.6%)
Compensation status:	n = 127
Involved in compensation claim or legal case	74 (58.3%)
Not involved in claim or legal case	53 (41.7%)
Pain duration (months)	n = 128
M (SD)	87.75 (99.45)
Range	7 - 573
Pain site	n = 128
Head, face and mouth	4 (3.1%)
Cervical region	6 (4.7%)
Upper shoulder and upper limbs	25 (19.5%)
Thoracic region	3 (2.3%)
Abdominal	1 (.8%)
Lower back, lumbar spine and sacrum	1 (.8%)
Lower limbs	8 (6.2%)
Pelvic region	0 (0%)
Anal, peri-anal and genital	0 (0%)
Lower back and lower limbs	46 (35.9%)
More than 2 major sites	34 (26.6%)

8.3.3. Main analyses

Onset group characteristics

Information about one participant's onset of pain was not available, so the main analyses were conducted with a sample size of N = 127. The onset of pain data for the Accident/Specific Incident and Insidious/Spontaneous groups is presented in Table 8.2. For the first group of comparisons, 92 participants (72.4%) were allocated to the Accident/Specific Incident group and 35 (27.6%) were allocated to the Insidious/Spontaneous group.

Table 8.2: Onset of pain data for the Accident onset comparisons

Variable	Accident/ Specific Incident	Insidious /Spontaneous
How did your pain begin?	n = 92	n = 35
Accident at work	55 (59.8%)	-
At work, not involving an accident	-	9 (25.7%)
Accident at home	5 (5.4%)	-
Motor vehicle accident	18 (19.6%)	-
After surgery	4 (4.3%)	-
After illness	1 (1.1%)	-
Pain just began, no clear reason	-	22 (62.9%)
Other	9 (9.8%)	4 (11.4%)

Inter-rater reliability between the two experts who coded the events associated with the onset of pain was high: Kappa value = 0.796, $p < 0.001$ (Landis & Koch, 1977). The raters disagreed regarding the nature of nine events. Rather than deleting these participants from the analyses an independent clinical psychologist with 10 years of experience in the area of chronic pain and anxiety disorders was asked to code these nine events. Two of the nine events were rated as traumatic while the other seven were rated as non-traumatic. The nine participants were allocated to the appropriate group on the basis of these ratings. Similarly to Study 2, Table 8.3 indicates that the raters judged the

majority of work-related accidents to be non-traumatic and all the motor-vehicle accidents as traumatic or potentially traumatic. Pain that was associated with surgery or illness was classified as non-traumatic.

Table 8.3: Onset of pain data for the traumatic onset comparisons

Variable	Traumatic	Non-traumatic	Insidious/Spontaneous
How did your pain begin?	n = 34	n = 58	n = 35
Accident at work	12 (35.3%)	43 (74.1%)	-
At work, not involving an accident	-	-	9 (25.7%)
Accident at home	1 (2.9%)	4 (6.9%)	-
Motor vehicle accident	18 (52.9%)	0 (0%)	-
After surgery	0 (0%)	4 (6.9%)	-
After illness	0 (0%)	1 (1.7%)	-
Pain just began, no clear reason	-	-	22 (62.9%)
Other	3 (8.8%)	6 (10.3%)	4 (11.4%)

Onset group comparisons

Cross-sectional comparisons revealed that compared with the Insidious/Spontaneous group, the Accident/Specific Incident group was younger, had experienced pain for a shorter period of time, and was more likely to be involved in a compensation claim or legal case (see Table 8.4). Similarly, compared with the Insidious/Spontaneous groups, the Traumatic and Non-traumatic groups were younger, reported shorter pain duration, and were more likely to be involved in a compensation claim or legal case (see Table 8.5). There were no significant differences between the Traumatic and Non-traumatic groups on these variables. There were no significant differences between the Accident/Specific Incident group and Insidious/Spontaneous group on the number of treatments the participants had trialled prior to the ADAPT program, or the number of medications they were taking at the time of starting the

program. There were also no differences on these variables between the Traumatic, Non-traumatic, and Insidious/Spontaneous groups.

Table 8.4: Demographic and clinical variables by group (Accident/Specific Incident comparisons)

Variable	Accident / Specific Incident	Insidious / Spontaneous	*F*-ratio / Fisher's Exact	*p*
Age (years)	n = 92	n = 35	8.902	.003
M (SD)	42.64 (10.52)	49.06 (11.62)		
Pain duration (mths)	n = 92	n = 35	8.511*	.005
M (SD)	69.86 (81.68)	136.43 (125.24)		
Gender	n = 92	n = 35		
Male	41 (44.57%)	7 (20.0%)	6.508	.014
Female	51 (55.43%)	28 (80.0%)		
Attendance related to claim or legal case?	n = 91	n = 35		
Yes	68(74.73%)	6 (17.14%)	34.58	< .001
No	23 (25.27%)	29 (82.86%)		
No. of medications	n = 88	n = 32	1.142	.287
M (SD)	2.4 (1.65)	2.06 (1.32)		
No. of treatments	n = 89	n = 30	.402	.527
M (SD)	6.87 (3.48)	7.33 (3.55)		

* Levene's test of homogeneity of variances was significant (p = 0.005) so Welch & Brown-Forsythe robust tests of equality of means statistic and p-value is provided.

Table 8.5: Demographic and clinical variables by group (Traumatic onset comparisons)

Variable	Traumatic	Non-traumatic	Insidious / Spontaneous	*F*-ratio or X^2	*p*
Age (years)	n = 34	n = 58	n = 35	4.428	.014
M (SD) [a]	42.41 (10.68)	42.78 (10.52)	49.06 (11.62)		
Pain Duration (mths)	n = 34	n = 58	n = 35	4.518/	.015/
M (SD) [a]	73.26 (107.50)	67.86 (62.85)	136.43 (125.24)	5.142	.008[b]
Gender	n = 34	n = 58	n = 35		
Male	19 (55.88%)	22 (37.93%)	7 (20.0%)	9.446	.009
Female	15 (44.12%)	36 (62.07%)	28 (80.0%)		
Attendance related to claim or legal case? [a]	n = 34	n = 57	n = 35		
Yes	27 (79.41%)	41 (71.93%)	6 (17.14%)	35.072	< .001
No	7 (20.59%)	16 (28.07%)	29 (82.86%)		
No. of medications	n = 31	n = 57	n = 32	.704	.497
M (SD)	2.29 (1.44)	2.47 (1.76)	2.06 (1.32)		
No. of treatments	N = 34	N = 55	N = 30	.203	.816
M (SD)	6.82 (3.52)	6.89 (3.49)	7.33 (3.55)		

[a] Traumatic and Non-traumatic differ from Insidious/Spontaneous.
[b] Levene's test of homogeneity of variances was significant (p = 0.014) so Welch & Brown-Forsythe robust tests of equality of means statistics and p-values are provided.

Analysis of treatment outcome

Overall, when the effectiveness of the ADAPT program for the entire sample was examined, the analysis revealed significant reductions in pain severity ($p = 0.0455$), pain-related disability ($p < 0.001$), and depression ($p = 0.0014$) over all time points (see Appendix G for details). Examination of the unadjusted means revealed that these reductions constituted clinically significant changes in the outcome variables. For example, when compared to percentiles derived from the Centre's normative database (Nicholas et al., 2008), average pain severity decreased from the 57th percentile prior to the program (M = 4.135, CI = 3.96 – 4.31) to the 75th percentile post-treatment (M = 3.622, CI = 3.404 – 3.84). Similarly, pain-related disability decreased from the 53rd percentile prior to the program (M = 12.48, CI = 11.51-13.46) to the 73rd percentile post-treatment (M = 8.342, CI = 7.368 – 9.315). Finally, depression decreased from the 38th percentile prior to the program (M = 15.82, CI = 13.84 – 17.99) to the 56th percentile post-treatment (M = 9.441, CI = 7.551 – 11.33). It should also be noted that reductions of 3 or more on the RMDQ have been considered clinically significant (Deyo, Battie, Beurskens, Bombardier, Croft, Koes, Malmivaara, Roland, Von Korff & Waddell, 1998) and the depression score changes reflect a shift from moderate to mild depression (Lovibond & Lovibond, 1995b). Table 8.6 provides the unadjusted means at each time point for the total sample and the different onset groups.

Table 8.6; Unadjusted means (standard deviations) for the outcome variables for each onset group and the total sample at all time points

Variable and Timepoint		A/S	I/S	T	NT	Total
MPI-PS	Pre	4.21 (.99)	3.91 (1.06)	4.41 (0.93)	4.11 (1.04)	4.13 (1.00)
	Post	3.78 (1.23)	3.19 (1.13)	3.87 (1.34)	3.73 (1.21)	3.62 (1.22)
	F/U	3.9 (1.28)	2.59 (1.05)	4.16 (1.24)	3.77 (1.33)	3.54 (1.35)
RMDQ	Pre	13.32 (5.45)	10.31 (5.52)	12.74 (5.35)	13.67 (5.62)	12.48 (5.61)
	Post	9.16 (5.32)	6.09 (5.37)	8.94 (5.98)	9.29 (5.12)	8.34 (5.44)
	F/U	9.35 (6.3)	4.46 (3.22)	10.26 (7.54)	8.89 (5.82)	8 (5.98)
DASS-D	Pre	16.61 (11.65)	13.68 (10.68)	16.35 (10.65)	16.76 (12.47)	15.82 (11.28)
	Post	10.53 (11.07)	6.5 (8.67)	10.17 (11.79)	10.72 (10.98)	9.44 (10.47)
	F/U	13.37 (11.53)	5.31 (7.66)	16.96 (12.75)	11.5 (10.86)	11.12 (11.07)

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale. A/S = Accident/Specific Incident; I/S = Insidious/Spontaneous; T = Traumatic; NT = Non-traumatic.
F/U = 1 month follow-up.

When the outcomes were examined over all time points (taking pre-treatment scores into account), both the Accident/Specific Incident and Insidious/Spontaneous groups reported decreases in all three treatment outcome variables (see Table 8.7). Compared with the Accident/Specific Incident group, the Insidious/Spontaneous group reported on average significantly greater improvement in pain severity (see Table 8.8). When the differences at each time point were compared, the Insidious/Spontaneous group reported on average significantly greater improvement in pain severity at the one-month follow-up, compared with the Accident/Specific Incident group (LS means difference = 0.585, 95% CI = 0.126, 1.044; p = 0.013). There were no statistically significant differences in improvements in pain severity between the groups at post-treatment. There

were no statistically significant differences between the groups on pain-related disability or depression over all time points (see Table 8.8). The MMRM models for the Accident/Specific Incident comparisons are provided in Appendix G.

Table 8.7: Adjusted mean (LS mean)* for the outcome variables across all time points (Accident/Specific Incident onset comparison)

Variable	Accident/Specific Incident	Insidious/Spontaneous	Total
	Mean (95% CI)	Mean (95% CI)	Mean (95% CI)
MPI - PS	-.32 (-.51, -.13)	-.71 (-1.04, -.39)	-.52 (-.7, -.34)
RMDQ	-3.41 (-4.15, -2.68)	-3.36 (-4.6, -2.12)	-3.39 (-4.08, -2.69)
DASS -D	-4.73 (-6.29, -3.16)	-5.39 (-8.05, -2.73)	-5.06 (-6.55, -3.57)

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale. *Taken from MMRM adjusting for fixed categorical effects of onset group, time point, and onset by time point interaction, the fixed continuous effects of age, pain duration, and the baseline score on the outcome variables, the fixed categorical effects of compensation status and gender, and the continuous predictor variables posttraumatic stress symptoms, self-efficacy, catastrophising, and fear-avoidance beliefs.

Table 8.8: Adjusted mean (LS mean)* differences overall and 95% CI (Accident/Specific Incident onset comparison)

Variable	Difference LS Mean (95% CI)	*p*-value
MPI-PS	.398 (.013, .783)	.043
RMDQ	-.05 (-1.55, 1.437)	.943
DASS-D	.66 (-2.52, 3.844)	.682

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale. *Taken from MMRM adjusting for fixed categorical effects of onset group, time point, and onset by time point interaction, the fixed continuous covariates of age, pain duration, and the baseline score on the outcome variables, and the fixed categorical covariates of compensation status and gender, and the continuous predictor variables posttraumatic stress symptoms, self-efficacy, catastrophising, and fear-avoidance beliefs.

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For the Traumatic onset comparisons, when the outcomes were examined over all time points, the Traumatic, Non-traumatic and Insidious/Spontaneous groups all reported decreases in all three treatment outcome variables (see Table 8.9). However, when these improvements were compared, there were no statistically significant differences between the groups on all outcome measures (see Table 8.10). For pain severity, the difference between the average improvement reported by the Insidious/Spontaneous and Traumatic groups over all time points approached significance. The MMRM models for the Traumatic onset comparisons are provided in Appendix G.

When the differences at each time point were compared, three group differences emerged. Firstly, the Insidious/Spontaneous group reported on average significantly greater improvement in pain severity at the one-month follow-up, compared with the Traumatic group (LS means difference = 0.76; p = 0.011). Secondly, the Insidious/Spontaneous group reported on average significantly greater improvement in pain severity at the one-month follow-up, compared with the Non-traumatic group (LS means difference = 0.519, 95% CI = 0.042, 0.996; p = 0.033). Finally, the Non-traumatic group reported on average significantly greater improvement in depression at the one-month follow-up, compared with the Traumatic group (LS means difference = -4.62 95% CI = -8.48, -0.75; p = 0.02). However, these group differences at the follow-up must be interpreted with caution given the lack of statistically significant differences between the groups over all time points.

Table 8.9: Adjusted mean (LS mean)* for the outcome variables across all time points (Traumatic onset comparison)

Variable	Traumatic Mean (95% CI)	Non-traumatic Mean (95% CI)	Insidious/Spontaneous Mean (95% CI)
MPI - PS	-.24 (-.57, .081)	-.34 (-.56, -.12)	-.72 (-1.04, -.39)
RMDQ	-3.41 (-4.66, -2.15)	-3.35 (-4.23, -2.48)	-3.35 (-4.6, -2.1)
DASS -D	-3.21 (-5.87, -.56)	-5.24 (-7.08, -3.4)	-5.42 (-8.1, -2.74)

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale.
*Taken from MMRM adjusting for fixed categorical effects of onset group, time point, and onset by time point interaction, the fixed continuous covariates of age, pain duration, and the baseline score on the outcome variables, and the fixed categorical covariates of compensation status and gender, and the continuous predictor variables posttraumatic stress symptoms, self-efficacy, catastrophising, and fear-avoidance beliefs.

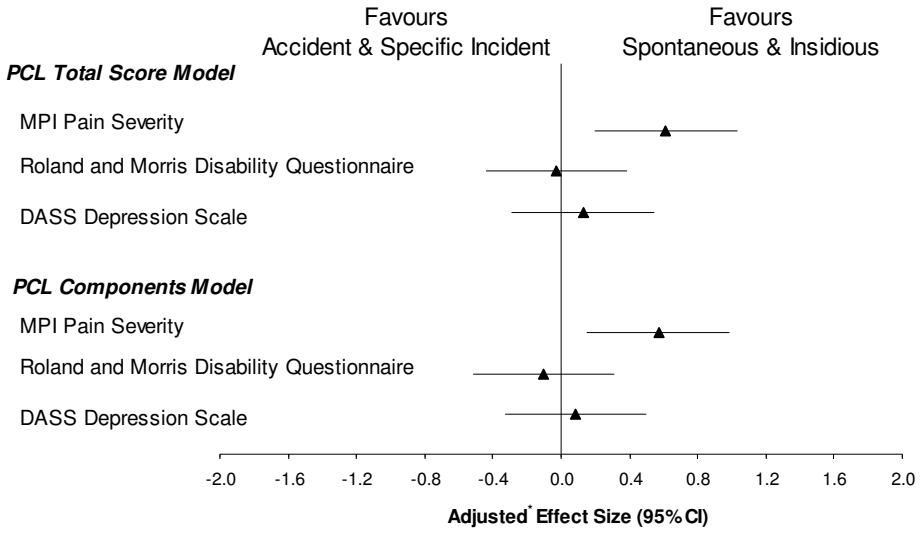
Table 8.10: Adjusted mean (LS mean)* differences overall and 95% CI (Traumatic onset comparison)

Variable	Difference (NT vs I/S) LS Mean (95% CI)	*p*	Difference (NT vs T) LS Mean (95% CI)	*p*	Difference (I/S vs T) LS Mean (95% CI)	*p*
MPI-PS	.377 (-.02, .775)	.063	-.1 (-.47, .282)	.617	-.47 (-.95, .006)	.053
RMDQ	-0 (-1.55, 1.549)	.997	.052 (-1.43, 1.532)	.944	.055 (-1.79, 1.905)	.953
DASS-D	.185 (-3.1, 3.472)	.911	-2.02 (-5.11, 1.065)	.197	-2.21 (-6.17, 1.75)	.271

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale. T = Traumatic; NT = Non-traumatic; I/S = Insidious/Spontaneous.
*Taken from MMRM adjusting for fixed categorical effects of onset group, time point, and onset by time point interaction, the fixed continuous covariates of age, pain duration, and the baseline score on the outcome variables, and the fixed categorical covariates of compensation status and gender, and the continuous predictor variables posttraumatic stress symptoms, self-efficacy, catastrophising, and fear-avoidance beliefs.

Effect sizes

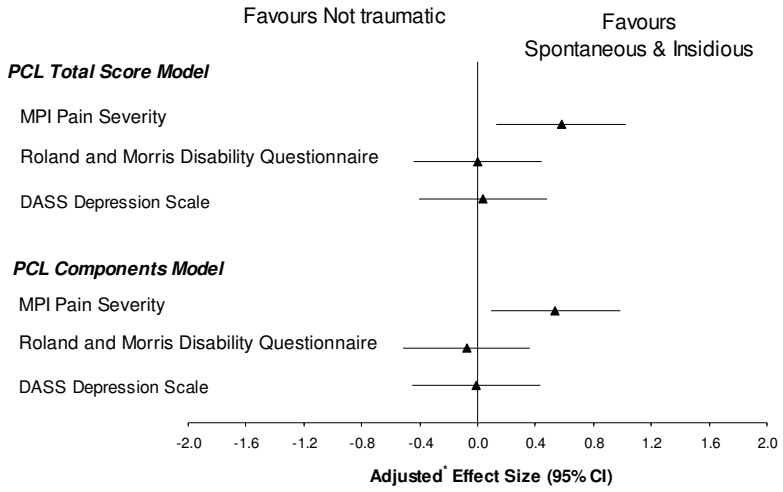
Inspection of the adjusted effect sizes (see Figure 8.1) for the Accident/Specific Incident onset group comparison indicated a moderate effect size of 0.6 for pain severity in favour of the Insidious/Spontaneous group.



*MMRM adjusted for group, time point, onset by time point interaction, age, pain duration, baseline score on outcome, compensation status, gender, PCL, PSEQ, PRSS-Cat, TSK.

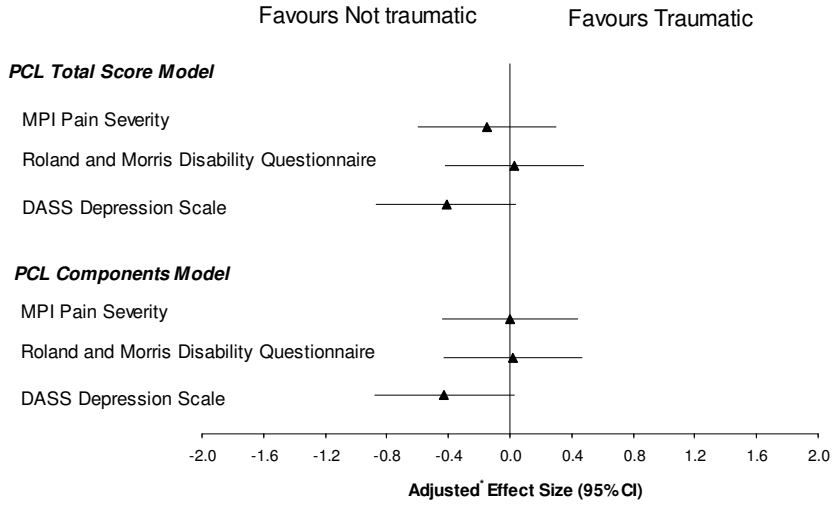
Figure 8.1: Adjusted effect sizes for the Accident/Specific Incident onset comparison

Figures 8.2 to 8.4 depict the adjusted effect sizes for the Traumatic Onset group comparisons. For changes in pain severity, every comparison which included the Insidious/Spontaneous group was in favour of this group. For changes in depression, the changes were in favour of the Insidious/Spontaneous and Non-traumatic groups.



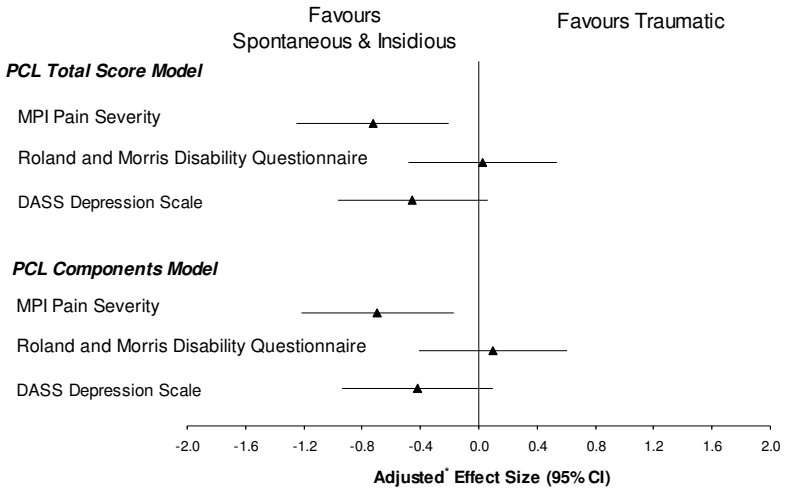
*MMRM adjusted for group, time point, onset by time point interaction, age, pain duration, baseline score on outcome, compensation status, gender, PCL, PSEQ, PRSS-Cat,

Figure 8.2: Adjusted effect sizes – Traumatic onset comparisons (Non-traumatic vs Insidious/Spontaneous)



*MMRM adjusted for group, time point, onset by time point interaction, age, pain duration, baseline score on outcome, compensation status, gender, PCL, PSEQ, PRSS-Cat,

Figure 8.3: Adjusted effect sizes – Traumatic onset comparisons (Traumatic vs Non-traumatic)



*MMRM adjusted for group, time point, onset by time point interaction, age, pain duration, baseline score on outcome, compensation status, gender, PCL, PSEQ, PRSS-Cat,

Figure 8.4: Adjusted effect sizes – Traumatic onset comparisons (Traumatic vs Insidious/Spontaneous

Predictors of treatment outcome

The results presented in this section are summarised in Table 8.11. The PCL Total score was not a significant predictor of overall changes in pain severity in both the Accident/Specific Incident and Traumatic onset group models (Accident/Specific Incident: $p = 0.546$; Traumatic: $p = 0.6354$). Catastrophising was a significant predictor of overall changes in pain severity in both onset group models, with scores below the median on the PRSS-Catastrophising scale associated with on average greater reduction in pain severity (Accident/Specific Incident: p = 0.02; Traumatic: $p = 0.0264$). Self-efficacy was also a significant predictor of overall changes in pain severity in both onset group models, with scores above the median on the PSEQ associated with on average greater reduction in pain severity (Accident/Specific Incident: $p = 0.0072$; Traumatic: $p = 0.0051$). Age and compensation status were significant predictors of overall changes in pain severity in both onset group models (Age - Accident/Specific Incident: $p = 0.0095$;

Traumatic: $p = 0.01$; Compensation status - Accident/Specific Incident: $p = 0.0475$; Traumatic: $p = 0.0499$). Older age and not being involved in a compensation claim or legal case were associated with on average greater reductions in pain severity compared with being younger and being involved in a compensation claim or legal case.

The PCL Total score was a significant predictor of overall changes in pain-related disability in the both the Accident/Specific Incident and Traumatic onset group models (Accident/Specific Incident: $p = 0.0353$; Traumatic: $p = 0.0457$). When the PCL Total score was divided into the three subscales, the Arousal subscale was a significant predictor of overall changes in pain-related disability in both group models, with higher scores associated with on average greater reductions in disability (Accident/Specific Incident: $p = 0.0162$; Traumatic: $p = 0.0253$). The MMRM model for the PCL subscales analysis is provided in Appendix G.

Self-efficacy was also a significant predictor of overall changes in pain-related disability in both onset group models, with scores above the median on the PSEQ associated with on average greater reduction in disability ($p < 0.0001$ for both onset models). Finally, fear-avoidance beliefs were a significant predictor of overall changes in pain-related disability in both group models, with scores below the median on the TSK associated with on average greater reduction in disability ($p = 0.024$ for both onset models).

The PCL Total score was a significant predictor of overall changes in depression in both the Accident/Specific Incident and Traumatic onset group models ($p < 0.0001$ in both models). When the PCL Total score was divided into the three subscales, the Arousal subscale was a significant predictor of overall changes in depression in the Accident/Specific Incident model only ($p = 0.0393$), with higher scores associated with greater reductions in depression. The Avoidance subscale was a significant predictor of

overall changes in depression in both group models, with higher scores associated with greater reductions in depression (Accident/Specific Incident comparison: $p = 0.0494$; Traumatic comparison: $p = 0.0485$). The MMRM model for the PCL subscales analysis is provided in Appendix G.

Self-efficacy was also a significant predictor of overall changes in depression in the Accident/Specific Incident model only, with scores above the median on the PSEQ associated with greater improvements in depression ($p = 0.0263$). Fear-avoidance beliefs were a significant predictor of overall changes in depression in the Traumatic onset model only, with scores below the median on the TSK associated with greater improvements in depression ($p = 0.0440$).

Table 8.11: Summary of treatment outcome predictors*

	Accident/Specific Incident model	**Traumatic model**
MPI – PS	Self-efficacy	Self-efficacy
	Catastrophising	Catastrophising
	Age	Age
	Compensation status	Compensation status
RMDQ	PCL Total score	PCL Total score
	PCL Arousal subscale	PCL Arousal subscale
	Self-efficacy	Self-efficacy
	Fear-avoidance beliefs	Fear-avoidance beliefs
DASS-D	PCL Total score	PCL Total score
	PCL Arousal subscale	PCL Avoidance subscale
	PCL Avoidance subscale	Fear-avoidance beliefs
	Self-efficacy	

Note: MPI-PS = Multidimensional Pain Inventory – Pain Severity Scale; RMDQ = Roland and Morris Disability Questionnaire; DASS-D = Depression Anxiety Stress Scales – Depression Scale.
*Taken from MMRM adjusting for fixed categorical effects of onset group, time point, and onset by time point interaction, the fixed continuous covariates of age, pain duration, and the baseline score on the outcome variables, and the fixed categorical covariates of compensation status and gender, and the continuous predictor variables posttraumatic stress symptoms, self-efficacy, catastrophising, and fear-avoidance beliefs.

Onset group by process variable interactions (sub-groups analysis)

The sub-groups analysis of the process variables (i.e. PRSS-Catastrophising, PSEQ, TSK) by onset group over time revealed no interaction effect in both the Accident/Specific Incident and Traumatic onset models. That is, although the process variables were predictive of treatment outcome as explained in the previous section, these relationships did not differ according to onset group. The MMRM models for the sub-groups analyses are provided in Appendix G.

8.4. Discussion

The aims of this study were to investigate the impact of type of onset of pain on response to a multidisciplinary, cognitive-behavioural pain management program, and to determine if posttraumatic stress symptoms are predictive of response to such a program. The key objectives of the study were: (1) to determine if patients with pain related to any specific event report on average significantly smaller changes in the core treatment outcome variables of pain severity, pain-related disability and depressed mood, compared with patients who have experienced an insidious or spontaneous onset of pain; (2) to determine if patients who have experienced a potentially traumatic onset of pain report on average significantly less change in the core treatment outcome variables, compared with the Non-traumatic and Insidious/Spontaneous groups; (3) to determine if posttraumatic stress symptoms are predictive of changes in the core treatment outcome variables; and (4) to determine if onset of pain is associated with higher levels of medication use and having trialled a greater number of treatments prior to referral to the Centre. These objectives will be discussed in the following sections.

8.4.1. Impact of type of onset of pain on treatment outcome

In summary, while the ADAPT program produced statistically and clinically significant improvements in the core outcome variables of pain severity, pain-related disability, and depression in the total sample, only a few differences were found between the improvements reported by the different onset groups after adjustment for the effects of the demographic and clinical covariates. The most significant difference between onset groups was found in improvements in pain severity, with the Insidious/Spontaneous group reporting on average significantly greater improvement in pain severity compared with the Accident/Specific Incident group over all time points, and at the one-month follow-up. This finding appeared to be part of a trend (albeit one that should be interpreted with caution) for the Insidious/Spontaneous group to maintain their post-treatment improvements in pain severity more effectively during the follow-up stage compared with the Traumatic and Non-traumatic groups. The effect size calculations were consistent with this trend, revealing that improvements in pain severity consistently favoured the Insidious/Spontaneous group.

These differences are consistent with previous research on type of onset of pain (see previous chapter). This research indicates that individuals who have experienced a spontaneous or insidious onset of pain exhibit higher levels of adjustment than individuals who experience the onset of pain in the context of an accident or another specific incident. The above findings extend this perspective to response to a cognitive-behavioural pain management program by suggesting that individuals who have experienced an insidious or spontaneous onset of pain may be more successful in maintaining treatment-related improvements in pain severity (at least in the short-term) than individuals who are experiencing pain related to a specific event.

Contrary to expectations, there were no statistically significant differences between the Accident/Specific Incident and Insidious/Spontaneous groups on improvements in pain-related disability or depression over all time points. This finding was not consistent with previous research, but was in line with the results of Study 2. As was the case in Study 2, inspection of the unadjusted means for the two groups at each time point appeared to favour the Insidious/Spontaneous group, but these differences did not remain once the effects of the covariates were taken into account.

Similarly, apparent differences in the unadjusted means between the Traumatic, Non-traumatic, and Insidious/Spontaneous groups were also not statistically significant in the adjusted models. However, in line with expectations, the effect size calculations indicated that improvements in depression favoured both the Insidious/Spontaneous and Non-traumatic groups.

Given that the most reliable differences were at the one-month follow-up, it is possible that a longer follow-up period would have revealed greater differences between groups. Despite the advantages of the MMRM approach, a larger sample size may also have produced more statistically significant differences. Alternatively, as proposed in the previous chapter, it is also possible that onset of pain does not have as much of an impact as previous research would suggest. Consistent with this, other results obtained in the current study indicated that a number of other variables were predictive of treatment outcome.

8.4.2. Predictors of treatment outcome

This aspect of the current study revealed that a number of the variables included in the different models were significant predictors of the core treatment outcome variables. Firstly, self-efficacy, catastrophising, age, and compensation status were predictors of

improvements in pain severity. Secondly, self-efficacy, fear-avoidance beliefs, and posttraumatic stress symptoms were predictors of improvements in pain-related disability. Finally, self-efficacy and post-traumatic stress symptoms were predictors of improvements in depression.

Self-efficacy, catastrophising, and fear-avoidance beliefs were expected to emerge as significant predictors of treatment outcome on the basis of numerous studies which have reported similar findings (e.g. Jensen et al., 1994b; McCracken & Gross, 1998; Jensen, Romano, Turner, Good & Wald, 1999; Burns et al., 2003; Spinhoven et al., 2004; Woby, Watson, Roach & Urmston, 2004; Smeets, Vlaeyen, Kester & Knottnerus, 2006). These results, and the fact that age and compensation status were significant predictors of improvements in pain severity were also consistent with those of Study 2. Importantly, the interaction between the cognitive variables and onset of pain was not significant in any of the models. This indicates that cognitive variables are important predictors of treatment outcome regardless of the nature of the onset of pain.

As predicted, posttraumatic stress symptoms were also a significant predictor of treatment outcome. The analyses revealed that higher levels of posttraumatic stress symptoms (in particular, the symptoms on the PCL Arousal and Avoidance subscales) were associated with smaller reductions in pain-related disability and depression. This finding that the co-occurrence of chronic pain and posttraumatic stress symptoms has a negative impact on response to treatment is consistent with theoretical perspectives on the relationship between chronic pain and PTSD (e.g. Sharp & Harvey, 2001; Asmundson et al., 2002; Asmundson & Hadjistavropolous, 2006; Otis et al., 2006), and with empirical evidence that the co-occurrence of the two conditions is associated with higher levels of pain-related disability and affective distress (e.g. Geisser et al., 1996; Sherman et al., 2000; Smith et al., 2002). In contrast to studies which have reported a relationship

between reexperiencing symptoms and chronic pain (Beckham et al., 1997; Asmundson et al., 2004) in this sample the PCL Reexperiencing subscale was not related to treatment outcome. This could have been a reflection of lower rates of endorsement of these types of symptoms in this sample compared to the other two scales, which as noted previously, contain items commonly experienced in chronic pain.

8.4.3. Onset of pain and treatment history

Based on previous research, it was hypothesised that onset of pain related to a specific incident would be associated with higher levels of medication use and having trialled a greater number of treatments prior to referral to the Centre. This hypothesis was not confirmed as no differences were found between the onset groups in treatment history or the number of medications being used at the time of entry into the ADAPT program.

Similar to other findings regarding onset of pain, evidence of differences in treatment history and current treatments comes from a study of fibromyalgia patients (Turk et al., 1996); and consequently, may not be applicable in heterogeneous samples of chronic pain patients.

8.5. Summary

The aims of this study were to investigate the impact of type of onset of pain on response to a multidisciplinary, cognitive-behavioural pain management program, and to determine if posttraumatic stress symptoms are predictive of response to such a program. A detailed analysis of differences in the improvements reported by the different onset groups in the core treatment outcome variables revealed a number of important differences. In particular, patients who had experienced an insidious or spontaneous onset of pain reported greater improvements in pain severity, and maintained these improvements more successfully, than individuals who had experienced the onset of pain

in the context of an accident or other specific event (whether or not the event was traumatic). Similarly, there was also some evidence that patients who had experienced an insidious or spontaneous onset of pain, and those who had experienced the onset of pain in the context of a non-traumatic event experienced greater improvements in depression than patients who had experienced a traumatic onset of pain. Consequently, this study indicates that onset of pain may influence at least some aspects of response to a multidisciplinary, cognitive-behavioural pain management program, particularly maintenance of gains after treatment.

The current study also added to the existing body of literature indicating that cognitive variables, such as self-efficacy, catastrophising, and fear-avoidance beliefs, are important predictors of treatment outcome. Consistent with previous studies highlighting the potential importance of posttraumatic stress symptoms in chronic pain, these types of symptoms were also significant predictors of treatment outcome. Importantly, the association between the cognitive process variables and onset group was not significant, indicating that the process variables are important across all types of onset of pain.

9. DISCUSSION

Since the advent of the biopsychosocial model and the development of cognitive-behavioural theories of chronic pain a substantial body of literature has accumulated highlighting the role of behavioural, cognitive, affective, social, and environmental variables in the chronic pain experience (Gatchel, Polatin & Mayer, 1995; Peters, Vlaeyen & Weber, 2005; Johnston, Jimmieson, Souvlis & Jull, 2007; Leeuw, Goossens, Linton, Crombez, Boersma & Vlaeyen, 2007). This research has led to the dissemination of effective cognitive-behavioural treatment approaches aimed at improving physical function and reducing distress in people suffering from chronic pain (Morley, Eccleston & Williams, 1999; van Tulder, Ostelo, Vlaeyen, Linton, Morley & Assendelft, 2000; Guzman, Esmail, Karjalainen, Malmivaara, Irvin & Bombardier, 2001; van Tulder, Ostelo, Vlaeyen, Linton, Morley & Assendelft, 2001). However, these approaches are not universally efficacious and research efforts have shifted towards improving treatment outcomes for larger proportions of patients. One approach recommended in the literature is identifying the characteristics of patients who do not tend to benefit from existing treatments, or who do not benefit to the same degree as other groups of patients (Turk, 2005). Alternatively, research aimed at identifying variables which act as moderators and mediators of treatment outcome has also been recommended to achieve a better match between patient characteristics and specific treatments or treatment components (Vlaeyen & Morley, 2005).

The research studies conducted for this thesis have focused on some of these issues by investigating the impact of the nature of the onset of pain and posttraumatic stress symptoms on adjustment to chronic pain and treatment outcome. This was based on existing studies indicating that onset of pain following any specific event (e.g. accident,

injury, illness, or surgery) is often associated with poorer adjustment to chronic pain (i.e. higher levels of pain severity, pain-related disability, and affective distress) compared with insidious or spontaneous onset of pain (e.g. Greenfield, Fitzcharles & Esdaile, 1992; Himmelstein, Feuerstein, Stanek, Koyamatsu, Pransky, Morgan & Anderson, 1995; Geisser, Roth, Bachman & Eckert, 1996; Turk, Okifuji, Starz & Sinclair, 1996), and a growing body of research pointing to elevated rates of PTSD in chronic pain samples (e.g. Aghabeigi, Feinmann & Harris, 1992; Sherman, Carlson, Wilson, Okeson & McCubbin, 2005; Demyttenaere, Bruffaerts, Lee, Posada-Villa, Kovess, Angermeyer, Levinson, de Girolamo, Nakane, Mneimneh, Lara, de Graaf, Scott, Gureje, Stein, Haro, Bromet, Kessler, Alonso & Von Korff, 2007).

At the outset, a review of the pertinent literature highlighted the need to validate a self-report measure of posttraumatic stress symptoms in a chronic pain patient sample, and this was the primary aim of the first study. The second study used a cross-sectional design to investigate the impact of type of onset of pain on pain severity and pain-related adjustment. The third study employed a prospective design to test the hypothesis that type of onset of pain, including the presence of posttraumatic stress symptoms, could influence the outcome of a cognitive-behavioural pain management program. In the following sections, the key findings of these studies are reviewed. The discussion will then outline the main limitations and strengths of these studies, before shifting to the principal theoretical, clinical, and research implications.

9.1. Main findings

9.1.1. Study One

The aim of the first study was to investigate the psychometric properties of a self-report measure of posttraumatic stress symptoms in a large sample of chronic pain

patients presenting for treatment at a tertiary referral pain management centre. A widely-used measure, the PCL (Weathers et al., 1993), was modified to prompt patients to respond to items with reference to the event associated with the onset of their pain, or the period during which their pain began. The results of this study provided preliminary support for the suitability of the PCL as a self-report measure of PTSD symptoms in chronic pain patients. Participants were able to complete the PCL with reference to the onset of their pain and the PCL exhibited good psychometric properties in this patient group. Exploratory factor analyses identified a two-factor solution similar to others reported in previous factor analytic studies of PTSD symptomatology (Buckley, Blanchard & Hickling, 1998; Taylor, Kuch, Koch, Crockett & Passey, 1998; Asmundson, Wright, McCreary & Pedlar, 2003), providing support for the construct validity of the PCL in a chronic pain setting.

However, the study also highlighted a number of issues with the use of self-report measures of posttraumatic stress symptoms in chronic pain patient samples. PCL items enquiring about symptoms which are a common aspect of the chronic pain experience (e.g. irritability, sleep problems, difficulty concentrating, and loss of interest in previously enjoyed activities) were endorsed by a large proportion of the sample, and mean scores on the subscales measuring these symptoms were higher than those reported in PCL studies of some trauma populations (e.g. Andrykowski, Cordova, Studts & Miller, 1998; Cordova, Studts, Hann, Jacobsen & Andrykowski, 2000). Application of diagnostic cut-off scores and an algorithm recommended for the PCL in other trauma groups suggested that a significant proportion of the sample (up to 36%) could have potentially been diagnosed with PTSD. While this is consistent with reports of high levels of PTSD symptoms in chronic pain patient groups presenting for treatment following a traumatic event (e.g. MVA; Hickling & Blanchard, 1992), this proportion is much higher than the

rates typically reported in chronic pain clinic settings (Muse, 1985; Aghabeigi et al., 1992; Sherman et al., 2005).

9.1.2. Study Two

The second study in this thesis examined the diagnostic utility of the PCL by comparing PCL responses to information about onset of pain and posttraumatic stress symptoms collected from the participants' medical files. This revealed a marked discrepancy between the large proportion of the sample identified as potentially meeting diagnosis for PTSD on the basis of the diagnostic algorithm and/or cut-off scores (i.e. up to 31%) and the number of patients who had reported PTSD symptoms at assessment according to the file notes (i.e. 7%). Furthermore, once the event associated with the onset of pain was taken into account (as the DSM-IV criteria for PTSD requires) only 18% of the sample could have satisfied diagnostic criteria because that was the proportion of the sample which was identified as having experienced a potentially traumatic onset of pain.

The primary aim of the second study was to investigate the impact of onset of pain on pain severity and pain-related adjustment. Comparisons between patients who had experienced different types of onset of pain revealed few significant differences between them. That is, analyses comparing Accident, Specific Incident, and Insidious/Spontaneous onset groups and Traumatic, Non-traumatic, and Insidious/Spontaneous onset groups revealed no significant differences between the groups on measures of pain severity, pain-related disability, and symptoms of affective distress after adjustment for age, pain duration, and compensation status.

These results were not consistent with previous studies; however, close examination of these studies suggested that it might not be appropriate to generalise

findings from their specific diagnostic groups (e.g. fibromyalgia) with certain demographic characteristics (e.g. predominantly female samples). Methodological considerations (see Section 9.2) and other differences between these studies and Study 2 were also identified as potentially contributing to the different outcomes (e.g. different proportions of participants involved in a compensation claim or legal case).

Despite the lack of significant findings between onset groups, the results of Study 2 were consistent with other areas of chronic pain research. Participants involved in a compensation claim or legal case reported significantly higher levels of pain severity, pain-related disability and symptoms of stress, compared with participants who were not involved in a claim. This is consistent with other studies which have indicated that involvement in a compensation claim is a significant predictor of poor outcome following injury, surgery, and in studies of individuals with chronic pain (e.g. Turk & Okifuji, 1996; Harris, Mulford, Solomon, van Gelder & Young, 2005; Gabbe, Cameron, Williamson, Edwards, Graves & Richardson, 2007). The finding that longer pain duration was associated with higher levels of pain-related disability has also been reported previously (e.g. Boersma & Linton, 2005; Dunn & Croft, 2006). Finally, consistent with a large body of previous literature (e.g. Crombez, Vlaeyen, Heuts & Lysens, 1999; Asghari & Nicholas, 2001; Severeijns, Vlaeyen, van den Hout & Weber, 2001), fear-avoidance beliefs, catastrophising, and self-efficacy were significant predictors of pain severity, pain-related disability, and depression.

9.1.3. Study Three

The final study of this thesis adopted a longitudinal approach to type of onset of pain and posttraumatic stress symptoms by investigating the impact of these variables on response to a multidisciplinary, cognitive-behavioural pain management program. Unlike

the previous study, this treatment outcome study revealed a number of differences between onset groups. Most notably, patients who had experienced an insidious or spontaneous onset of pain reported greater improvements in pain severity and maintained these improvements more effectively over a one month period than patients who had experienced pain in the context of an accident or other specific incident. There was also limited evidence that improvements in depression favoured patients who had experienced an insidious or spontaneous and non-traumatic onset of pain. Consistent with this, posttraumatic stress symptoms (as measured by the PCL) were a significant predictor of treatment outcome, with higher levels of symptoms being associated with smaller improvements in pain-related disability and distress. The cognitive variables discussed above (i.e. catastrophising, self-efficacy, and fear-avoidance) were also significant predictors of treatment outcome. These findings were all consistent with expectations and with previous research. Importantly, the association between posttraumatic stress symptoms and treatment response was consistent with evidence that the co-occurrence of PTSD and chronic pain is associated with higher levels of disability and distress than chronic pain alone (e.g. Geisser et al., 1996; Sherman et al., 2000; Smith et al., 2002; Sherman et al., 2005). For the first time these findings were extended to treatment outcome, suggesting that chronic pain patients who have experienced a traumatic onset of pain and who are experiencing PTSD symptoms may not benefit from current or standard treatment approaches to the same degree as other patients.

9.2. Limitations of the current studies

As noted in the previous chapters, the three studies described above did have a number of methodological limitations which are important to consider.

Firstly, participants were allocated to onset of pain groups on the basis of information obtained from the participants' medical files. This constituted use of retrospective data as opposed to a contemporaneous source of information and could be seen as a limitation. Despite the experience of the clinical psychologists who coded this information, and the high degree of agreement between them, incorrect categorisation could have prevented the detection of greater differences between groups, particularly in comparisons of the Traumatic, Non-traumatic, and Insidious/Spontaneous groups. Similarly, instead of using validated, structured clinical interviews, PTSD symptom information was also obtained from medical files, which may not have been a reliable indicator of the true level of PTSD symptoms experienced by the Centre's patients.

Secondly, although this was a necessary part of examining the appropriateness of use of the PCL in a chronic pain clinic setting, asking patients who had experienced a spontaneous or insidious onset of pain to complete a measure of PTSD symptoms with reference to the onset of pain arguably lacked validity and could have influenced some of the results. It may be more appropriate in future research to limit the focus of studies to individuals who are experiencing pain related to a specific event.

Thirdly, although all three studies were conducted in a heterogeneous sample, and not a specific diagnostic group, all of the participants had been referred to a tertiary level pain management centre. Accordingly, the findings of these studies might only be able to be generalised to similar settings or to groups of patients who exhibit similar levels of pain-related dysfunction. In addition, although to date the results of studies conducted in treatment-seeking samples of chronic pain patients and PTSD patients have been consistent, it is possible that there are some differences in the relationships between the two conditions according to the primary presenting problem. Thus, these studies might

only be applicable to understanding posttraumatic stress symptoms as they present in chronic pain clinic settings.

A final limitation of these studies was the reliance on self-report of pain- and PTSD-related variables. Self-report measures could have been supplemented by actual measures of physical activity, particularly in the third study given that participants had also engaged in the process of upgrading functional activities despite their pain.

9.3. Strengths of the current studies

Despite the above limitations, the current series of studies also featured a number of important strengths.

Firstly, unlike many previous studies, all three of the current studies linked posttraumatic stress symptoms endorsed on a self-report measure to the onset of pain. Secondly, these studies constituted the first systematic, detailed examination of the psychometric properties of a self-report measure of PTSD symptoms in samples of chronic pain patients. Related to this, the issue of symptom overlap was also examined in detail for the first time.

The parts of the studies that investigated onset of pain were conducted in a heterogeneous sample, not one diagnostic group, and controlled for a wider range of pertinent clinical and demographic variables compared to previous studies in this area. This should enhance the validity of the findings reported. The inclusion of a longitudinal design was particularly important because it revealed group differences that had not been evident in the cross-sectional comparisons made in Study 2.

Finally, selection of the MMRM approach to analysing the data in the third study meant that all time points could be considered, a wide range of variables accounted for,

and a number of different questions could be posed using real-life clinical data. This would not have been possible using more traditional statistical approaches.

9.4. Theoretical implications

Taken together, the current studies indicated that onset of pain may influence some aspects of response to a cognitive-behavioural pain management program, but that overall, other variables, particularly cognitive variables, are probably more important influences on pain and pain-related dysfunction. The different results revealed by the longitudinal approach supports the view that the relationships between pain and the full range of potentially important variables might vary over time (Von Korff et al., 2002), and accordingly, onset of pain may only play an important role in the early stages following injury.

This is probably particularly relevant when the injury is sustained in the context of a traumatic event that also leads to the development of PTSD symptoms. As research has shown, and as was confirmed in Study 3, the co-occurrence of pain and PTSD is associated with a more complex clinical picture, and onset of pain may be important only in the sense that it may lead to PTSD.

Having said this, the current studies did not support the view that PTSD symptoms are highly prevalent in chronic pain patient samples, although it is worth noting that the lower rates of symptoms reported by the patients in these studies might also have been due to a referral bias. That is, patients are referred to the centre because pain is the primary problem and rates of PTSD in individuals with chronic pain probably differ in different treatment settings. Consistent with this, rates in the general population appear to be approximately 7-10%, a slightly wider range has been reported in chronic pain clinic

studies (6-12%), and the highest rates have been reported in samples of patients who have all experienced a potentially traumatic event (13-80%).

The current studies also have some interesting implications for current conceptualisations of PTSD. In particular, the recurring question of symptom overlap, and the fact that the PCL Avoidance and Arousal subscales were the two subscales that exhibited significant relationships with other variables, points towards the identification of nonspecific aspects of PTSD as an anxiety disorder. As Simms et al. (2002) have argued, irritability, impaired concentration, restricted affect, and disturbed sleep are basically symptoms of general distress and several studies have provided support for the four-factor model of PTSD symptoms they originally identified, which includes a "Dysphoria" factor (Baschnagel et al., 2005; McWilliams, Cox & Asmundson, 2005; Elklit & Shevlin, 2007; Palmieri et al., 2007). As all of these investigators have noted, a dysphoria factor associates the symptom structure of PTSD to models of depression and anxiety (e.g. Clark & Watson, 1991; Brown, Chorpita & Barlow, 1998). These models propose that the anxiety and mood disorders all share symptoms of general distress and negative affectivity (i.e. depressed or anxious mood, sleep disturbance, irritability, and impaired concentration). In addition to this nonspecific component, each disorder is characterised by specific symptoms that distinguish it from other disorders. In the case of PTSD, reexperiencing symptoms would constitute this specific component (Simms et al., 2002; Palmieri et al., 2007).

This conceptualisation has been used to explain comorbidity and overlap between the anxiety and mood disorders (Simms et al., 2002), and may be especially important for understanding both the relationship between chronic pain and PTSD and the performance of the PCL in chronic pain settings. For example, in a population characterised by high levels of anxiety and mood disorders (such as chronic pain), the symptoms that load onto

the dysphoria factor may be more of a reflection of this nonspecific component of general distress rather than specific symptoms of PTSD (i.e. hyperarousal). This approach to understanding posttraumatic stress symptoms in chronic pain patients is consistent with the literature on the potential role of anxiety sensitivity reviewed in Chapter 4, and has important implications for the interpretation of self-report measures of PTSD symptoms when used in chronic pain samples.

From this perspective, studying PTSD in people with chronic pain can be seen as an opportunity to improve our understanding of the relationship between PTSD and other disorders. The third study indicated that there may be an important interplay between the two conditions, as the theoretical models reviewed in Chapter 4 suggest. While this study did not elucidate the specific mechanisms involved, it did provide some perspective on the relative roles of both PTSD symptoms and cognitive variables in adjustment to persisting pain and treatment response.

9.5. Clinical implications

The issue of symptom overlap has implications for interpreting the PCL scores of chronic pain patients and for use of the PCL as a screening measure for PTSD in chronic pain treatment settings. Specifically, the PCL might be most useful as a screening tool given to patients who have experienced a sudden onset of pain or who have a clear history of trauma, but it might not be that useful to include as a standard part of a battery of questionnaires. The findings reported here also indicate that interpretation of the arousal and avoidance subscales should be made with a degree of caution. Application of the diagnostic algorithm and cut-off scores previously recommended should probably also be avoided until more appropriate guidelines are developed using structured clinical interview measures of PTSD diagnostic status in samples of heterogeneous chronic pain

patients (given that the only study to address this to date was conducted in an orofacial pain clinic; Sherman et al., 2005).

The current studies support previous recommendations to assess for PTSD in chronic pain patients, even if only on the basis that many of the events associated with the onset of pain could potentially be experienced as traumatic.

The repeated importance of cognitive variables in the current studies also supports current treatment approaches in their focus on addressing beliefs about pain, teaching patients to challenge catastrophising-type responses to pain, and fostering increased self-efficacy. The current studies underscore the importance of maintaining this focus and continuing to develop an understanding of how these variables influence treatment outcome.

9.6. Implications for future research

The current series of studies highlights important avenues for future research. Further validation of the PCL in chronic pain settings is required, particularly to determine if symptom overlap is as significant an issue as some of the current findings seemed to indicate. This could be achieved by use of structured clinical interviews and assessing the correlation between individual PCL items and specific questions included in gold-standard interviews such as the CAPS. Structured assessment of the potentially traumatic nature of the event associated with the onset of pain and linking it to any PTSD symptoms reported should also be ensured in future studies. Further factor analytic studies could also be conducted to determine if the two-factor structure identified in Study 1 can be replicated in other samples of heterogeneous chronic pain patients. Alternatively, confirmatory factor analysis could be employed to determine if the four-factor model including the dysphoria factor is valid in chronic pain samples.

Further investigation of the role of onset of pain is warranted despite the few differences observed between groups in these studies. However, the focus of future research on onset of pain should be on understanding the interaction between onset and early reactions to injury, particularly in the context of a traumatic event. This will not only improve our understanding of the development of chronic pain, but also shed light on the mechanisms underlying the development of comorbid pain and PTSD. Ideally this research should include prospective studies that capture patients as soon as possible following the onset of pain so they can be tracked longitudinally, similarly to some recent studies (Martin et al., 2007; Peters, Sommer, de Rijke, Kessels, Heineman, Patijn, Marcus, Vlaeyen & van Kleef, 2007). This would overcome many of the problems of retrospective data collection common to most existing studies in this area.

Finally, controlled investigations of treatment outcome in patients who present with both chronic pain and PTSD (even if below the diagnostic threshold) are urgently required. Although the third study was the first to demonstrate that posttraumatic stress symptoms as measured by the PCL are associated with smaller improvements in core outcome variables, it is not possible to determine what proportion of the sample actually were experiencing clinically significant PTSD symptoms or whether a different intervention would have produced different results.

9.7. Conclusion

Although many questions about the impact of type of onset of pain and posttraumatic stress symptoms remain, the current studies have highlighted a number of key methodological, theoretical, and clinical issues which warrant further investigation. Pursuing the avenues for research identified above should ensure that current treatments

for chronic pain are modified, or new treatments developed, in order to address the needs of individuals who present with both chronic pain and PTSD more effectively.

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