Caught in a Jam:
The preserving impact of psychological trauma on poor adjustment to and management of chronic pain.

Belinda A. Barker

A thesis submitted in partial fulfillment for the degree of Doctor of Philosophy of The Australian National University

January 2006

School of Psychology
The Australian National University
Canberra A.C.T. Australia
Declaration

I declare that this thesis reports my original work, that no part has been previously accepted and presented for an award of any degree or diploma from any university and that, to the best of my knowledge, no material previously published or written by any other person is included, except where due acknowledgement is given.

Belinda A. Barker
DEDICATION

This thesis is dedicated to David Ronald Barker (1948 – 2001), who appreciated curiosity, learning and adventures.
ACKNOWLEDGEMENTS

I would like to thank the following people:

Prof. Don Byrne for his steadiness as a supervisor. Without his breadth of knowledge, unflappable patience and timely words of encouragement this thesis would not have been possible.

The Canberra Injury Management Centre as a valuable incubator of thought that provided a stimulating environment, intellectually and professionally. The “same room, different windows” perspective of working amongst a dedicated multi-disciplinary team was invaluable. Thank you to Dr Lorimer Moseley, Dr Garth Eaton, Jac Cousin, Kathy Conroy, Jo Stillwell, Vicki Coghlan, Jeff Parsons, David Lester & Tony Robb for ideas, encouragement and support. Katrina Guy deserves extra special thanks for her dogged persistence following up potential participants.

The School of Psychology technical staff – Riccardo, Alan and Mary – and Darren Lipnicki for their valuable knowledge and effort in obtaining, building and setting up laboratory equipment, teaching me about the software and ironing out problems with it.

The individuals who participated in the research for their kind donation of time and willingness to share their personal experiences. I hope I have done justice to their contributions so that others may benefit from the knowledge gained.

My friends and family, especially Mum, Nenie, Mark, Matt, Rhi, Dee, Pete, Jo, Matt, Amaly, Cristian, Kylie & Shawn for giving me opportunities to talk and think about something else. A special mention goes to Ro for the “news on tues” emails, which brought a smile on tedious days and a sense of comfort that someone else out there knew what it was all about; to Dee for her much appreciated assistance and ability to think that helping was “fun”; and to Pete for his backstage efforts before the show.

Ian, a wonderful husband, partner and friend. I cannot thank you enough for your unconditional love and tireless support over the years. I think we have now filled the Life Event Checklist. With due reference to Frank Lloyd Wright, now the necessity is done, let’s live the luxuries!

BB.
ABSTRACT

Very little is known about the mechanisms underlying the presentation of comorbid chronic pain and PTSD, not least because prior research has been relatively atheoretical, ad hoc and subject to methodological limitations. Guided by recent theoretical models (i.e. Sharp & Harvey, 2001; Asmundson et al., 2002), the current program of research explored the nature of the relationship over five studies.

In Study 1, a sample of 120 patients with heterogenous chronic pain complaints was classified into two groups, according to scores on the Posttraumatic Diagnostic Scale (PDS). The aim was to assess whether these groups (PTSD: \( n = 57 \); no PTSD: \( n = 63 \)) differed in terms of anxiety-related variables, pain-specific coping strategies and indicators of adjustment. The groups were similar in age, gender and prior number of traumatic events. Results indicated a non-significant trend toward higher pain severity in the PTSD group compared to the No PTSD group. The PTSD group reported higher anxiety sensitivity, pain anxiety symptoms and maladaptive coping strategies. They were less well adjusted to pain, demonstrating more depression, pain-related disability and utilisation of health care providers and medication than the No PTSD group. They were more pessimistic about their prognosis, less likely to be working and more likely to be receiving compensation than their non-traumatised counterparts. A significant but small proportion of variance in pain severity (20%) and greater proportions of pain-related disability (52%) and depression (65%) were explained by the self-report measures using multiple regression analyses.

Improving on the methodology of Study 1, Study 2 more clearly differentiated PTSD diagnosis, using both the PDS and PTSD-module of the Structured Clinical Interview for DSM-IV (SCID) to assess for current injury-related PTSD amongst 29 chronic pain patients (PTSD: \( n = 14 \); No PTSD: \( n = 15 \)). Data obtained from self-report measures broadly replicated Study 1 regarding the effects of PTSD on pain severity, distress, disability, compensation status and pain-related anxiety. The PTSD group also demonstrated higher fear of movement/reinjury. PTSD severity was moderately correlated with resting skin conductance but not other physiological measures at baseline or during imaginal exposure to feared movements. Pain-related anxiety and fear of movement/reinjury was negatively correlated with heart rate and positively correlated with respiration during imaginal exposure. PTSD severity and pain ratings
were not significantly related to physiological arousal. Regression analyses indicated that PTSD severity only significantly contributed to the prediction of distress, in contrast to fear of movement/reinjury, which was a better predictor of pain severity, pain-related disability and distress.

The next two studies involved an experimental manipulation to test the prediction that PTSD directly exacerbates pain perception in chronic pain patients, under controlled conditions of movement (Study 3) or regarding global pain threshold (Study 4). In both cases, assessment was performed under standard conditions and repeated after exposure to individualised trauma-related cues. During initial assessment in Study 3, the PTSD group demonstrated less flexibility than the No PTSD group for two of ten specific movements: cervical and lumbar flexion during forward bend. Range of movement (ROM) was significantly correlated with pain severity, PTSD severity, pain-related anxiety and fear of movement/reinjury. When re-assessed post-exposure, the PTSD group demonstrated a significantly greater percentage change in ROM from initial measurement than the No PTSD group. Greater post-exposure restriction was significantly correlated with PTSD severity, negative affective response to exposure, pain-related anxiety and fear of movement/reinjury. No group x time interactions were evident for measures of pain, affect, tension or physiological reactivity.

Global pain threshold was assessed via the application of a thermode in Study 4. Contrary to hypotheses, the two groups (PTSD: \( n = 11 \); no PTSD: \( n = 9 \)) demonstrated equivalent thermal pain thresholds (TPT), under standard conditions and post-exposure. This result suggested that PTSD did not affect pain threshold either in its capacity as a general disorder or under circumstances of direct symptom activation. On the other hand, ratings of usual pain and negative affect increased during post-exposure TPT, especially in the group with PTSD, however, these changes did not predict changes in TPT.

Study 5 used a vignette-style questionnaire to assess the public and health professionals’ (GPs, psychologists, physiotherapists and chiropractors) ability to identify PTSD and chronic pain in an individual injured in a motor vehicle accident. Second, it assessed beliefs about interventions and outcomes regarding chronic pain and chronic pain with comorbid PTSD. Results showed that comorbidity significantly affected the level and accuracy of problem detection, as well as choices about treatment.
and outcomes. Several discrepancies emerged with respect to problem identification and treatment, not only between the public and professionals (e.g., physical injury versus psychological/pain formulation, endorsement of psychological treatment and recommended medications), but also between professionals and evidence-based clinical guidelines (e.g., in endorsement of passive physical treatments, counselling versus CBT).

The theoretical and clinical implications of the results and the need for development and refinement of our understanding of these comorbid conditions through future research are discussed.
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GLOSSARY FOR ABBREVIATIONS OF PSYCHOLOGICAL MEASURES

AIS  Abbreviated Injury Scale
ASI  Anxiety Sensitivity Index
BDI  Beck Depression Inventory
BSPAS  Burn Specific Pain Anxiety Scale
CAPS  Clinician-Administered PTSD Scale
CPCI  Chronic Pain Coping Inventory
CSQ  Coping Strategies Questionnaire
DASS  Depression Anxiety Stress Scales
FABQ  Fear-Avoidance Beliefs Questionnaire
FPQ-III  Fear of Pain Questionnaire III
ISS  Injury Severity Score
MMPI  Minnesota Multiphasic Personality Inventory
MPQ  McGill Pain Questionnaire
NRS  Numerical Rating Scale
PASS  Pain Anxiety Symptoms Scale
PCPT  Posttraumatic Chronic Pain Test
PDI  Pain Disability Index
PDS  Posttraumatic Stress Diagnostic Scale
PHODA  Photograph Series of Daily Activities ®
PSI  Pain Severity Index
PSSS  (Modified) Posttraumatic Stress Symptom Scale
PTSD-I  Posttraumatic Stress Disorder Interview
PVAQ  Pain Vigilance and Awareness Questionnaire
ROM  Range of movement
SCID  Structured Clinical Interview for DSM-III-R / DSM-IV
SDS  Zung Self-rating Depression Scale
STAI  Spielberger’s State Trait Anxiety Inventory
TPT  Thermal Pain Threshold
TSK  Tampa Scale for Kinesiophobia
THQ  Trauma History Questionnaire
VAS  Visual Analogue Scale
WHYMPI  West Haven-Yale Multidimensional Pain Inventory
CHAPTER 1

GENERAL INTRODUCTION AND OVERVIEW

The advent of the biopsychosocial approach to health problems catapulted psychology squarely into areas previously the domain of medical or biological faculties. Consequently, our understanding of chronic pain problems has expanded to consider the role of psychological and social factors relevant to the experience of pain, especially its development, course and treatment. In this way, the literature now carries a wealth of knowledge regarding the impact of various cognitive, affective and behavioural factors (Gatchel & Turk, 1996), as well as comorbid disorders (such as depression) on adjustment to and management of chronic pain.

In the past decade, interest has grown regarding the interrelationships between physical injury, chronic pain, trauma and Posttraumatic Stress Disorder (PTSD), however, pointed research in this area is in its relative infancy. Review of the empirical literature highlights an ad hoc approach to research to date, often atheoretical and or poorly controlled. To some extent, this is due to two lines of enquiry proceeding almost independently, one from the perspective of chronic pain and the other from the perspective of psychological trauma, both faltering without the valuable insight of the other perspective. The purpose of this thesis is thus to provide a review and synthesis of the existing literature, both empirical and theoretical, before embarking on an empirical investigation of the impact of PTSD on adjustment to and management of chronic pain. A greater knowledge of the relationship between PTSD and chronic pain may assist early identification, treatment and management of those at risk of poor adjustment.

Chapter 2 introduces the problem of chronic pain. It begins with a definition of chronic pain, then summarises prevalence rates and highlights the burgeoning costs of chronic pain for society. An overview of theoretical models of chronic pain is presented, illustrating the historical shift from a biomedical to a biopsychosocial approach and detailing recent fear-avoidance models. The supporting empirical evidence for fear-avoidance models is reviewed, revealing the potential impact of PTSD as a gap in our current understanding of persistent problems with pain.
The following chapter (Chapter 3) first provides background on PTSD, summarising issues regarding diagnosis, prevalence, comorbidity and risk factors for onset and maintenance. Obvious similarities between chronic pain problems and PTSD, including overlapping symptomatology, are discussed. The chapter then reviews empirical evidence of a relationship between PTSD and chronic pain. A range of cross-sectional studies indicates a high prevalence of prior trauma and or PTSD amongst chronic pain patients and, conversely, a high prevalence of chronic pain amongst individuals with PTSD or a prior history of trauma. Prior research also indicates that the presence of both conditions negatively impacts on presentation and outcomes, with reports of greater pain severity, affective distress, disability, dysfunctional coping and health care utilisation and poor response to current treatments amongst individuals who have chronic pain and PTSD. Further, although a range of peri-traumatic and post-traumatic factors has been investigated in the development and maintenance of PTSD, pain has attracted minimal attention in prospective post-injury studies. In particular, pain has failed to thrive in the shadow of objective physical injury, which has drawn much greater attention. However, it is argued that as a dynamic post-event factor, chronic pain has the potential to explain conflicting findings regarding the impact of injury on PTSD. The methodological limitations of prior research are discussed throughout, as too are the difficulties drawing firm conclusions or generalising results from the paucity of theoretically driven research.

Chapter 4 therefore turns to an examination of two theoretical models that have been proposed to explain the relationship between chronic pain and PTSD – the “mutual maintenance” model (Sharp & Harvey, 2001) and the “shared vulnerability” model (Asmundson, Coons, Taylor & Katz, 2002). The relative strengths of these models are discussed with reference to supporting empirical evidence. Drawing on and extending these models, a parsimonious theory based on the perception of current threat (Ehlers & Clark, 2000) is applied.

The broad aim of the empirical section of this thesis is to investigate the relationship between chronic pain and PTSD in the context of theoretical considerations discussed in Chapter 4. Specifically of interest is the impact of a current diagnosis of PTSD on adjustment to and management of chronic pain directly arising from a traumatic injury.
The first empirical study, as set out in Chapter 5, was designed to replicate and extend prior research by considering differences between pain patients with and without PTSD on a range of variables implicated in the onset and maintenance of chronic pain. In particular, the primary focus was whether the two groups differed on pre-injury variables and, more importantly, self-report measures of anxiety sensitivity, pain-related anxiety and coping strategies and whether differences in these influenced outcome variables such as pain severity, depression and pain-related disability. The meaning of the results in terms of mechanisms underlying the relationship between chronic pain and PTSD are discussed, with acknowledgement of the cross-sectional nature of the study.

The studies presented in subsequent chapters were designed to explore the differences and connections found in Study 1 more directly, using improved methodology to enhance the validity and reliability of results. Specifically, Study 2 included a formal diagnostic assessment of PTSD to classify individuals with chronic pain into groups (Chapter 6). Physiological correlates of PTSD and pain-related fear were considered in conjunction with self-report measures and responses to imaginal exposure to feared movements. The implication of these factors in terms of outcome variables, pain severity, affective distress and pain-related disability are discussed.

Chapter 7 describes two related studies with similar experimental designs, examining the possibility that PTSD directly exacerbates the perception of pain in chronic pain patients. In particular, the studies investigated, first, the general effect of PTSD on pain patients’ range of movement (Study 3) and global pain threshold (Study 4). The second aim of these studies was to test the effect of direct PTSD symptom activation, via employment of an individualised trauma narration on subsequent range of movement and assessment of pain threshold. Each study included consideration of changes in pain severity and affective and physiological reactivity during the tasks, as well as the relative contributions of PTSD diagnosis, PTSD severity, pain-related anxiety and fear of movement/reinjury. The results are discussed in terms of their implications for theoretical models.

Using research findings to inform development of effective treatments for comorbid pain and PTSD is vital, however, it is also important to investigate current public clinical understanding and the effect comorbidity has on current assessment and opinion about treatment delivery. Chapter 8 presents a vignette study designed to investigate
current knowledge and beliefs of both health professionals and the community about the dual presentation of chronic pain and PTSD. This study highlights that chronic pain patients with and without PTSD are perceived and treated differently in light of comorbidity. Differences in beliefs between the public and a range of health professionals, including general practitioners, psychologists, physiotherapists and chiropractors (amongst whom differences may also emerge) are also addressed. Data from this study suggest avenues for future research and has significant implications for education needs in the public and clinical sector.

Chapter 9 summarises the results of the present empirical investigation, with reference to the theoretical framework and directions for future research, clinical practice and educational needs. Greater understanding of the impact of PTSD on adjustment to and management of chronic pain has important clinical implications, particularly for the assessment and treatment of existing patients with chronic pain and the planning of early intervention strategies for patients with acute or sub-acute conditions to avert a chronic course. This may help to minimise individual suffering and disability, improve the allocation of health resources and reduce associated health care costs. Of course, the findings of the current studies also raise questions that require empirical investigation and refinement.
CHAPTER 2

UNDERSTANDING THE MAINTENANCE OF CHRONIC PAIN

2.1 Background

2.1.1 Definition of Chronic Pain

Pain is commonly understood as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey, 1979; Merskey & Bogduk, 1994). The International Association for the Study of Pain (IASP) adopted this definition in 1979 and it is widely accepted in research and clinical understanding of pain. Important in the IASP definition is the reference to the subjective quality of pain and the acknowledgement that organic pathology alone does not entirely explain an individual’s report of pain.

Although most pain is acute, bearing a close relationship to tissue damage and subsiding within four to six weeks, some people go on to develop chronic pain (Fordyce, 1995). In such cases, pain persists beyond normal healing periods and appears resistant to usual treatments. Within the medical literature, pain that has persisted for more than three months is classified as chronic pain (Merskey & Bogduk, 1994). This classification is adopted here due to its wide acceptance in the research literature, in preference for the criterion of six months necessary for psychiatric diagnosis of Pain Disorder, Chronic in DSM-IV TR (American Psychiatric Association, 2000).

2.1.2 Origins of Chronic Pain

Chronic pain can be attributed to a variety of general medical problems, most commonly musculoskeletal, neuropathic and malignant conditions. Work-related injury (Australian Bureau of Statistics, 1998) and motor vehicle accidents also pose common sources of injury leading to chronic pain (Mayou, 1999). Not all pain has an identifiable source, however. One study presented evidence that more than 85% of people complaining of back pain have non-specific complaints for which no structural lesion can be detected, that do not fit into accepted diagnostic classifications or that fluctuate in ways unexplained by biomedical mechanisms (Fordyce, 1995). Conversely, symptom-free people are often unaware that they have diagnostic abnormalities. For
example, MRI evidence suggests that a significant proportion of the population suffer from signs such as disc bulges without corresponding pain (Jensen, Brant-Zawadzki, Obuchowski, Malkasian, & Ross, 1994). Taken together, these findings indicate that the mere presence of a physical abnormality does not strongly predict reported pain levels. Indeed, different people respond differently to similar pathology, just as one individual can respond differently to the same pathology at different times (Evers, Kraaimaat, van Riel, & Bijlsma, 2001; Turk, 1996a). In summary, biomedical ‘precursors’ bear a weak relationship with reported pain, which in turn bears a weak relationship with distress and disability (Waddell, 1998).

### 2.1.3 The Burden of Chronic Pain

A relatively high proportion of people experience persistent pain at enormous financial cost and personal suffering. In a nationally representative study of Australians, Blyth et al. (2001) reported the prevalence rates for pain persisting on a daily basis for at least three months as 17.1% for males and 20% for females (Blyth et al., 2001). Similar rates have been identified in other studies (Harstall & Ospina, 2003; Mantyselka et al., 2001). Many individuals with chronic pain report psychological distress, including comorbid anxiety and depressive disorders (Becker et al., 1997; Harstall & Ospina, 2003; McWilliams, Cox, & Enns, 2003) and interference with their daily activities (Waddell, 1992). In turn, chronic pain is also associated with high health care utilisation and insurance claims (Nachemson, 1992; Spengler et al., 1986). In 1995, the National Health Survey revealed 24% of the population reported recent use of medication for pain relief (Australian Bureau of Statistics, 1999). Other Australian data indicates back pain is the greatest source of worker's compensation claims and lost work time (Ganora, 1986); see also (Australian Bureau of Statistics, 1998), with back problems accounting for approximately one quarter of all work-related injuries and conditions (Australian Bureau of Statistics, 1998; Comcare, 2001-2002). In any given year, between 10 and 15% of adults in the United States have some form of work disability due to back pain alone (American Psychiatric Association, 1994). The practical costs for Australia of severe pain, including lost productivity, welfare payments and compensation, run to over $30 billion per annum (Presley & Cousins, 1992). According to the 2003-2004 Annual Report for Comcare, payments to injured government employees for time off work and medical costs totalled $165.43 million for the financial year.
The degree of suffering and financial cost of chronic pain marks the importance of better understanding how it develops and why it persists. This chapter provides first a review of pain perception, followed by a review of current theoretical explanations of the processes by which pain becomes persistent. The chapter then narrows to a discussion of recent cognitive-behavioural models proposing fear and anxiety as determinants of chronic pain and empirical support for these. The chapter concludes with questions about the factors that might determine the initiation and maintenance of fear-avoidant mechanisms.

2.2 Models of Pain Perception

2.2.1 Early Theories of Pain

Early biomedical models of pain understood pain in terms of a linear mechanism of nociception, as a purely sensory-physiological response to harm or damage (Gatchel, 1999; Melzack & Wall, 1965, 1982; T. E. Rudy & Turk). A discrete physical stimulus was believed to cause pain, a sensation that was in turn registered by the brain. For example, the specificity theory of pain, dating back to Descartes in the mid 17th century, proposed that unique sensory systems existed for different sensations (such as pain, temperature, touch and pressure) each with their own structures and sensitive to different stimulation. The theory argued that pain was a direct and specialised transmission originating at a pain-specific receptor in injured tissue and passively traversing the spinal cord to specific receptor sites in the brain (Gatchel, 1999). Another example was the pattern theory of pain, in which Goldsneider suggested that the intensity and pattern of stimulation determined whether a stimulus was experienced as painful (Gatchel, 1999). As opposed to distinct systems, he believed pain sensation resulted from more extreme patterns of tactile stimulation (e.g., pressure), coded as painful by the nervous system based on their intensity, frequency and or duration.

2.2.2 Problems with Early Theories of Pain

Both theories advanced an inevitable and proportional relationship between tissue damage and pain sensation. The nociceptive system was presented as a relay channel, the resulting pain sensation driven by features of the noxious stimuli, rather than characteristics of the individual. In this regard, the traditional theories were unable to fully explain pain phenomena, such as the typical incongruence between patients' reported pain and disability, and underlying pathology (Gatchel, 1999; Melzack & Wall,
1965, 1982). None of the purely biomedical accounts could explain how individuals experience pain in the absence of (identifiable) pathology, the absence of symptoms in individuals with significant pathology, the variability between pain reports, distress and impairment, nor differential responses to treatment by individuals identified as having similar objective pathology (Turk, 1996a; Turk & Flor, 1999; Waddell, 1998; Waddell & Main, 1984). Indeed, traditional treatment approaches based on these early theories have been largely unsuccessful in treating many patients with chronic pain.

The traditional biomedical explanations of pain also created an unhelpful distinction between “organic” and “psychogenic” pain (G. L. Engel, 1959). Organic pain had a physical basis corresponding to objective pathology findings, whereas psychogenic pain was suspected when pain occurred without physical explanation, appeared excessive for underlying pathology, or persisted despite treatment. As a term, psychogenic pain is pejorative, based on a premise that pain under the latter circumstances is not “real” but rather exists “all in the patient’s head”, as a specious complaint arising as a function of personality, repressed emotion, or psychiatric disorder or motivated by secondary gain. As Rudy and Turk (1991) argued, there are several faulty assumptions inherent in the distinction. It assumes, first, that accurate, reliable and objective methods exist for measuring pain, detecting all organic pathology and assessing the correspondence between the two. Further, a dichotomous categorisation of pain that presumes pain is unimodal (either psychological or physical) overlooks the likelihood of individual difference factors in pain perception and adheres to a lop-sided view of causation ignoring the probability of dynamic interactions between psychological and physical processes.

These dubious assumptions were retained in the classification of Pain Disorder in the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-IV TR (American Psychiatric Association, 2000). The DSM-IV TR criteria are over-inclusive, requiring that psychological factors play an “important role” in the experience of pain for psychiatric diagnosis, and suggest that there can be a non-psychiatric sub-type of Pain Disorder – Due to a General Medical Condition – thereby ignoring the inherent perceptual and emotional aspects of pain. For these reasons, it has been argued elsewhere that the disorder has little diagnostic utility (Eifert, Lejuez, & Bouman, 1998; Fishbain, 1995; Nicholas & Wright, 2000; Sharp & Harvey, 2001; M. D. Sullivan, 2000). Indeed, the present thesis discards the DSM-IV definition of
chronic pain in favour of the more commonly accepted IASP definition discussed above.

2.2.3 The Gate Control Theory of Pain

To explain the inconsistent findings and address the shortcomings of the unidimensional biomedical models, Melzack and his colleagues (Melzack & Casey, 1968; Melzack & Wall, 1965, 1982) published the gate control theory of pain, proposing a physical means by which psychological factors and central nervous system (CNS) mechanisms — in addition to basic nociception — could influence the experience of pain. The proposed theory made an important contribution to the study of pain by precipitating a significant shift in the way pain was conceptualised. Melzack and Casey (1968) argued that pain is a complex perceptual process that entails the integration of sensory-discriminative information with information from cognitive-evaluative and motivational-affective systems. By contrast to the reductionist approach of previous models, the theory postulated that pain occurs as a dynamic synthesis of psychological and physiological processes rather than as a distinct physiological or psychogenic response.

The gate control theory introduced the notion of a neural ‘gating’ mechanism, existing in the dorsal horn of the spinal cord, to permit modulation of incoming sensory information, prior to perception, through a variety of excitatory and inhibitory influences and the effects of ascending and descending neural tracts of the CNS. Pain perception was thus viewed as dependent on bottom-up and top-down processing, which can effectively ‘open’ or ‘close’ the gate. For example, perception of pain is influenced by the amount of excitatory activity in Aβ and C fibres (similar to pattern theory), competition from activity in other peripheral (Aδ) fibres and the effects of inhibitory fibres descending from the brain (Turk & Flor, 1999). The gate control theory thereby paved the way for psychological and environmental factors, such as attention, learning, subjective interpretation, motivation, affect and context, to exert inhibitory and excitatory effects on pain perception.

The development of the gate control theory coincided with the general emergence of a biopsychosocial approach to health and illness (Caltabiano, Byrne, Martin, & Sarafino, 2002) and spawned a new generation of research. Although subsequent physiological research has challenged some of the details of the theory, demanding
alterations and revisions, in principle it continues to provide an overarching heuristic for understanding the perception of pain (Turk, 1996a). Subsequent models have continued to share the conceptualisation of pain as multidimensional. In shifting the focus from a narrow emphasis on the aetiology of pain, these models offer an integration of biological, psychological and environmental factors to explain the development, modulation and maintenance of pain and associated problems such as distress and disability.

2.3 Theoretical Framework for Understanding Persistent Pain

2.3.1 Behavioural Theories of Chronic Pain

To explain how chronic pain problems develop, behavioural approaches addressed how acute “pain behaviours”, observable reactions to pain, come under the maintaining influence of anticipatory anxiety and reinforcement contingencies. Fordyce first described the role of behavioural principles in the persistence of chronic pain problems, distinguishing these from etiological processes and the essentially unmeasurable subjective experience of pain (Fordyce, 1976, 1999; J. Lethem, P. D. Slade, J. D. G. Troup, & G. Bentley, 1983b; S.J. Linton, 1985; Philips, 1987). He suggested that learning and experience can influence pain behaviours to persist beyond the expected healing time for an injury, arguing that pain behaviour was subject to both classical and operant conditioning.

As with other aversive stimuli, Fordyce suggested that several responses automatically follow exposure to noxious sensation associated with tissue damage or harm. These responses include activation of the defensive system and thus muscle tension, sympathetic activation, fear and anxiety. Classical conditioning may result in external stimuli (e.g., specific activity, movement, or situations) eliciting the same response and, where the stimulus helps to predict pain, withdrawal, escape and avoidance will normally arise. Where avoidance continues, the learned anxiety response continues. Other pain behaviours may include the adoption of compensatory postures and gaits, utilising rest and medication and seeking assistance from health care providers, family or friends through specific requests or non-verbal expressions (e.g., grimacing).
Although these pain behaviours might be useful in managing acute pain — and even this is now being questioned (Fritz, George, & Delitto, 2001) — problems persist when operant conditioning comes into play (Sanders, 1996). Attention from family or health care providers, the avoidance of undesirable activity, such as work and domestic chores, and escape from aversive arousal can operate as positive and negative reinforcements, respectively, encouraging these behaviours. Of course, where reinforcement contingencies for pain behaviours exceed those for well-behaviours the former are likely to persist, despite reductions in sensory qualities and recovery from physical pathology. Not only is avoidance reinforcing of itself, the anxiety and fear that occurs if escape or avoidance is not possible perpetuates the cycle.

A large body of research has illustrated that pain behaviour, whether observed or self-reported, is associated with poor long-term outcomes (Jensen, Turner, Romano, & Strom, 1995; Vlaeyen et al., 1990). Treatments based on operant theory have also gained strong support (Morely, Eccleston, & Williams, 1999), however the underlying mechanism of change is not necessarily attributable to operant principles (Sharp, 2001; Turk, 1996b). Moreover, a purely operant approach does not explain the entire pain experience (Sharp, 2001). Similar to the traditional biomedical theories described above, behavioural theories viewed pain behaviour as secondary, a pain reaction, rather than an integrated component of pain perception (Turk, 1996a; Turk & Flor, 1999). In particular, this perspective failed to address the cognitive and affective aspects of pain and was unable to explain the maintenance of pain in the absence of actual reinforcement. Cognitive behavioural models arose to address these shortcomings. This has been reviewed in detail by others (Sharp, 2001; Turk, 1996b).

2.3.2 Cognitive-Behavioural Theories of Chronic Pain: The Role of Pain-related Fear-Avoidance

Cognitive-behavioural models illustrate pain as a multidimensional phenomenon extending beyond sensory input to comprise a complex integration of cognitive, affective, behavioural, physiological and environmental factors (Cioffi, 1991; Novy, Nelson, Francis, & Turk, 1995; Sharp, 2001; Turk, 1996a). While several models have been proposed, each differing in specific detail and focus, they have in common recognition that individuals’ processes of interpretation and appraisal influence their experience of and reactions to chronic pain. One area that has received significant attention from cognitive-behavioural theories is the contribution of pain-related fear to
the maintenance of chronic pain (Lethem et al., 1983b; S.J Linton, Melin, & Gotestam, 1985; Philips, 1987; Sharp, 2001; Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995a; Vlaeyen, Kole-Snijders, Rotteveel, Ruesink, & Heuts, 1995b; Vlaeyen & Linton, 2000).

In 1993, McCracken and Gross argued that the theoretical placement of fear responses to pain “antecedent to the coping responses makes intuitive sense because the former are probably more immediate responses to pain, whereas coping may require additional mediating responses” (McCracken & Gross, 1993). Of course, each may influence the other as part of a dynamic process occurring over time.

Like Fordyce, Philips articulated a dominant role for behavioural learning in maintaining chronic pain, however, her theory extended beyond his operant formulation by arguing that avoidance is influenced also by an individual’s thoughts and beliefs about the negative consequences of exposure (Philips, 1987). Her inclusion of a cognitive perspective helped to explain why avoidance may occur in the absence of sufficient reinforcement and, indeed, generalise to new situations. She argued that, individuals may acquire a fear of an activity that is expected to cause pain, based on their prior experience, current beliefs and perceived self-efficacy. Mere anticipation of exposure to the pain-related activity (conditioned stimulus) can thus generate anxiety resulting in autonomic arousal and avoidance (conditioned responses), both of which may maintain chronic pain problems even after the unconditioned stimulus (tissue damage) and response (pain, autonomic arousal) recede. Because repeated avoidance does not afford individuals a chance to discover whether their prediction was accurate as to whether pain would occur, its magnitude or their ability to cope with the consequences, a situation of confirmatory bias arises and thus a self-perpetuating cycle of distress and disability.

Other cognitive-behavioural models based on similar premises have also implicated specific pain-related fears in the development and maintenance of chronic pain. For example, one study described a fear-avoidance model based on the specific fear of exaggerated pain perception (Lethem et al., 1983b). This model argued that avoidance develops from a fear of motor activities, based on the (perhaps, originally accurate) expectation that the activity will cause pain. When pain does not occur, the avoidance is strongly reinforced and thus may extend beyond the resolution of nociceptive stimulation or associated responses of pain and sympathetic activation.
Vlaeyen and his colleagues (Vlaeyen et al., 1995a; Vlaeyen et al., 1995b; Vlaeyen & Linton, 2000) built on the above models in proposing a cognitive-behavioural model to explain how pain-related fear can result in chronic disability. In this model, the individual with acute pain may follow one of two behavioural pathways, resulting in either resolution of symptoms or a chronic pain problem. If individuals do not interpret pain as threatening or insurmountable, Vlaeyen et al., suggested, they will learn to adaptively confront movement and activities, and so recover gradually, as tissue damage heals and nociception subsides. By contrast, the second pathway begins with a tendency to negatively appraise pain and its consequences, coupled with a perceived inability to cope with pain. This exaggerated interpretative process, termed “catastrophising”, initiates and maintains a fear that certain movements will continue to cause pain and or re-injury. Pain-related fear then leads to pervasive escape and avoidant behaviours (reactive and anticipatory), which, over time, lead to disability, disuse and depression, compounding the pain experience and reinforcing fear. Functional disability occurs when avoidance prevents completion of goals and activities of daily living and the avoidance of expected pain is self-reinforcing. Physical deconditioning, a consequence of inactivity and disuse, is proposed to complete the cycle in three ways. It increases the likelihood of experiencing pain, fatigue and reduced mobility, as well as further reinforcing inaccurate predictions about pain and activity. Deconditioning also promotes stimulus generalisation as more and more activities become associated with pain or fear of pain. The loss of distraction, sense of pleasure and achievement normally obtained through activity is also likely to lead to depression and other negative affective states, which in turn may compound the experience of pain. In addition, the model argues that pain-related fear produces hypervigilance, whereby individuals are overly attentive to the possibility of threat and have problems disengaging attention from pain-related information, interfering with general task performance. Exposure to “threatening” situations also elicits psychophysiological reactivity. The second pathway thus spirals, maintaining or exacerbating fear and thus disability.

Recently, Sharp drew on the work of Salkovskis and his colleagues in the anxiety literature to argue for a reformulation of the cognitive-behavioural model of chronic pain. His reformulation hinges on elevating patients’ appraisals and interpretations to a position of primary importance, contrasting with the above models dominated heavily by behavioural constructs (such as avoidance). In his reformulation, Sharp emphasised
that chronic pain problems arise from and are maintained by patients' reactions to pain because of the idiosyncratic meaning they ascribe to pain. Meaning is a function of the individual's beliefs and interpretations, which have developed from prior learning experiences, culture, environmental contingencies (e.g., significant others, compensation and litigation) and iatrogenic influences (e.g., medication, investigations) (Sharp, 2001).

According to Sharp, the meaning derived from individuals' evaluative processes thus determines their affective responses and behaviour. When the meaning applied is maladaptive (e.g., “pain is a sign that something is seriously wrong with me”, “pain means I am forever disabled”), this results in maladaptive behaviour and contributes to autonomic arousal, which in turn feed cognitive biases and negative affect. The presence of reciprocal interactions leads to a self-perpetuating cycle of chronic pain. Reliance on avoidance and other “safety” behaviours, such as medication reliance, use of mobility and support aids and seeking reassurance, provides not only little opportunity to disconfirm maladaptive beliefs but may actually confirm them by making the feared outcome more likely to occur (Sharp, 2001). The absence or non-aggravation of pain when attributed to protective behaviours confirms to the individual that certain activities are dangerous and would have led to more damage if only they had not protected themselves. Likewise, bodily sensations associated with autonomic arousal when avoidance is not possible may also be misconstrued, confirming inaccurate beliefs about harm or damage. Heightened negative affect of itself further elevates the likelihood of cognitive distortions, selective attention and somatic focus. Thoughts about pain are also subject to metacognitive processes, in which attempts to suppress (i.e. ignore) or neutralise (by distraction) the thoughts, paradoxically increase their frequency and aversiveness. The overall consequence is a cycle driven by maladaptive beliefs leading to affective distress, maladaptive coping and disability.

Alternatively, Aldrich and her colleagues conceptualised the problem of chronic pain as a problem of perceiving a “chronic threat to self” (Aldrich, Eccleston, & Crombez, 2000). They argued that “the sensory, cognitive, affective and behavioural aspects of pain are not stable or static, but dynamic events” (p. 458) resulting from the interplay that occurs when an individual who perceives his or her pain as threatening proceeds to prioritise the threat and “solve” the pain. This vigilance to threat can be adaptive when it promotes problem solving, such as the initiation and maintenance of
appropriate defensive and recovery responses. However, in the case of chronic pain (pain that is insoluble or inescapable), Aldrich et al. (2000) proposed that vigilance can result in ruminative worry about pain and its actual or potential consequences and perseverative attempts to escape, both of which are maladaptive and distressing. They argued that the enduring perception of threat from persistent pain results in heightened bodily awareness and hypervigilant monitoring of symptoms, leaving the individual susceptible to attentional interference from intense pain (see also Eccleston & Crombez, 1999) and prone to high levels of symptom reporting. The combined effect of hypervigilance and repeatedly frustrated attempts to escape compounds the threat value of pain, creating a self-perpetuating cycle. The model shifts the focus from the persistence of pain itself onto the “chronification ... of vigilance to threat and of the perseveration of ineffective and misdirected problem-solving”.

Norton and Asmundson (2003) have also argued for an amendment to Vlaeyen and Linton’s (2000) fear-avoidance model, in which physiological symptoms and arousal are promoted to a more central role than in the earlier model. They proposed that physiological symptoms such as increased muscle tension and autonomic activity (e.g., blood flow, heart rate) might directly result in pain sensations, thereby “confirming” fears that activities are painful and so driving the proliferation of avoidance. Alternatively, they suggested that bodily sensations, such as muscle tension, associated with increased arousal might be misinterpreted as pain-related, with similar effects. In this way, physiological reactivity arguably facilitates anxiety, fear, catastrophising and performance deficits.

2.4 Empirical Support for Fear-Avoidance Models of Chronic Pain

Over the past decade, a growing body of evidence has lent support to cognitive-behavioural fear-avoidance models of the development and maintenance of chronic pain. The following section reviews findings of cognitive, affective, behavioural and physiological factors distinguishing individuals with chronic pain from three comparison groups: pain-free individuals, individuals who have acute pain that does not persist and individuals who have persistent pain but low distress and disability. The interdependence of these factors is also discussed.
2.4.1 Evidence and Effects of Cognitive-Affective Factors in the Maintenance of Chronic Pain Problems

A large body of evidence supports cognitive behavioural models of chronic pain, by demonstrating the significant impact of specific pain-related thoughts, beliefs and fears and cognitive biases on pain patients’ distress and disability, irrespective of pain severity and biomedical factors (Gatchel & Turk, 1996; Jensen, Turner, Romano, & Karoly, 1991a; Turk & Rudy, 1992; Vlaeyen & Linton, 2000; D. Williams, Robinson, & Geisser, 1994). For example, one study found that individuals’ expectations of increased pain, but not the pain they actually experienced, predicted poor behavioural performance (Crombez, Vlaeyen, Heuts, & Lysens, 1999). Predictions of high pain and low ratings of self-efficacy for managing despite pain also have been found to correlate with higher levels of depression (Rosenstiel & Keefe, 1983) and poor performance or avoidance of physical activities (Arnstein, Caudill, Mandle, Norris, & Beasley, 1999; Arntz & Peters, 1995; Dolce, Crocker, Molettiere, & Doleys, 1986; McCracken, Gross, Sorg, & Edmunds, 1993; T. Rudy, Kerns, & Turk, 1988). Further, cognitive factors influence an individual’s choice and use of coping strategies (Jensen et al., 1991a). Coping is generally defined as “a person’s cognitive and behavioural efforts to manage (reduce, minimise, master, or tolerate) the internal and external demands of the person-environment transaction that is appraised as taxing or exceeding the person’s resources” (Folkman, Lazarus, Gruen, & DeLongis, 1986). Jensen et al. (1991) found that patients’ beliefs about pain-related coping strategies were reliably associated with their self-reported use of these coping strategies.

2.4.1.1 Catastrophising

Individuals who catastrophise, believing their pain indicates more damage or is a sign of actual or imminent harm, are generally more avoidant and more disabled than those who do not catastrophise (Vlaeyen et al., 1995a; Vlaeyen et al., 1995b). A longitudinal study followed patients with acute back pain over the course of one year (Burton, Tillotson, Main, & Holliss, 1995). This study found that self-reported catastrophising, based on scores obtained on the Catastrophising sub-scale of the Coping Style Questionnaire (CSQ), was seven times more powerful than a host of clinical and historical factors in predicting the development of chronic pain. Individuals with back pain who sought treatment also reported greater catastrophising than those who did not seek treatment (Reitsma & Meijler, 1997). Another longitudinal study showed catastrophising predicted a worsening of various pain outcomes in patients with
rheumatoid arthritis (Evers, Kraaimaat, Geenen, & Bijlsma, 1998a; Keefe, Brown, Wallston, & Caldwell, 1989), including intensity ratings of pain (Keefe et al., 1989). In experimentally induced pain, catastrophising mediated pain tolerance and reported pain intensity, with lower tolerance and higher pain intensity recorded for patients prone to catastrophising (Geisser, Robinson, & Pikken, 1992; Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979; M. J. Sullivan, Bishop, & Pivik, 1995). These findings are interesting in light of other research that found no association between catastrophising and objective physical pathology (Peters, Vlaeyen, & Weber, 2005).

Catastrophising amongst pain patients correlates strongly with fear of pain, as measured by the Pain Anxiety Symptoms Scale (PASS) (McCracken & Gross, 1993) and is a better predictor of fear of movement, as measured by the Tampa Scale for Kinesiphobia (TSK), than pain severity or biomedical factors (Vlaeyen et al., 1995a; Vlaeyen et al., 1995b). The correlation between catastrophising and fear of pain is not exclusive to pain patients. In a 1998 study of students who were currently pain-free, individuals’ level of catastrophising was associated with self-reported fear of pain (Crombez, Eccleston, Baeyans, & Eelen, 1998a). Those individuals who were generally prone to catastrophic thinking were more likely to be fearful in anticipation of receiving an intense pain stimulus than students who were less likely to engage in catastrophic thinking. These finding suggest catastrophising is a risk factor for developing chronic pain, rather than a consequence of experiencing chronic pain.

Individuals who catastrophise, appraising pain and its meaning in an exaggerated, negative way, are also at risk of greater affective distress. Indeed, those patients who tend to engage in catastrophic interpretations of pain report higher levels of depression (Rosenstiel & Keefe, 1983) and higher ratings of pain aversiveness (Geisser, Robinson, Keefe, & Weiner, 1994; M. J. Sullivan et al., 1995; M. J. Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998). Geisser et al. (1994) controlled for the potential overlap between catastrophising and depression and found that catastrophising mediated the relationship between depression and ratings of the affective dimension of pain (i.e. unpleasantness). By contrast, catastrophising did not influence ratings of the sensory dimension of pain (i.e. pain intensity). M. J. Sullivan et al. (1998) found that catastrophising was also associated with disability amongst patients with soft tissue injuries, even after controlling for depression and anxiety.
2.4.1.2 Negative affect

Pain is undoubtedly a subjective experience that incorporates a significant affective component, cemented in the IASP definition. As Merskey (1986) stated, pain is "unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience" (Merskey, 1986)(p.217). Affective distress – collectively referring to a range of negative affective states including depression, anxiety, anger and frustration – is commonly reported by some, though not all, chronic pain patients (Price, 1999). Where it is reported, affective distress is often associated with poor adjustment (Fernandez & Turk, 1995; J. Lethem, P. D. Slade, J. D. Troup, & G. Bentley, 1983a; Philips, 1987).

2.4.1.2.1 Depression. More specifically, chronic pain is associated with elevated rates of depression compared to a normal population (Banks & Kerns, 1996; Comcare, 2001-2002; Tollison & Langley, 1995; von Korff & Simon, 1996). The depressive symptoms generally reported by these patients, however, appear different from the typical symptoms apparent in a pure, clinically depressed population. For patients with chronic pain, somatic or vegetative symptoms and affective distress dominate their presentation (due to the physical correlates of pain) rather than the negative self-evaluation seen in a depressed population (de C. Williams, 1998; de C. Williams & Richardson, 1993; Pincus & Morley, 2001; von Korff & Simon, 1996). Nonetheless, chronic pain patients who suffer from depression report higher pain intensity and lower activity levels compared to those who do not have significant mood disturbance (Haythornthwaite, Sieber, & Kerns, 1991; Holzberg, Robinson, Geisser, & Gremillion, 1996). The view that chronic pain represents masked depression is not, however, supported by empirical evidence and is perhaps only true for a small selection of patients. Depression is more likely a consequence rather than an antecedent of chronic pain (de C. Williams, 1998; Fishbain, Cutler, Rosomoff, & Rosomoff, 1997; Turk & Salovey, 1984).

Empirical evidence also suggests that depressed mood selectively influences the affective component of pain perception, but does not necessarily change the sensory quality of pain. An experimental study found that an odour distractor used to induce negative mood altered the perceived unpleasantness of pain, without any reported alteration in the perceived intensity of pain. That is, participants reported pain as more unpleasant after exposure to an unpleasant odour that evoked a negative mood state.
(Villemure & Bushnell, 2002). Similarly, in another study, a mood induction using written statements with positive or depressive valence was shown to influence pain tolerance, an affective dimension of pain (Zelman, Howland, Nichols, & Clelland, 1991). Individuals whose mood deflated after reading depressive statements demonstrated reduced pain tolerance, whereas pain tolerance increased when a positive mood was induced. Meanwhile, ratings of pain intensity, the sensory aspect of pain, were unchanged across trials.

2.4.1.2.2 Anger. Anger is often reported by patients with chronic pain (Kinder & Curtiss, 1988; Pilowsky & Spence, 1975; Schwartz, Slater, Biorchler, & Atkinson, 1991) and is associated with poorer outcomes (Burns, Wiegner, Derleth, Kiselica, & Pawl, 1997; Kerns, Rosenberg, & Jacob, 1994). State anger has accounted for significant variation in measures of pain intensity, frequency of pain behaviours and perceived life interference (Kerns et al., 1994). Others similarly found a significant relationship between pain severity and anger and hostility (Summers, Rapoff, Varghese, Porter, & Palmer, 1991). Following a review of the literature, Fernandez and Turk (1995) suggested anger and frustration may complicate chronic pain problems via elevated autonomic arousal, poor acceptance of rehabilitative treatment and decreased motivation to seek treatment or implement strategies.

2.4.1.2.3 Anxiety. With respect to the perception of pain itself, anxiety has been shown to increase reported pain (Ferguson & Ahles, 1998). Pain patients are also more generally anxious than the general population. In a study of 146 disabled workers with chronic pain, it was found almost 18% met DSM-IV criteria for a concurrent anxiety disorder (Asmundson & Taylor, 1996). Several researchers have found that patients with persistent pain were more likely than the general population to exhibit anxiety disorders not directly related to their pain, such as social anxiety (Philips & Jahanshahi, 1986; Vlaeyen et al., 1995a). Further, others have reported that 35% of the chronic pain population met DSM-III-R criteria for an Axis I anxiety disorder, compared to only 18% of the general population (McWilliams et al., 2003). This broad propensity for anxiety in chronic pain patients could be interpreted as evidence of stimulus generalisation or suggestive of an underlying factor common to both anxiety and chronic pain. A general cognitive bias in interpretation (e.g., catastrophising, anxiety sensitivity) and memory has been suggested to underlie the elevated risk of anxiety (discussed below), however, other possible factors remain to be investigated.
2.4.1.3 Pain-related anxiety, fear of pain and kinesiophobia

Compared to relevant comparison groups, chronic pain patients score higher on measures specifically assessing fear of pain, such as The Fear of Pain Questionnaire III (McNeil & Rainwater, 1998) and pain-related anxiety, such as the PASS (McCracken, Zayfert, & Gross, 1992). Pain-related anxiety is operationalised by the PASS as a specific negative emotional reaction to pain-eliciting stimuli, assessed across four dimensions: fearful appraisals of pain, cognitive-affective distress, physiological arousal and behavioural escape/avoidance. Pain patients also demonstrate fear of movement (kinesiophobia), based on a belief that activity will lead to an aggravation of pain or reinjury (TSK)(Kori et al., 1990) and hold pain-related fear-avoidant beliefs about work according to the Fear-Avoidance Beliefs Questionnaire (FABQ) (Waddell, Newton, Henderson, Somerville, & Main, 1993).

2.4.1.3.1 Links with pain severity. Patients with high fear of pain and or kinesiophobia tend to report greater pain severity (McCracken, Gross, Aikens, & Carnrike, 1996; Peters et al., 2005), make more inaccurate predictions of future pain (Crombez, Vervaet, Baeyens, Lysens, & Eelen, 1996; Crombez et al., 1999; McCracken et al., 1993; Murphy, Lindsay, & de C. Williams, 1997) and report more non-specific physical complaints (McCracken, Faber, & Janeck, 1998). Physiologically, pain-related fear is also associated with heightened autonomic reactivity to pain (Vlaeyen et al., 1999). Despite these connections to pain intensity and arousal, there is no association between objective physical pathology and fear of pain or fear of movement (Peters et al., 2005).

Experimental studies also highlight the role of fear in the experience of pain. One group used a cold-pressor task to investigate pain threshold and tolerance following a mood induction task (Meagher, Arnau, & Rhudy, 2001). They found that after watching slides designed to evoke fear and disgust individuals rated their pain as more intense and unpleasant on a visual analogue scale than when they watched neutral slides. By contrast, a positive mood induction in males evoked an opposite effect of higher pain thresholds, whereby pain was rated as less intense and unpleasant if preceded by erotic slides. In addition, individuals in the fear condition also experienced greater autonomic arousal and displayed reduced pain tolerance, an effect not present in
the other conditions. This suggests that both valence and arousal may be important features of fear (Rhudy & Meagher, 2000, 2001).

2.4.1.3.2 Links with dysfunctional coping. High levels of fear and anxiety are also associated with signs of dysfunctional pain-related coping (Asmundson, Norton, & Allerdings, 1997; McCracken & Gross, 1993). In turn, pain-related fear and anxiety constructs have been strongly linked to a wide range of factors indicative of poor adjustment to chronic pain. This is reviewed in detail elsewhere (Asmundson, Norton, & Norton, 1999; Vlaeyen et al., 1995a; Vlaeyen & Linton, 2000). Using the PASS, McCracken and Gross (1993) argued that the primary modality through which pain-related anxiety manifests determines the choice of coping responses. Compared to low scores, high scores on the cognitive anxiety subscale of the PASS were correlated with lower use of cognitive coping strategies (e.g., distraction) and a reduced sense of control over pain. Indicators of higher physiological arousal were associated, surprisingly, with self-reported superior coping, while pain behaviours were more evident in those who scored high on the avoidance subscale.

2.4.1.3.3 Links with avoidance. As with general fears, pain-related fear is strongly predictive of avoidant behaviour. Indeed, several researchers concluded that pain-related fear and anxiety combined with avoidant behaviour may be “more disabling than pain itself” (Crombez et al., 1999; Waddell et al., 1993).

Numerous studies have demonstrated under experimental conditions that patients with high levels of pain-related fear display poor performance on a number of physical tests. Individuals with high fear of pain or kinesiophobia have performed worse than individuals with low fear on a range of tasks, such as lifting 5kg (Crombez et al., 1999; Vlaeyen et al., 1995a) and completing isokinetic tests, such as the straight leg raise and knee or lumbar extension-flexion tests (Al-Obaidi, Nelson, Al-Awadhi, & Al-Shuwaie, 2000; Crombez et al., 1996; Crombez et al., 1999; Goubert, Francken, Crombez, Vansteenneven, & Lysens, 2002; McCracken et al., 1993; Vlaeyen et al., 1995b).

For example, in a study of 33 patients with chronic low back pain, Vlaeyen et al. (1995) investigated the relationship between kinesiophobia, pain, distress and behaviour. Participants were asked to stand and lift a 5.5kg bag with their dominant arm for as long as they could. Results indicated that patients with high kinesiophobia
demonstrated less tolerance of pain and exhibited more state anxiety and fear, even after the task was terminated, than patients with low kinesiophobia. In another study, kinesiophobia (TSK) and fear-avoidance beliefs about work (FABQ) were found to best predict individuals’ ability to complete back extension and flexion exercises, over measures of general affective distress (Negative Emotionality Scale) and pain intensity (VAS) (Crombez et al., 1999). This finding negates claims that poor performance measures are just a sign that something hurts rather than “avoidance”.

Similarly, where individuals’ actual maximal ability has been tested, task performance below this level can be interpreted with greater certainty as avoidance (Crombez, Vervaet, Lysens, Baeyens, & Eelen, 1998). Using a clustering procedure of kinesiophobia scores, Crombez et al. classified patients with chronic low back pain as either “avoiders” or “confronters”. Although the groups did not differ on their perceived control over pain, comparisons of their physical performance showed differences between the two groups, even after controlling for differences in pain status. Compared to confronters, avoiders performed below their maximal ability on a Cybex knee extension and flexion task, achieving less torque and more variance.

2.4.1.3.4 Links with disability. Pain-related fear and anxiety is also associated with greater self-reported disability and interference with activities of daily living (Asmundson, Norton et al., 1997; Crombez et al., 1998; Crombez et al., 1999; McCracken et al., 1996; McCracken et al., 1992; Peters et al., 2005; Vlaeyen et al., 1995b; Waddell et al., 1993). For example, Crombez et al. (1992) found scores on the PASS better predicted pain-related interference and disability, than did measures of sensory pain (McGill Pain Questionnaire, MPQ), trait anxiety (Spielberger’s State Trait Anxiety Inventory, STAI) and emotional distress (BDI). These results were confirmed by Waddell et al. (1993) who found that fear-avoidance beliefs about physical activities and work (FABQ) specifically accounted for 46% of variance in perceived disability and actual work loss associated with chronic low back pain. By contrast, biomedical measures of pain (e.g., anatomical pattern of pain, time pattern of pain, severity of pain) accounted for only 14% of the variance.

Other signs of disability found to be associated with pain-related fear include more work leave (S.J. Linton & Hallden, 1998a, 1998b; Waddell et al., 1993), greater pain-behaviour and more frequent help-seeking (McCracken et al., 1996). Prospective
studies in acute low back pain patients (Klenerman et al., 1995) and healthy people (S.J. Linton, 2000) have also provided support for the idea that pain-related fear may be an important precursor of pain disability. Indeed, pain-related fear seems a better predictor of disability than factors such as biomedical signs, pain severity and duration and negative affect (Crombez et al., 1999; Jensen, Romano, Turner, Good, & Wald, 1999; McCracken & Gross, 1993; McCracken et al., 1993; Vlaeyen et al., 1995b; Waddell et al., 1993).

2.4.1.4 Catastrophising, negative affect and fear: pain-specific or a general cognitive bias?

The evidence outlined above firmly anchors the role of cognitive-affective processes in pain experience, however the research suffers from the usual claims of response bias, due to the largely conscious, subjective, self-reported methods of data collection. More recently, there has been a surge of experimental research testing cognitive processing in chronic pain patients, using paradigms from cognitive psychology that have been now applied to study clinical disorders. The application of experimental paradigms enhances causal reasoning, which suggests cognitive biases may be involved in the onset and maintenance of chronic pain, and are not merely symptoms of pain.

2.4.1.4.1 Memory biases. Firm evidence exists for a memory bias in patients with chronic pain. In a general sense, patients with chronic pain show enhanced recall of sensory-pain descriptors over neutral and negative non-pain words, compared to pain-free controls (S. A. Pearce et al., 1990; Pincus, Pearce, & McClelland, 1995). Under circumstances where the task is self-referential, patients with pain, who are also currently depressed or distressed, show a bias toward recalling negative personal descriptors and health- and illness-related descriptors. If presented simultaneously, however, facilitated recall occurs only for stimuli related to health and illness but not for the negative personal descriptors. That is, the memory bias is for health and illness information (threat to physical self) rather than information about negative affect (threat to psychological self), even amongst patients with comorbid depression. A detailed review is presented elsewhere (Pincus & Morley, 2001). Furthermore, the bias is egocentric, disappearing when the information relates to someone else, even in a health or medical context (Pincus & Morley, 2001). Taken together, these two findings
suggest a memory bias toward information that the individual perceives as threatening to his or her physical self.

2.4.1.4.2 Interpretational biases. Pincus and Morley (2001) reviewed the experimental evidence for an interpretational bias in patients with chronic pain. Three main paradigms are typically used to examine interpretation bias: homophone tasks in which participants are asked to respond to words that sound identical but have two possible spellings (e.g., pane-pain); homonym tasks in which participants respond to a word with identical spelling but two meanings (e.g., terminal: airport vs disease); and word-stem completion tasks in which participants are given three letters and asked to complete the word (e.g., ten...: tennis vs tender). For each task it is of interest whether the individual generates or chooses a neutral or negatively charged word in the face of ambiguity.

Overall, the evidence suggests that patients with chronic pain tend to interpret ambiguous stimuli in terms of health and illness-related concepts and sensory aspects of pain (Pincus & Morley, 2001). The interpretative bias appears to be influenced by current experience of pain (Pincus & Morley, 2001; Pincus, Pearce, McClelland, Farley, & Vogel, 1994), however, the available evidence does not elucidate whether the bias is global (all negative information), generalised (for illness-related information), or specific (to pain). In contrast to clinical observations and findings from self-report studies, experimental research bears little support for the suggestion that the interpretation biases are driven by emotional status, such as depression and anxiety (Pincus et al., 1994; Pincus, Pearce, & Perrot, 1996). However, the experimental samples may not be representative of a clinical population, given the low rates of depression and anxiety reported therein (Pincus & Morley, 2001) in comparison to Banks and Kerns (1996).

2.4.1.4.3 The specific case of “Anxiety Sensitivity”. Anxiety sensitivity is a stable dispositional factor attracting recent attention for its potential to moderate maladaptive responses to chronic pain (e.g., fear of pain) and its links with poorer outcomes (Asmundson & Norton, 1995; Asmundson, Norton, & Veloso, 1999; Asmundson & Taylor, 1996; Zvolensky, Goodie, McNeil, Sperry, & Sorrell, 2001).
Anxiety sensitivity is defined as the fear of anxiety-related bodily sensations based on the belief that they are harmful (Reiss & McNally, 1985). In summary, it is a meta-construct — being anxious about being anxious — whereby the fear of negative social, physical or psychological consequences attributed to anxiety sensations amplifies the original anxiety response. In the context of chronic pain, it has been argued that high anxiety sensitive patients are at elevated risk of fearing pain for the affective and somatic arousal it produces and the perceived consequences of these sensations (Asmundson, 1999; Asmundson & Taylor, 1996).

Asmundson and Norton (1995) provided support for the role of anxiety sensitivity in adverse reactions to pain in 70 patients with unexplained back pain. While anxiety sensitivity, measured on the Anxiety Sensitivity Index (ASI), did not influence the level of pain severity reported by patients, patients prone to fearing anxiety-related body sensations responded more negatively to their pain. Even after controlling for pain severity, those who scored high on the ASI reported more pain-related cognitive anxiety and greater fear of harm due to pain and were more likely to experience negative affect, compared to patients with medium to low anxiety sensitivity.

Asmundson and Taylor (1996) tested the hypothesis that anxiety sensitivity increases the risk of developing fear of pain, using structural equation modeling on data obtained from 254 patients with non-specific musculo-skeletal chronic pain. Results indicated that anxiety sensitivity contributed to fear of pain, which, in turn, contributed to avoidant behaviour. Indeed, anxiety sensitivity accounted for more of the variance in pain-related fear (30%) than did pain severity (13%). Fear of pain subsequently predicted escape/avoidance (accounting for 68% of variance), whereas pain severity did not make a unique contribution to the prediction of avoidance.

Associations between ASI and pain-related fear and anxiety have been replicated in heterogeneous chronic pain (Zvolensky et al., 2001) and recurrent headache populations (Asmundson, Norton, & Veloso, 1999). Zvolensky et al. (2001), for example, assessed the effects of anxiety sensitivity on pain-related anxiety and fear of pain in 68 patients with various chronic pain complaints. First, fear of pain, and subscale scores for fear of medical, minor and severe pain assessed by the FPQ-III, were significantly predicted by anxiety sensitivity but not by self-reported depression and pain severity. Further analyses revealed that the physical concerns subscale of the ASI was a better predictor.
of pain-related fear dimensions than were the psychological and social concerns subscales, depression, pain severity and pain duration. The ASI also significantly predicted pain-related anxiety, as measured by the PASS. ASI psychological concerns contributed to total pain-related anxiety and — in conjunction with depression — cognitive symptoms and fearful appraisals, while ASI physical concerns contributed to escape and avoidance behaviours and physiological anxiety symptoms. Zvolensky et al. (2001) suggested their results "highlight the theoretical distinction between anxiety and fear states for pain-related concerns, such that greater psychological concerns are most apparent for anxiety states, and greater somatic reactivity is associated with fear (P. J. Lang, Levin, Miller, & Kozak, 1983; McNeil, Turk, & Reis, 1994). This is consistent with Rhudy and Meagher’s (2000, 2001) argument, as is discussed below.

For Asmundson et al.’s (1999) sample with headaches, higher scores for anxiety sensitivity corresponded with greater self-reported fear of pain, cognitive disruption due to pain, escape/avoidance behaviour and depression. These differences were found in the absence of any apparent differences in pain severity or reported changes in lifestyle attributed to pain. Again, anxiety sensitivity and cognitive anxiety symptoms accounted for a significant proportion of variance in fear of pain (39.8% and 12.7%, respectively) On the other hand, anxiety sensitivity was not related to sensory descriptions of or physiological reactivity to the pain experience. This finding is consistent with the construction of anxiety sensitivity, which suggests that it is not anxious arousal per se that is problematic, but rather that the problem is one of anticipating negative consequences arising from arousal. In other words, these individuals appear to experience similar levels of arousal in response to pain compared to low anxiety sensitive patients, however, they respond differently to it.

Similar effects for anxiety sensitivity have been recorded in the laboratory. In response to experimentally induced pain (Keogh & Mansoor, 2001), healthy women with high anxiety sensitivity provided stronger ratings of the sensory and affective aspects of pain, despite no differences in pain threshold or tolerance when compared to the low anxiety sensitivity group. Notably, the higher ratings occurred under instructions to avoid attending to pain, but not under instructions to focus on it. In the face of experimentally induced stress, individuals with elevated anxiety sensitivity also reported higher levels of pain during a cold pressor task (Schmidt & Cook, 1999).
Combined with the data on fear-avoidance discussed above, these findings suggest anxiety sensitivity as an underlying risk factor for poor outcomes for patients with chronic pain. One group explored the association between anxiety sensitivity and functional status in a chronic pain population (Plehn, Peterson, & Williams, 1998). Although anxiety sensitivity did not predict physical functioning, it accounted for a significant proportion of variance in vitality (10%), mental health functioning (25%) and social functioning (9%), whereby higher anxiety sensitivity scores were associated with lower functioning in these areas, as measured by the SF-36. Use of analgesic medication was also more likely amongst patients with high anxiety sensitivity (71%) compared to those with medium and low anxiety sensitivity (34% and 25%, respectively) (Asmundson & Norton, 1995; but cf. Asmundson et al., 1999).

In summary, the preceding review provides evidence that patients with chronic pain display biased information processing, however, this may not be specific to pain.

2.4.2 Evidence and Effects of an Attentional Bias and Hypervigilance

Attention refers to selecting for focus certain stimuli from a potential range of simultaneously available stimuli (Eysenck & Keane, 1993). In cognitive psychology, selective attention is measured in terms of facilitated responses to stimuli or disruption of ongoing responses to concurrent stimuli. In this way, an attentional bias exists where a facilitated or disrupted effect consistently occurs in the context of a particular stimulus.

Knowledge about attentional bias under circumstances of chronic pain (and what impact this may have) comes from two sources: studies of people with chronic pain and of pain-free people subjected to experimentally induced pain. Pain clearly demands attention (for a review see Crombez, Eccleston, Baeyens & Eelen, 1998a), which makes sense from an evolutionary perspective. At the very least, high levels of pain are more disruptive to performance than low to moderate levels of pain, even in the absence of affective distress (Eccleston, 1994, 1995a, 1995b; Grisart & Plaghki, 1999). However, task performance seems to be disrupted only when high intensity pain is coupled with a generally high awareness of bodily sensations (Eccleston, Crombez, Aldrich, & Stannard, 1997).
In addition, the experience of chronic pain is associated with an attentional bias toward pain-related stimuli, particularly sensory aspects of pain (Crombez, Hermans, & Andriaensen, 2000; J. Pearce & Morley, 1989; Snider, Asmundson, & Wiese, 2000). Experimental paradigms borrowed from cognitive psychology (e.g., the stroop and dot probe tasks) have illustrated that individuals with chronic affective states display ‘threat-related attentional biases’ consistent with their clinical disorder (Pincus & Morley, 2001; J. M. Williams, Mathews, & MacLeeod, 1996). For the reasons outlined above, it has been predicted that patients with chronic pain should show either task interference or speeded responses when presented with pain-related stimuli, depending on the nature of the task (Pincus & Morley, 2001). Unlike the clear findings in other emotional disorders, the results of such studies in chronic pain populations are mixed. In addition, one criticism of this research is the use of single descriptors to reflect what is a complex and abstract phenomenon (Pincus & Morley, 2001).

In the Stroop task individuals must name the print colour of words whilst ignoring their meaning. Delayed reactions are interpreted as reflecting increased interference due to an attentional bias to the meaning of the word. Pearce and Morley (1989) used the Stroop task to compare responses of chronic pain patients and controls to pain-specific words (threatening) and non-specific negative words. They concluded from their results that, compared to controls, pain patients selectively attended to words associated with pain and its consequences, but showed no bias toward words associated with negative affect. The finding was interpreted to mean that chronic pain patients had a “more salient cognitive representation of both sensory and affective components of pain” rather than a more general bias toward negative stimuli (Pearce & Morley, 1989, p.120).

Other studies have been unable to replicate this simple effect under different paradigms. Asmundson et al. (1997), for example, used the dot-probe task to present pairs of pain-related, injury-related and neutral words to chronic pain patients. On some trials a dot is presented after the word pairs in the same geographical location as one of the words and participants respond to presentation of the dot with a key press. An attentional bias is assumed if reaction times are quicker when the dot correlates with the location of a ‘meaningful’ (i.e. threatening) word rather than a neutral word. Asmundson et al. (1997) found that chronic pain patients who rated themselves as relatively unconcerned by anxiety (low anxiety sensitivity) diverted attention away from
pain-related words, an effect not evident amongst chronic pain patients with high anxiety sensitivity. Rather than supporting pain-specific hypervigilance, Asmundson et al. (1997) argued that the absence of an effect in anxious participants suggested a difference in coping style.

2.4.2.1 Impact of anxiety on attentional bias

It is not clear from these experimental studies whether attentional bias is a cause or a consequence of maladaptive responses to pain (Keogh, Ellery, Hunt, & Hannent, 2001). Attention toward pain-related stimuli may not be an inherent feature of pain but may instead be mediated by affective distress, such as anxiety (Asmundson, Kuperos, & Norton, 1997; Asmundson, Norton et al., 1997; Crombez et al., 1998a; Crombez et al., 2000; Keogh et al., 2001; Pincus, Fraser, & Pearce, 1998; Pincus & Morley, 2001; Snider et al., 2000) and depression (Pincus et al., 1998; Pincus, Pearce, McClelland, & Turner-Stokes, 1993). Indeed, hypervigilance to threat is a function of anxiety designed to facilitate the early detection of potentially threatening situations and thus promote escape or avoidance (Eysenck, 1997).

Crombez et al. (1998) added that if one interprets pain as threatening the pain is likely to be even more demanding of attention and therefore more distracting. By grabbing attention, pain could subsequently lead to the amplification of somatosensory information and to the priming of fear mechanisms (Crombez, Eccleston, Baeyens, & Eelen, 1998b; Eccleston, 1995; Eccleston et al., 1997). In support of these claims, empirical studies have found that those who catastrophise and report high levels of fear of pain (whether they be pain patients or healthy controls) pay more attention to threat-based information, are vigilant to painful sensations, are less able to direct or sustain attention away from pain-related information and report elevated rates of “non-specific physical complaints” suggestive of heightened somatic awareness (Asmundson, Kuperos et al., 1997; Crombez et al., 1998b; Eccleston et al., 1997; Heyneman, Fremouw, Gano, Kirkland, & Heiden, 1990; McCracken, Faber et al., 1998; J. Pearce & Morley, 1989; M. J. Sullivan et al., 1995), particularly in the face of threat (Crombez et al., 1998b).

Based on self-report questionnaires, pain-related fear and attention to pain sensations are strongly related (McCracken, 1997). In the case of pain-free individuals, Keogh et al. (2001) used a dot probe task to examine the relationship between fear of
pain and attentional bias, aiming to disentangle the causal effects. They found participants with a high fear of pain responded more quickly following presentation of pain-related stimuli, compared to participants with a low to moderate fear of pain. The attentional bias was pain-specific, as fear of pain did not predispose an attentional bias for socially threatening or positive stimuli. This positive finding in pain-free individuals suggests that increased attention to pain-related stimuli predisposes the development of chronic pain, and is not simply a by-product of a persistent state of pain. Further, attentional bias was not correlated with an independent measure of depression (DASS), indicating that the effect was not simply a subset of generalised negative affectivity.

2.4.2.2 Effect of attention on pain intensity and coping

Irrespective of the placing of hypervigilance as a cause or a consequence, it is important to consider the impact of directed attention on the actual experience of chronic pain. A clear effect is difficult to identify, however, as results from research examining the effects of focused attention, distraction and thought suppression strategies are conflicting: some suggest focused attention amplifies pain, yet others suggest the opposite (Villemure & Bushnell, 2002).

Higher ratings of pain were given under instructions to frequently rate acute pain in a post-surgical setting than if ratings were made less often (Levine, Gordon, Smith, & Fields, 1982). By contrast, males, but not females, reported less acute pain during a cold pressor task when they attended to the pain rather than avoided it (Keogh, Hatton, & Ellery, 2000). Ciocco (1991) reported that distraction was more helpful than attention during acute pain episodes, yet recommended the converse for dealing with persistent pain. Harvey and McGuire (2000) experimentally manipulated use of cognitive coping strategies in a sample of patients with chronic pain, divided into three groups (Harvey & McGuire, 2000). The study consisted of a typical thought suppression paradigm, in which all participants were instructed to think about anything during two five-minute blocks. During the intervening five-minute block, one group continued with this instruction, the second group was instructed to suppress pain-related thoughts and the third group was instructed to attend to these thoughts. During the second block, the individuals asked to attend to pain-related thoughts reported that their pain-related thoughts were more frequent, enduring and unpleasant than individuals in the
suppression and control groups, an effect that was sustained during the third phase of the experiment.

Anxiety also appears to influence the impact of attention on pain coping. In a series of studies using healthy adults, anxiety and attentional focus were independently manipulated in response to painful electric shocks (Arntz & de Jong, 1993; Arntz, Dreessen, & de Jong, 1994; Arntz, Dreessen, & Merckelbach, 1991). First, participants who were asked to attend to the painful stimulus gave greater pain ratings than participants who were asked to focus on non-painful anxiety-provoking stimulus. Second, participants gave higher ratings of pain when the painful stimulus was preceded by a pain-related anxiety induction. By contrast, pain ratings did not rise in the face of a personally relevant, but non-pain related threat (presence of a spider for spider phobics). Supported by the effects of the attentional manipulation, the researchers argued that the anxiety induction operated via specific hypervigilance for pain-related stimuli. On the other hand, another group found that patients with chronic pain who were classified as health anxious described less pain and anxiety when they were instructed to focus on physical sensations rather than ignore them (Hadjistavropoulos, Hadjistavropoulos, & Quine, 2000).

2.4.3 Evidence and Effects of Avoidant Behaviour

As noted above, pain specific cognitions and associated fear of pain can contribute to pain patients avoiding activities directly related to their injury. In turn, avoidance of movement and of activities can lead to deconditioning and disability. Based on cognitive-behavioural models it is expected that avoidance leads to fewer opportunities to correct expectancies and beliefs about pain, so that fear of pain becomes dissociated from actual pain experiences (Crombez et al., 1999).

Within a clinical treatment setting, one group argued that avoidance is more likely a product of patient-specific beliefs than simple behavioural learning (Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, 2001). In their report of a series of four clinical cases, they found gradual exposure to individually selected activities extinguished fear-avoidance beliefs more effectively than an activity program based on operant shaping principles alone. This supports the theoretical claim that anticipatory avoidance reinforces fear by preventing the opportunity to correct maladaptive beliefs and expectations. As a case series this study is limited, however, it is supported by findings
of experimental studies. These have shown, for example, that individuals who usually engage in physical activity are less likely to report fear of reinjury during a behavioural test (Crombez et al., 1996) and that repeated exposure to physical tests permitted fear-avoidant chronic pain patients to adjust their initially inaccurate expectations of pain (Crombez et al., 1996). Difficulties generalising the correction suggest that operant conditioning on its own is insufficient to explain avoidance. Rather avoidance is more likely determined by underlying beliefs about danger or harm.

These findings are clouded by two studies in which an opposite effect was reported. Murphy, Lindsay and de C Williams (1997) found the majority of chronic low back pain patients under-predicted the actual pain they experienced during the physical task selected for their study. Similar problems with prediction were demonstrated under conditions of experimentally induced pain. Arntz and Peters (1995) found that patients with chronic back pain significantly under-estimated the level of pain, while healthy control subjects were more accurate in their expectations of pain. One possible interpretation of these conflicting findings is that by limiting accurate feedback, avoidance of pain-eliciting stimuli leads to an inability to predict pain, which can result in either under-estimation or over-estimation of pain. That is, over time, avoidance may result in a lack of predictive knowledge rather than a unidirectional bias toward high pain. Arguably, regular under-prediction may create higher levels of fear.

### 2.4.4 Evidence and Effects of Psychophysiological Reactivity

#### 2.4.4.1 Stress-related physiological reactivity in chronic pain patients

Following a review of the psychophysiological literature, Flor and her colleagues concluded that, relative to healthy controls, chronic pain patients appear to experience elevated physiological arousal and reactivity in response to pain and stress, despite recording equivalent baseline levels (Flor, Birbaumer, & Turk, 1990; Flor & Turk, 1989). In a series of four, counter-balanced tasks, Flor et al. found patients with back pain showed elevated and delayed EMG reactivity in paraspinal musculature during exposure to a meaningful stressor tasks, either discussing pain or a personally relevant stressor (Flor, Turk, & Birbaumer, 1985). The individual’s level of depression and cognitive coping style were also better predictors of this reactivity than were objective features of their pain, such as pain duration or surgery. The findings did not extend, however, to general stressors (arithmetic performance) or neutral tasks (reciting the alphabet) (Flor et al., 1985).
Based on these findings, Flor, Birbaumer & Turk (1990) thus proposed a symptom-specificity model, whereby patients with distinct musculoskeletal pain (e.g., lower back pain) exhibit stress-induced muscular tension at the site of their pain (i.e. lower back). Consistent with the model, psychological stress has been found to also induce reactivity in symptom-specific body musculature for tension headache patients and patients with temporomandibular disorder (Flor, Birbaumer, Schugens, & Lutzenberger, 1992; T. E. Rudy, 1990). Some contradictory evidence exists, however. Others found no group-based evidence of stress-related or symptom specific elevations in muscle tension or muscle reactivity duration in patients with low back pain or upper limb cumulative trauma disorder, compared to patients with arthritis or pain free controls (Spence, Sharpe, Newton-John, & Champion, 2001).

2.4.4.2 Pain-related fear and physiological reactivity

Several factors consistent with the fear-avoidant models appear to mediate the physiological reactivity seen in chronic pain patients. Vlaeyen et al. (1999) demonstrated symptom-specific reactivity in patients with chronic low back pain as they watched a video of a physical task they were told they would later perform. Specifically, highly fearful patients reported significantly higher levels of subjective tension, independent of differences in baseline tension. Higher levels of fear of movement also predicted reactivity of the left paraspinal musculature. Highly fearful patients also tended to exhibit greater startle amplitudes than low fear patients in response to noise bursts presented during a similar video presentation.

Pain-related fear does not, however, appear to influence autonomic arousal. Vlaeyen et al. (1995) found no significant difference in heart rate and skin conductance levels between highly fearful and low fearful patients with chronic low back pain during a lifting task. They thus concluded that muscular but not autonomic function reactivity is relevant.

2.4.4.3 Physiological reactivity and pain perception

While self-reported physiological reactivity has been associated with pain severity, an association between actual physiological reactivity and pain perception is not well-established. Cross-sectional studies conducted by McCracken and his colleagues demonstrated that the level of self-reported physiological arousal generally experienced
in response to pain also predicted greater reported pain severity and physical complaints amongst chronic pain patients (McCracken, Faber et al., 1998; McCracken et al., 1996). The association was further borne out by Evers et al. (2001), who investigated the effects of physiological reactivity in a prospective study of patients with rheumatoid arthritis. After controlling for the effects of initial pain severity, disease activity and neuroticism, they found that self-reported physiological reactivity to pain was the only significant predictor of reported pain one year later.

Turning to the effects of muscular reactivity, Burns, Wiegner, Derleth, Kiselica and Pawl (1997) reported evidence that muscle reactivity and emotional vulnerability to stress may maintain and exacerbate pain in chronic low back pain patients, particularly so in patients who are depressed. Results of their study, which involved a stress induction, found that increases in lower paraspinal muscular reactivity induced by stress predicted greater reported pain severity. Vlaeyen et al. (1999) also found that a lack of habituation in lower paraspinal EMG reactivity predicted higher levels of reported pain in a subsequent lifting task but not a cycling task, supporting the specificity effect. Contrary to prediction, however, the finding only held true for patients scoring low on general negative affectivity. By contrast, the effect did not hold for headache patients. A further study found similar levels of EMG reactivity during headaches and pain-free episodes, in both headache patients and controls. Further, they did not find any significant correspondence between EMG reactivity and stress, negative affect or pain Hatch (Hatch et al., 1991).

2.4.5 Summary

In summary, empirical investigation has broadly supported cognitive-behavioural theories of chronic pain, providing evidence of the presence and effects of cognitive, affective, behavioural and physiological factors resulting in the development and maintenance of persistent pain. As individual factors, variables such as catastrophising pain-related fear and avoidance have been shown to be much stronger predictors of disability than traditional biomedical variables and pain characteristics (e.g., anatomical location, time pattern, severity, duration) (Waddell et al., 1993). Indeed based on this research, a much poorer prognosis is expected for those chronic pain patients prone to interpreting pain or movement as threatening and thus exhibiting intense affective distress, somatic and environmental hypervigilance, avoidance and physiological hyperarousal.
2.5 Limitations of Current Research on Pain-related Fear and Anxiety Models

While there is evidence for a multidimensional pattern of factors impacting on pain and prognosis, many of the studies focus on only one response system or use composite constructs that confound analysis of the interrelationship between response systems and their relative contributions to persistent pain problems (Evers et al., 2001). Further systematic investigation is necessary to clarify their common and independent response effects on chronic pain and to identify underlying mechanisms.

A related difficulty with much of the above research on pain-related fear and anxiety, and possibly an explanation for some of the mixed results, is that there is poor differentiation between the two constructs of anxiety and fear; the terms are used interchangeably (cf. Rhudy & Meagher, 2000; Symreng & Fishman, 2004). Although both constructs involve intense and aversive feelings due to the perception of threat or danger, they can be distinguished on the nature and temporal basis of the threat (for a discussion, see Rhudy & Meagher, 2000). According to Rhudy and Meagher (2000), anxiety is the “apprehensive anticipation of potential threats”. In this way, the focus is a future-orientation to threats that are usually diffuse and often intangible, thus resulting in hypervigilance to somatic sensations and the environment and the mobilisation of somatic tension in preparation for response (p.65). By contrast, fear is “an immediate alarm reaction to present threat”, whereby the threat is current, specific and identifiable, motivating strong escape behaviours and intense autonomic arousal (p.65).

Importantly, Rhudy and Meagher (2000, 2001) posited the valence of affect (pleasant versus unpleasant emotion) and the intensity of arousal as orthogonal factors, such that their combination may result in distinct modulatory effects on pain. That is, they proposed that emotions with negative valence and low arousal would result in pain facilitation, whereas highly arousing negative emotions would cause pain inhibition. Specifically, they argued that a model distinguishing between fear and anxiety would predict that direct exposure to an aversive event would result in an analgesic effect (due to high levels of fear and arousal), whereas anticipation of threat in the absence of actual exposure would elicit a hyperalgesic effect or increased sensitivity to pain (due to lower levels of arousal but heightened somatic vigilance and tension). In support of this, they
contrasted previous research findings of trauma victims’ reports of numbness and the absence of pain during the fear-eliciting trauma with findings of amplified perceptions of pain under conditions of anxiety (as discussed above).

To specifically investigate their proposal, Rhudy and Meagher (2000) conducted an experimental laboratory study testing radiant heat pain threshold in 60 healthy university students under one of three conditions. Participants’ pain thresholds were tested before and after either the induction of fear (via the administration of three electric shocks) or anxiety (shocks were anticipated but not administered) or under neutral circumstances (no threat or actual shock administered). Their hypothesis was supported by the finding that students in the fear condition had significantly higher arousal and pain thresholds (i.e. suggesting an analgesic effect) than students in the neutral condition, whereas students in the anxiety condition demonstrated lower pain thresholds than those in the neutral condition (i.e. suggesting an hyperalgesic effect). From a clinical perspective, these data suggest that a patient anticipating an unpredictable threatening event will experience anxiety that enhances pain, while a patient exposed to a threatening event will experience fear that inhibits pain. Further research is thus required to support this proposal in a clinical sample using designs that manipulate valence and arousal and assess pain on a variety of outcome measures.

2.6 The Nature of the Originating Injury: A Determinant of Pain-related Fear Mechanisms?

The evidence reviewed above also raises important questions about the factors underlying the initiation of fear-avoidant mechanisms. Better understanding of the variables influencing fear-avoidant mechanisms and thus the maintenance of pain problems has important implications for early identification and treatment of those individuals with acute pain, at risk of developing chronic pain problems, and the treatment of individuals who already have chronic pain.

Fritz, George and Delitto (2001) highlighted that “[a]n individual’s fear of pain and the degree to which he or she will seek to avoid painful experiences or behaviours is conditioned by the psychological context within which the painful event occurs” (p.8). Of course, a range of factors could potentially influence the psychological context,
including prior experiences and knowledge of pain and stress, concurrent psychological disorder, personality traits and personal cognitive and coping styles (e.g., see Fritz et al., 2001). Some of these influencing factors may exist before the onset of pain whilst others may arise simultaneously from the same episode. One specific area deserving of attention is the nature of the originating injury itself and associated psychological sequelae.

There has been some discussion in the chronic pain literature that maladaptive fear-avoidant beliefs and behaviour may be particularly prominent when the original, acute pain condition resulted from a sudden or traumatic injury (Crombez et al., 1999; Sharp & Harvey, 2001; Turk & Holzman, 1986; Turk & Okifuji, 1996; Vlaeyen et al., 1995a). One study, purporting to test the assumption that sudden pain onset may result in greater pain-related fear, classified the type of onset by the patient’s ability to recall the day when they first felt their current back pain (Crombez et al., 1999, Study 3). Obviously, this assumes that if one cannot remember a specific date then the pain must have been gradual. Chronic pain patients with sudden initial onset of back pain reported significantly greater fear of physical movement and reinjury (TSK), but not pain-related anxiety (PASS), compared to patients who described a gradual onset of pain. There were some inconsistent findings, however, as patients with gradual onset of pain reported greater disability (despite less fear) than patients with sudden onset, contrary to predictions based on past findings. Although these findings are supportive of the likely impact of trauma, the relatively crude classification of participants (sudden vs gradual; phobic rather than traumatised) limits generalisation.

Even though a general relationship between chronic pain and fear/anxiety constructs has been well-established in the past decade, the impact of trauma per se remains poorly understood due to a paucity of empirical research in this area. There have been no studies to date that have comprehensively examined pain-related fear constructs in the context of posttraumatic psychological symptoms and it is not clear whether or how trauma might impact on fear-avoidant mechanisms in the context of pain. Given the nature of posttraumatic reactions it is reasonable to believe that they could interact with if not amplify pain and fear-avoidant mechanism. The next chapter reviews empirical evidence that illustrates a possible link between chronic pain and psychological trauma.
A growing body of empirical research is uncovering relationships between injury, pain and psychological trauma with important implications for the prevention and management of chronic pain and posttraumatic reactions. Discovery of the associations has emerged from three types of investigation. The first type of investigation is the most basic of the three, offering simple estimates of the high prevalence of trauma-related factors in chronic pain samples and of injury and chronic pain in traumatised samples. The second type of study selects a cross-section of individuals with chronic pain conditions and or Posttraumatic Stress Disorder (PTSD) to explore the apparently negative impact of comorbidity on the individual’s ability to manage the respective conditions. The third type of investigation is prospective, usually commencing from the point of exposure to a traumatic stressor. Most commonly, this research is interested in discovering risk factors for the development and maintenance of either Posttraumatic Stress Disorder or chronic pain.

This chapter reviews findings from all three perspectives and highlights gaps in our current understanding of the relationships between various constructs related to injury, pain and psychological trauma. As reviewed below, the prevalence of PTSD and other trauma symptoms is generally higher and the prognosis poorer amongst injured individuals and chronic pain patients compared to community rates. Conversely, amongst individuals with prior exposure to trauma or current PTSD the prevalence of chronic pain is also higher and the prognosis poorer than the rates for community samples that were reported in Chapter 2. Together these findings suggest an interdependence between chronic pain and PTSD that extends beyond a simple, additive effect of comorbid injury and psychological trauma.

Unfortunately, very little research has focused on the simultaneous evaluation of chronic pain and formally diagnosed PTSD. Rather, the avenues of inquiry conducted within the pain and trauma literatures have remained relatively isolated from each other, providing inconclusive data when conducted independent of an overarching theoretical framework to guide investigations. As discussed below, an emphasis on trauma
exposure (objectively defined) and broad traumatic reactions (rather than PTSD per se) restricts much of the pain-directed research. The trauma-directed research has likewise floundered due to an outdated emphasis on objective injury rather than subjective adjustment to injury and processes related to persistent pain in understanding the maintenance of PTSD. Indeed, exploration of the underlying mechanisms linking injury, pain and psychological trauma remains a fertile field in need of systematic research. Before embarking on a review of these issues, the next section provides key definitions and background about trauma and PTSD.

3.1 Background: Psychological Trauma and Posttraumatic Stress Disorder

3.1.1 Definition of Psychological Trauma

The definition of “traumatic” continues to be disputed, but most commonly research relies upon the DSM-IV definition of a traumatic stressor (American Psychiatric Association, 1994), as retained in the text revision (DSM-IV TR) (American Psychiatric Association, 2000). In this, a traumatic stressor is one involving “actual or threatened death or serious injury or other threat to the physical integrity of self or others” (p. 467, my emphasis). Further, the individual must respond to the experience with “intense fear, helplessness or horror” (p. 467). This definition, as with the definition of pain in Chapter 2, therefore involves subjective perception, in this case, of threat to life or person, coupled with an emotional response. March (1993) also discussed at length the role of subjective perception of threat in trauma, as distinct from objectively identified threats. He argued that the meaning of the event as perceived by the traumatised person is more crucial than what factually transpires; just as the personal meaning ascribed to an injury may be more predictive of pain than actual tissue damage. Subsequent references to trauma in this thesis rely on the psychological definition, as distinct from the physical/medical definition of trauma as “an injury to living tissue caused by an extrinsic agent” (Plus, 2005).

3.1.2 Disorders Stemming from Psychological Trauma

Two disorders most clearly related to a traumatic event are PTSD and Acute Stress Disorder (ASD) (American Psychiatric Association, 2000). Other Axis I disorders, such as Major Depressive Disorder and Panic Disorder, may also ensue from traumatic exposure and are often comorbid with PTSD (Breslau, Davis, Peterson, & Schultz,
2000; Creamer, Burgess, & McFarlane, 2001; R. C. Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Indeed, in an Australian sample, 85% of males and 80% of females with PTSD met criteria for another Axis I disorder in the previous 12 months (Creamer et al., 2001). Estimates from the United States are similar, with approximately 80% of individuals with PTSD meeting criteria for at least one other DSM disorder, such as mood or anxiety disorders and substance abuse or dependence (R. C. Kessler et al., 1995).

### 3.1.3 Definition of Posttraumatic Stress Disorder

The hallmark symptoms of PTSD, as reflected in DSM-IV TR criteria (pp. 467-468), are persistent re-experiencing of the traumatic event (at least one of: either recurrent intrusive recollections, dreams or flashbacks, or psychological distress or physiological reactivity on exposure to reminder cues – Cluster B), persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (at least three of: avoiding thoughts, feelings or conversations; avoiding activities, places or people; inability to recall an important aspect of the trauma, diminished interest; social detachment or estrangement; restricted affect; sense of a foreshortened future – Cluster C) and persistent symptoms of increased arousal (at least two out of: sleep disturbance, irritability/anger, difficulty concentrating, hypervigilance, exaggerated startle – Cluster D). In addition, some researchers have considered a sub-syndromal condition, “Partial PTSD”, in which individuals display the requisite re-experiencing symptoms and either sufficient Cluster C or D symptoms but not both (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994). Some criticism has been directed towards such a move, not least because it blurs the distinction between disorder and ‘normal’ responses to stress (R. J. McNally, 2003).

Keeping in mind the key features of chronic pain reviewed in Chapter 2, *prima facie*, there are clear similarities between the two conditions. Specifically, fear, anxiety, avoidance, hypervigilance and physiological hyperarousal are not only typical of chronic pain, they also appear in the diagnostic criteria for PTSD. Research highlighting the confluence of these features is reviewed below, provoking questions as to whether the similarities run deeper, reflecting “mutual maintenance” (Sharp & Harvey, 2001) or a “shared vulnerability” (Asmundson, Coons, Taylor, & Katz, 2002).
3.1.4 Definition of Acute Stress Disorder

ASD is distinguished from PTSD, primarily by the duration and number of symptoms necessary for diagnosis. As the name suggests, the disorder is acute and symptoms must resolve within 4 weeks of the event to meet DSM-IV TR criterion. Diagnosis of ASD involves the same stressor criterion and similar re-experiencing, avoidance and hyperarousal symptoms as PTSD but only one symptom from each cluster is necessary. There is also the added requirement of experiencing at least three dissociative symptoms during the actual event (i.e. a subjective sense of numbing, detachment, or absence of emotional responsiveness; reduction in awareness of surroundings; derealisation, depersonalisation or dissociative amnesia: Criterion B, DSM-IV TR, pp. 471–472). The disorder has been criticised for the lack of evidence to support the dissociative symptom criterion and for its low sensitivity in predicting PTSD, that is, many who develop PTSD did not initially satisfy ASD criteria (R. A. Bryant & Harvey, 1997; Marshall, Spitzer, & Liebowitz, 1999).

Whilst possible associations between injury, acute pain and ASD are acknowledged, in keeping with this thesis’ focus on chronic disorder, a choice has been made to emphasise findings related to PTSD.

3.1.5 General Prevalence of Trauma and PTSD

Even though exposure to traumatic stressors is quite common, onset of PTSD is much less so. In Australia, data from the National Survey of Mental Health and Wellbeing (ABS, 1998) indicated that of 10,641 participants assessed in 1997, 65% of men and 50% of women reported experiencing at least one traumatic event in their lifetime (excluding childhood abuse) (Creamer et al., 2001; Rosenman, 2002). The community incidence of PTSD in this study was only 1.5%, substantially lower than the 3.9% incidence rate reported for community samples in the United States (National Comorbidity Study) (R. C. Kessler et al., 1995). Lifetime prevalence of PTSD in US community samples is higher again and tends to fall around 5 to 10% of the general population, or 15 to 25% of those exposed to traumatic events (Breslau, 2001).

Australian data, however, indicates that the incidence amongst those exposed to traumatic events is lower in Australia than for the US, with only 3.8% of women and 2% of men meeting criteria for PTSD in the 12 months covered by the study (Creamer et al., 2001; Rosenman, 2002). Similarly, only 2.6% of individuals who had a lifetime experience of trauma developed PTSD (Rosenman, 2002).
Indeed, most people develop at least some psychological symptoms and distress in the immediate aftermath of a traumatic incident, as part of a 'normal' human response to an extreme stressor or 'abnormal' event. What distinguishes the symptoms as pathological is their intensity, frequency and duration (greater than 1 month for PTSD), as well as the degree of distress and impairment the symptoms cause – together indicating a failure of stress adaptation. Although the majority of people go on to recover from exposure to trauma within a few days or weeks, a significant minority exhibit disordered symptoms, or a significant subset of symptoms, that persist, in some cases for years. For example, one study showed that individuals who did or did not develop PTSD after an MVA exhibited similar levels of anxiety on the first day, with differences not emerging until the tenth day (McFarlane, 2000). In another study of MVA survivors, half of the individuals initially diagnosed with PTSD no longer met full criteria at 6 months and two thirds had remitted by 12 months post-trauma (Blanchard, Hickling, Barton et al., 1996). Similarly, between 5 and 11 weeks after a rape, there was a 17% drop in the number of victims meeting criteria for PTSD (47% down from 64%) (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). The National Comorbidity Survey found that about 60% of PTSD cases remitted, with or without treatment (R. C. Kessler et al., 1995).

3.1.6 Factors Related to the Development and Maintenance of PTSD

Clearly, exposure to a traumatic stressor is a necessary but not sufficient precursor of PTSD. Rather, the development of PTSD most likely depends on a complex interaction between pre-existing, peri-traumatic and post-event variables that determine risk and resilience to the disorder (Brewin, Andrews, & Valentine, 2000; Yehuda, 1999). Although a broad range of pre-existing (“vulnerability”) factors has been investigated for PTSD, consistent evidence has only been obtained for the effects of prior psychiatric history, pre-trauma vulnerability to anxiety and depression and prior exposure to trauma, with multiple exposures resulting in additional risk (B. L. Green, Goodman, & Krupnick, 2000).

Investigations of peri-traumatic variables – variables occurring at the time of the event or immediately afterwards – have revealed strong effects for the severity of the actual event (Brewin et al., 2000; Creamer et al., 2001; R. C. Kessler et al., 1995), the nature of the event (Rosenman, 2002) and of peri-traumatic responses. With respect to
the severity and nature of the event, events involving violence, high threat to life, exposure to the suffering of others and prolonged duration are important precursors of PTSD. Immediate responses to trauma, such as the individual’s interpretation of the trauma (Ehlers & Clarke, 2000), elevated physiological arousal (R. A. Bryant, Harvey, Guthrie, & Moulds, 2000; Shalev, Sahar, Freedman, & al, 1998) and dissociation (Fullerton, Ursano, Epstein, & al, 2000) have also been linked with a higher risk of developing PTSD.

Notably, the longer the disorder persists, the less trauma exposure explains symptoms (McFarlane & Yehuda, 1996), pointing to the maintenance role of post-traumatic variables. Post-event variables, such as the individual’s interpretation of trauma sequelae and subsequent cognitive and behavioural avoidance of trauma-related stimuli have been shown to impact on the development and, moreover, the maintenance of PTSD (Ehlers & Clarke, 2000).

As peri-traumatic factors central to the issue of threat and bodily harm, injury and pain could be important to the onset of PTSD. When persistent, they may take on the additional maintenance role of post-event factors. For example, pain may be experienced as an intrusive memory (then misconstrued as a current threat or sign of permanent change) or an unwanted reminder of the traumatic event (thereby triggering distress, hyperarousal and avoidance). Either way, this creates feedback between PTSD and chronic pain. Thus, in addition to understanding how PTSD may affect relevant aspects of the chronic pain experience as reviewed in Chapter 2, it is also important to understand the reciprocal relationship of how injury and chronic pain may affect PTSD.

The remainder of this chapter reviews the current evidence on both of these points, drawing attention to gaps in the existing evidence base. It begins with a summary of comorbidity data, before moving through an evaluation of cross-sectional and longitudinal studies investigating the relationships between injury, pain, trauma and PTSD.

3.2 Comorbidity between Prior Trauma Exposure and Chronic Pain

Examination of the prevalence of childhood physical and sexual abuse in chronic pain patients supports an association between abuse, particularly sexual abuse, and the
existence of pain complaints in adults. Elevated rates of abuse in chronic pain patients compared to non-pain controls or controls with specific “organic” pain disorders suggest pain and abuse share a connection, at least at an incidental level. Amongst patients with heterogeneous pain complaints, prevalence estimates range between 28% and 48% (R. T. Goldberg, 1994; Toomey, Seville, Mann, Abashian, & Grant, 1995; Wurtele, Kaplan, & Keairnes, 1990). Similarly high rates of abuse, from 11% to 67%, have been reported in conjunction with various specific chronic pain conditions, including chronic headache (Domino & Haber, 1987), chronic low back pain (Pecukonis, 1996; Schofferman, Anderson, Hines, Smith, & Keane, 1993; Schofferman, Anderson, Hines, Smith, & White, 1992), fibromyalgia and rheumatic disease (Alexander et al., 1998; Boisset-Pioro, Esdaile, & Fitzcharles, 1995; J. L. Taylor, Trotter, & Csuka, 1995; E. A. Walker et al., 1997), temporomandibular disorders (Riley, Robinson, Kvaal, & Gremillion, 1998) chronic pelvic pain (Fry, Crisp, Beard, & McGuigan, 1993; Jamieson & Steege, 1997; Rapkin, Kames, Darke, Stampler, & Naliboff, 1990; Toomey, Hernandez, Gittelman, & Hulka, 1993; E. Walker et al., 1988; Walling et al., 1994) and chronic gastro-intestinal pain (Drossman et al., 1990; Leserman et al., 1996; Scarinci, McDonald-Haile, Bradley, & Richter, 1994; E. A. Walker, Katon, Roy-Byrne, Jemelka, & Russo, 1993).

### 3.2.1 Estimates Based on Treatment-seeking Individuals

Data obtained from general medical and gynaecological samples generally supports an association between a history of trauma and chronic pain (Dickinson, deGruy III, Dickinson, & Candib, 1999; Finestone et al., 2000; Lechner, Vogel, Garcia-Shelton, Leichter, & Steibel, 1993; Leserman, Li, Drossman, & Hu, 1998; Moeller, Bachman, & Moeller, 1993). Elevated rates of general pain complaints have been found in female family practice clients who reported a history of severe sexual abuse (Dickinson et al., 1999). Abused women presenting with gastroenterology complaints also presented with increased musculoskeletal symptoms compared to non-abused counterparts (Leserman et al., 1998). At a gynaecological clinic, women with a history of abuse reported experiencing more frequent headaches and abdominal pain than women without such a history (Moeller et al., 1993). In a primary care setting, women who had a history of childhood sexual abuse were no more likely to report pain conditions than women with no such history; however, pain complaints were more common when women were abused as both children and adults (Jamieson & Steege, 1997). In a similar setting, women who reported childhood sexual abuse suffered from more medical symptom
complaints but not more musculoskeletal complaints when compared to women without abuse histories. It is thus not clear from these conflicting results whether a prior history of abuse actually influences the presentation of pain.

3.2.2 Estimates Based on Community Samples

As prior abuse is associated with higher rates of health care utilisation (Aaron et al., 1997; Alexander et al., 1998; Bendixen, Muus, & Schei, 1994; Drossman et al., 1990; Fillingim, Wilkinson, & Powell, 1999; Finestone et al., 2000; Talley, Boyce, & Jones, 1997; Toomey et al., 1995), the elevated rates discussed above may arise from sampling bias. Community-based studies are thus valuable in limiting this potential confound by also sampling the population of individuals who are not seeking treatment for pain conditions.

Evidence of a correlation between childhood abuse and chronic pain within community populations (Fillingim et al., 1999; Golding, 1994; S.J. Linton, 1997, 2002; McBeth, MacFarlane, Bengamin, Morris, & Silman, 1999) indeed suggests that the association is more than just a spurious relationship detected when sampling patient populations. At the most basic level, (McBeth et al., 1999) found that individuals who reported a history of childhood adversity (a broad concept including abuse, parental loss, family illness etc) reported more points of tenderness to palpation than did those without such a history. A study of 1610 women drawn from the Los Angeles region revealed that the 14% of women who reported a lifetime history of sexual assault also reported more pain symptoms than non-assaulted women (Golding, 1994). From a study of 426 college students, Fillingim, Wilkinson and Powell (1999) reported higher ratings of pain and more pain sites over the previous month for students who also reported a history of abuse, compared to those who denied a history of abuse. Linton (1997) conducted a large population study of 949 individuals, selected from 3000, 35-45 year-olds randomly selected from Swedish census data. Similar rates of self-reported abuse were found amongst a group of patients reporting pronounced pain drawn from the general population as compared to a clinical reference group of 142 patients with chronic musculoskeletal pain. Females, but not males, who reported a history of abuse were found to be at increased risk of experiencing pronounced pain. A four-fold increase in risk was predicted by physical abuse, sharpening to a five times greater risk in those who reported sexual abuse. Linton (1997) concluded that abuse could be a useful predictor of chronic pain.
As a follow-up to his 1997 study, Linton (2002) completed a prospective study evaluating the effect of abuse history on the risk of developing pain complaints. Linton (2002) re-surveyed female community participants 12 months after they completed the initial survey, discussed above. He found that amongst participants who were pain free at baseline, those who also reported a history of physical abuse had two and a half times the risk of reporting back pain and four times the risk of reporting more physical function problems 12 months later; and those who reported a history of sexual abuse were three times more likely to report poorer physical function after 12 months; compared to participants who did not report a history of abuse. By contrast, there was no obvious impact of abuse on risk of worse pain or decreased functioning for patients who reported pain at baseline. Despite the otherwise prospective nature of this research, one of the obvious limitations is that it relied on retrospective reports of childhood abuse.

3.3 Comorbidity between PTSD and Chronic Pain

For those individuals seeking treatment for chronic pain, PTSD (as a specific diagnosis) is frequently comorbid. In an investigation of 55 consecutive referrals to a pain clinic secondary to an MVA, 21 were diagnosed as having “accident phobia,” with 8 of these also meeting criteria for PTSD, and a further 5 exhibiting PTSD in partial remission (Kuch, Cox, Evans, & Shulman, 1994). Another cross-sectional study assessed 20 consecutive referrals to a private psychology practice (Hickling & Blanchard, 1992). Although referral was for treatment of post-MVA pain, half of the patients also met diagnostic criteria for PTSD and three others were sub-syndromal. A further study assessed the presence of PTSD within the more specific population of fibromyalgia patients, classified according to the nature of pain onset (Kuch, Evans, Watson, & Bubela, 1991). Administration of the Structured Clinical Interview for DSM-III-R (SCID) revealed that patients who developed fibromyalgia following a road accident were more likely to be diagnosed with PTSD or a phobia than patients with another type of onset. Prevalence data collected at a veterans’ outpatient pain clinic indicated that 10% of the 225 patients met DSM-III criteria for PTSD (Benedikt & Kolb, 1986). Interestingly, these veterans had never been diagnosed with PTSD, suggesting that the diagnosis may go undetected when pain is the primary reason for presentation, despite the high rates of trauma documented in this population. Data from
the National Comorbidity study showed that patients with musculoskeletal pain had a four times greater risk of having PTSD than those without pain (Cox & McWilliams, 2002).

Two studies provide exceptions to this pattern of generally elevated rates. The first, demonstrated a prevalence rate similar to community rates (1.7%) in a small sample of chronic pain patients (Kouyanou, Pither, Rabe-Hesketh, & Wessley, 1998). In the second study, of 571 fibromyalgia patients, the increased odds of lifetime PTSD (i.e. 20% of the sample) were confined to those who also had a lifetime diagnosis of Major Depressive Disorder (Roy-Byrne, Smith, Goldberg, Afari, & Buchwald, 2004).

Evidence for an association between chronic pain and PTSD also has been collected in studies of individuals diagnosed with PTSD. A review of 8000 patient discharge summaries at a veteran medical centre, revealed 25% of PTSD patients reported either a musculoskeletal or pain problem (White & Faustman, 1989). Similarly, Amir, Kaplan, Neumann, Sharabani, Shani and Buskila (1997) found a higher prevalence of fibromyalgia in a group of PTSD patients (21%) compared to controls (0%). PTSD has also been identified as a predictor of subsequent pain problems in a non-treatment seeking, community-based population (Amir et al., 1997). Using a random sample of 1200 individuals from a list of 21-30 year-old members of a Michigan health maintenance organisation, another study examined the lifetime associations between "somatisation" symptoms and PTSD (Andreski, CHilcoat, & Breslau, 1998). Cross-sectional analyses conducted at baseline showed individuals with and without a history of PTSD did not differ in their report of pain symptoms. However, prospective analyses based on combined data from 3- and 5-year follow-ups demonstrated that a history of PTSD at baseline increased the risk of first onset of pain symptoms by two-fold. Andreski et al. (1998) argued that physiological correlates of PTSD may lower the threshold for pain perception or cause pain directly.

PTSD alone, and when accompanied by depression, has a negative relationship with the general physical health status of veterans (Calhoun, Bosworth, Grambow, Dudley, & Beckham, 2002; Deykin et al., 2001; C. Engel, Liu, McCarthy, Miller, & Ursano, 2002; Calhoun, Bosworth, Grambow, Dudley, & Beckham, 2002; Deykin et al., 2001; C. Engel, Liu, McCarthy, Miller, & Ursano, 2002).

1 It must be noted, however, that a zero prevalence rate in the control group does not represent the normal prevalence rate of fibromyalgia.
Further, in the year after being diagnosed with PTSD, combat veterans are more likely to present to outpatient physical health services (18 visits) than outpatient mental health clinics (7 visits) (Calhoun et al., 2002). Moreover, findings from the same study showed that young veterans with PTSD utilised physical and mental health care at a much higher rate (124% and 97-260%, respectively) than young veterans without PTSD. One possible explanation for these findings is that veterans with PTSD (as with abuse populations and, perhaps, other traumatised populations) are simply more likely to seek out health care. This pattern may be a function of coping style, socio-cultural biases or access issues, however, it is possible that PTSD directly influences physical health. Indeed, in Calhoun et al.'s (2002) study although a positive service-connected disability status interacted with rates of health care utilisation at lower levels of PTSD severity, those with moderate to severe levels of PTSD were still more likely to seek out health care even when they were billed personally for the service, suggesting the problem is not simply an artifact of compensation. Further, the relationship between PTSD and rates of general health care use was mediated by a correlation between PTSD and physician-diagnosed health complaints, indicating that the tendency of individuals with PTSD to seek health care is due to higher rates of diagnosable health problems and is not just motivated by greater levels of distress (Deykin et al., 2001).

In summary, while reported prevalence rates vary, for the most part, the reviewed studies typically point to higher rates of PTSD in pain patients and, conversely, higher rates of pain in PTSD patients when contrasted with rates detected in the general population.

3.4 The Unique Contribution of PTSD versus Trauma History to Chronic Pain Problems

Studies of holocaust survivors, combat veterans, burns patients, patients seeking treatment for chronic pain and survivors of abuse have supplied mixed results about the impact of prior trauma on pain problems, such as onset and severity of symptoms. Based on the available evidence, there is reason to believe that PTSD and other subjective responses may mediate the apparent relationship between trauma and chronic pain problems.
3.4.1 Trauma History and Pain Severity

Yaari, Eisenberg, Adler and Birkhan (1999) compared the pain characteristics of 33 survivors of the Holocaust (civilians) with those of a control group who did not witness war atrocities. Fifty years after World War II, Holocaust survivors continued to report higher pain levels, more pain sites, greater depression, and utilised health services more often than their non-exposed counterparts. Toomey, Seville, Abashian, Finkel and Mann (1994) also compared patients with pain of traumatic and non-traumatic origin (defined according to the objective nature of the event). Those with traumatic-onset pain reported greater pain intensity and psychological distress. Disability did not differ between these two groups, however the assessment was based on a narrow definition of functional activity.

Support for the existence of a link between a prior history of abuse and the severity of pain problems is equivocal. Several studies sampling the treatment seeking population provide some support for a link. Leserman et al. (1996) found differences in ratings of pain, non-gastrointestinal somatic symptoms, bed disability days and functional disability between women with or without a history of sexual abuse in a group of women reporting gastro-intestinal pain. Moreover, a dose-specific effect appeared. Women who reported being exposed to more serious abuse, such as rape and life-threatening physical abuse, had worse health complaints than women subjected to less serious abuse, such as minor physical assault, sexual touching or attempted sexual assault. Scarinci et al. (1994) investigated pain perception in women with gastro-intestinal pain, finding that abused and non-abused groups did not differ on ratings of pain intensity. During experimental investigation abused patients demonstrated lower thresholds for pain detection and biased responses in tests of sensory discrimination. Scarinci et al. (1994) reasoned that the altered sensory processing occurred because abused patients were hypervigilant to noxious stimuli and thus more likely to perceive stimuli as painful. It is possible, however, that chronic physiological arousal led to hypersensitivity to pain in these patients (Butler & Moseley, 2003).

In another study of women drawn from the general community, Taylor et al. (1995) did not find any difference in the prevalence of abuse history reported by women with fibromyalgia compared to those without fibromyalgia. Amongst the women with fibromyalgia however, those who reported a history of sexual abuse reported a greater severity of illness than did non-abused patients. They also demonstrated allodynia (a
symptom wherein normally innocuous stimuli is perceived as painful). Women seeking treatment for fibromyalgia, who also reported a history of sexual or physical abuse, demonstrated greater pain, fatigue, functional disability and stress compared to women who did not report an abuse history.

By contrast, two independent studies conducted by Toomey and colleagues found equivalent ratings of pain intensity, frequency, duration and disability across patients with pelvic pain and heterogeneous pain complaints, irrespective of abuse history, indicating that abuse history did not impact on pain severity (Toomey et al., 1993; Toomey et al., 1995). The possibility of a ceiling effect in the latter study, however, might have suppressed significant findings.

Criticism about the lack of objective indicators of abuse and the potential for memorial bias in retrospective reports has been directed at the above cross-sectional studies. In addition, the lengthy time course between the trauma and assessment of pain (Yaari, Eisenberg, Adler, & Birkhan, 1999) introduces wide opportunity for the influence of multiple confounds. Prospective studies commencing closer to the period of abuse, on the other hand, aim to reduce the influence of memorial bias and enable greater confidence in causal attributions (not possible from cross-sectional studies) in the investigation of the potential impact of abuse on the development of pain.

In one study, child and adolescent victims of documented sexual abuse were followed-up over a two-year period (Rimsza, Berg, & Locke, 1988) to determine whether abuse increased their risk of developing pain. The study relied on caregiver reports and medical records and supported the existence of a relationship in the expected direction, over a relatively short follow-up period. Compared to matched non-abused controls, abused children and adolescents demonstrated higher rates of muscle tension and gastrointestinal symptoms, including stomach pain, when re-assessed two years later.

Using a more rigorous, double-blind, prospective cohort design, Raphael et al. (2001) investigated the longer-term consequences of childhood victimisation on various pain outcomes (Raphael, Spatz Widom, & Lange, 2001). Children identified as having experienced court-substantiated physical abuse, sexual abuse or neglect before the age of 11 were followed up approximately 20 years later. The control group of non-abused
children was matched according to demographic data obtained from birth and school records. Retrospective self-reports of childhood victimisation, current pain symptoms and lifetime history of major depression were obtained via structured and semi-structured interviews. Analysed as a group, adults with documented reports of early childhood victimisation did not differ from adults without a documented abuse history on any of the pain measures. That is, there were no overall differences in the number of reported pain symptoms, the amount of pain attributed to physical illness or injury, unexplained pain, problematic pain (i.e. pain complaints that led to treatment seeking or caused life interference), or the percentage of problematic pain. Further, univariate analyses indicated no significant relationships existed between sexual abuse or neglect and pain symptoms. A trend suggesting physical abuse was associated with more problematic pain was non-significant following adjustments for multiple comparisons.

In addition, there was no evidence of depression moderating a positive link between childhood victimisation and pain in adulthood. In fact, the contrary was true: only those without a history of physical abuse demonstrated a positive relationship between major depression and unexplained pain.

Raphael et al.'s (2001) study becomes more interesting, however, when one compares the results obtained using retrospective self-reports of victimisation. On this basis, all measures of adult pain complaints (except the percentage of problematic pain) demonstrated a significant relationship with childhood victimisation, consistent with earlier findings from cross-sectional studies. The interest lies not so much in this finding, as in the authors report that victimisation was self-reported by only 73% of individuals with a documented history and 49% of individuals without a documented history (i.e. controls). As much as this disparity speaks strongly to the existence of a memory or reporting bias and thus the problems of retrospective study, arguably it also speaks to a problem in objectively defining exposure to child victimisation to address apparent links between abuse history and chronic pain (Raphael, Widom, & Lange, 2002a, 2002b). The disparity between documented and self-reported abuse could signify that documented cases substantially underreport the incidence of abuse and there is real doubt cast over the supposed control sample as it was substantially tainted. Raphael et al. (2001) discounted the possibility of Type II errors, however, citing equivalent results across welfare status as an indication that children were appropriately classified. They further assumed the existence of a dose-response effect of abuse on documentation, taking reassurance from their belief that documented reports would
include the most serious cases. Perhaps serious cases of physical abuse and neglect are more likely to come to the attention of public authorities as the physical effects are more readily observable, however it would be unwise to assume that serious cases of sexual abuse are necessarily reported to or detected by authorities. Finally, Raphael, Widom & Lange (2002a,b) in response to criticism, argued that “misclassification error was not so severe as to negate findings for other mental health outcomes...suggest[ing] that any possible minor differences in rates of pain-related symptoms that may have been attenuated by measurement error in classification of victimisation status among our controls are unlikely to have important clinical significance.” (p.234). Although plausible, the argument is still speculative and cannot rule out that findings were attenuated by the tainted control sample.

A prospective study investigating changes during the acute stage of whiplash injury following an MVA demonstrated that initial posttraumatic reactions were linked with worse pain symptoms over time (Sterling, Jull, Vicenzino, Kenardy, & Darnell, 2005). Impact of Event Scale (IES) scores at 1 month together with cold pain threshold, age and initial pain severity and disability significantly predicted classification of whiplash symptoms as moderate to severe (cf. mild or resolved) at 6 months. Notably, the addition of IES and cold pain threshold increased the accuracy of prediction by 32% compared to the other traditional factors. More specifically, an earlier report on the same sample indicated that participants’ whose whiplash symptoms were either mild or abated had low IES scores, which were relatively stable, across the 6 month period (Sterling, Kenardy, Jull, & Vicenzino, 2003c). In contrast, individuals with moderate to severe whiplash symptoms at 6 months initially had moderate elevations on the IES. While IES scores dropped to the mildly elevated range by 6 months, the change was due to a reduction in intrusion not avoidance, suggesting that avoidance was more likely to underlie the stability of whiplash symptoms in this group. These findings differ from those reported in a series of papers based on a prospective study of 117 Swiss motor vehicle accident survivors followed over two years (e.g., Radanov, Begre, Sturzenegger & Augustiny, 1996; Radanov, di Stefano, Schnidrig & Sturzenegger, 1994; Radanov, Sturzenegger, di Stefano & Schnidrig, 1994). Radanov and his colleagues found that psychosocial factors were not predictive of the course of whiplash symptoms and argued that psychological symptoms were most likely the consequence not the cause of somatic symptoms.
3.4.2 PTSD and Pain Severity

A glaring issue shared by many of the above studies is the emphasis on trauma exposure at the expense of considering posttraumatic reactions, including the specific case of PTSD. Most of the studies' classification of the “trauma” group is based only on exposure to a particular type of incident. That is, it is presumed that some incidents are inherently traumatic (e.g., combat, abuse, MVAs, disasters), without regard to the subjective experience of the person (a problem common to stress research, too). The association with chronic pain, however, may not depend so much on exposure to a traumatic event, but rather the subjective perception of trauma and response to such an experience. This argument is in keeping with current conceptualisations of PTSD (DSM-IV TR, APA, 2000; March, 1994). Alternatively, where traumatic reactions were considered, the range of symptoms was broad, rather than specific to PTSD. This distinction is relevant as path analyses have indicated that 50-75% of the effects of traumatic exposure on self-reported health and the number of health problems were mediated by PTSD (Friedman & Schnurr, 1995).

A link between PTSD and pain severity has been established in the context of acute pain, such as burn injuries (Perry, Cella, Falkenberg, Heidrich, & Goodwin, 1987) and HIV/AIDS-related pain (Smith, Egert, Winkel, & Jacobson, 2002). For example, Perry et al. (1987) conducted their study of 134 patients approximately nine days after the burn injury occurred (i.e. acute phase), examining ratings of pain before and during wound debridement on four consecutive days. They found reports of procedural and non-procedural pain were greater in patients diagnosed with PTSD (using the SCID), compared to patients not diagnosed with PTSD. Dosages of morphine were kept equivalent, indicating that administration of analgesia was not causal (although differences in endogenous opioids may have been present). There is doubt as to whether these results can be generalised, however, as PTSD patients also had slightly larger burns and delirium and differed on demographic variables (male, married, employed) compared to patients without PTSD. Smith et al. (2002) considered a sample of 142 HIV/AIDS patients enrolled in a clinical trial of pain-communication strategies. Acceptance into the clinical trial required persistent pain over the past two weeks, rated above 5 on a scale ranging from 0-10. Using symptom clusters on the PTSD Checklist-Civilian version, approximately half were identified as having PTSD. Patients with PTSD reported significantly higher levels of worst pain and pain-related interference than those patients without PTSD. After controlling for health status, risk factors and
socio-demographic factors, ratings of ‘worst pain’ made by HIV/AIDS patients with PTSD were sustained over 6 months, whereas ratings declined significantly for HIV/AIDS patients without PTSD.

A number of studies involving veterans, sexual assault survivors and burn patients have described differential contributions of the three PTSD symptom clusters to pain and related problems. For example, one such study explored the relationship between chronic pain and PTSD in a study of veterans at an out-patient PTSD clinic (Beckham et al., 1997). PTSD diagnosis was established by consensus of the clinical team and based on responses to the Combat Exposure Scale, the Mississippi Scale for Combat-Related PTSD and the Clinician-Administered PTSD Scale (CAPS). Chronic pain was reported by 80% of the 129 veterans with an established PTSD diagnosis. Moreover, amongst the PTSD patients who reported pain, regression analyses revealed that the severity of re-experiencing symptoms was significantly associated with overall pain index, current pain rating and pain disability. The finding persisted even after adjusting for age, SES, combat exposure, depression and somatisation scores. Other studies have also reported an effect of re-experiencing on pain amongst veterans (Helzer, Robins, & McEvoy, 1987; R.K. Pitman, Altman, & Macklin, 1989).

The findings are not limited, however, to the veteran population. A study of 76 female sexual assault survivors showed that PTSD severity was a better predictor of self-reported physical symptoms than depression, anger and negative life events (Zoellner, Goodwin, & Foa, 2000). Moreover, the association specifically occurred between the re-experiencing cluster and not hyperarousal. An effect involving re-experiencing symptoms was likewise pronounced in a study of fire-fighters who were all exposed to the Ash Wednesday bush-fires in South Australia (McFarlane, Atchison, Rafalowicz, & Papay, 1994). Among other somatic symptoms, fire-fighters who experienced PTSD reported higher rates of musculoskeletal symptoms than their counterparts who were exposed to the fires but were unaffected by PTSD. The most common physical complaint was pain with 45% reporting back pain and 34% headaches. Moreover, a dose-response effect appeared. Within the PTSD group, those who reported physical symptoms also reported more severe PTSD total symptoms and PTSD re-experiencing symptoms and were more likely to report major depression. This apparent association between re-experiencing and pain severity in cross-sectional studies suggests that pain could be either a somatosensory intrusion or a reminder of the
traumatic event that provokes emotional and physiological reactivity. Because of the
design of the studies, however, causal interpretation is not possible.

Turning to burn patients, there is some limited evidence of a relationship between
avoidance and pain severity. In a study of 180 hospitalised burn patients sought to
explore the relationship between acute pain and PTSD symptom clusters (Difede, Jaffe,
Musngi, Perry, & Yurt, 1997). Consistent with the general pain literature showing
disproportional relationships between objective findings and pain reports, total body
surface area burned did not contribute significantly to subjective pain ratings. Although
pain and IES scores were correlated, the correlation with intrusive and avoidant
symptoms did not persist after controlling for general distress. A gender interaction
revealed, however, that while the absence of an effect was true of men, IES avoidance
scores superseded general distress as a more powerful predictor of pain amongst the
small group of women.

On the other hand, other studies have identified a role for hyperarousal. For
example, pain interference was found to be a better predictor than the presence of pain
per se for each symptom cluster of PTSD, especially hyperarousal (Asmundson, Wright,
& Stein, 2004). Similarly, a further study found that hyperarousal accounted uniquely
for the variance associated with health problems in female veterans, above the
contributions made by other symptom clusters (Kimerling, Clum, & Wolfe, 2000).

Thus, while the above studies demonstrate general support that PTSD impacts on
pain severity, the specific mechanism of operation has not been clearly established.
One argument is that the link between pain severity and PTSD simply indicates that
individuals with PTSD have worse injuries. Evidence on this point is examined below
in Section 3.7.

3.4.3 Trauma History and Pain-Related Problems

Several researchers have raised the possibility that relationships between a history
of trauma exposure and chronic pain do not just involve effects on the presence or
severity of pain but also on the way in which one experiences and copes with pain. For
example, patients with a history of abuse displayed more maladaptive responses to
chronic pain, including catastrophising (Fillingim et al., 1999), greater functional
disability (Scarinci et al., 1994), less resourceful coping, a reduced sense of control and
greater psychological distress (Toomey et al., 1995). Higher disability and lower physical activity was also reported by fibromyalgia patients who first experienced pain after injury, surgery or illness compared to patients who reported a gradual onset (Greenfield, Gitzcharles, & Esdaile, 1992). Patients with posttraumatic headaches also reported more frequent pain and had a poorer prognosis than patients with nontraumatic headaches (Tsushima & Stoddard, 1990).

To examine the issue of how pain onset may affect adaptation, Turk and Okifuji (1996) selected a group of 63 patients from 158 consecutive referrals to a university pain management centre. Participants were divided into two groups according to whether their pain resulted from a specific physical traumatic event (injury, accident or surgery, \( n = 37 \)) or was of an insidious nature (presumed if patients could not identify a specific precipitant, \( n = 26 \)). The type and sites of pain were heterogeneous. The trauma group reported higher pain levels, more interference by pain and greater affective distress than the insidious-onset group, although physical examination (of only 16 and 14 individuals, respectively) revealed no actual group differences (Turk & Okifuji, 1996). Selection of patients was dependent on them not having sought compensation, as compensation status was found to have a significant effect on psychological distress, reported pain and life interference. Excluding patients seeking compensation may under-represent significant findings, given that more traumatised patients could be more inclined to bring about a claim on the basis of the perceived severity of their condition. Indeed, a larger percentage of participants with traumatic onset (66%) were seeking compensation compared to those with an insidious onset (33%). Alternatively, potentially maladaptive beliefs and hypervigilance may account for differences between compensated and non-compensated patients (Turk & Okifuji, 1996).

Spertus, Burns, Glenn, Lofland and McCracken (1999) studied the effect of prior exposure to trauma on 73 patients with chronic musculoskeletal pain attending an outpatient rehabilitation program. Patients were administered the Trauma History Questionnaire, an instrument assessing the prevalence of a range of traumatic events, including crime, general disaster and trauma, and non-consensual physical and sexual experiences, during childhood and adulthood. An interviewer confirmed whether the traumatic event met DSM-IV Criterion A for PTSD (but did not assess PTSD). Patients were then classified into one of two groups: low trauma (no or only one traumatic event) or high trauma (more than one traumatic event). The key outcomes measured
were pain severity, general activity, affective distress, depression (Beck Depression Inventory) and pain-related anxiety symptoms (PASS). The results showed a significant interaction between trauma group and gender for the measures of affective adjustment. Univariate analyses indicated that males allocated to the high trauma group had significantly higher scores for affective distress, pain-related anxiety, and depression, compared to males in the low trauma group. No such effect of trauma was found for females, nor was there a difference for either gender on pain severity or activity measures.

Although Spertus et al.’s (1999) findings suggest prior exposure to trauma may influence affective components of chronic pain rather than pain severity and disability, some caution in interpretation is warranted. First, the study was unusual in finding greater reports of emotional distress in males compared to females, thereby raising questions as to whether results may be generalised. Second, trauma was defined broadly, determined by exposure to a traumatic event, not the actual experience of posttraumatic symptoms, which may or may not follow from exposure, as discussed above. This, coupled with the grouping of individuals who had no prior history of trauma with those who had experienced one traumatic event, clouds understanding of the relationship between trauma and chronic pain.

3.4.4 Posttraumatic Stress and Pain-Related Problems

A study of workers making compensation claims failed to differentiate pain-related variables based on PTSD diagnoses alone (Asmundson, Norton, Allerdings, Norton, & Larsen, 1998). The researchers assessed 139 consecutively injured workers referred to a tertiary-care rehabilitation program. Based on responses to the modified Posttraumatic Stress Symptom Scale (Falsetti, Resnick, Resick, & Kilpatrick, 1993), 35% of patients met the criteria for PTSD and a further 18% were classified as having partial PTSD. No differences were recorded, however, between the three groups for pain duration or use of analgesics.

In contrast to this null result, studies classifying participants in terms of their posttraumatic stress reactions offer evidence of maladjustment, generally similar to the findings outlined in Section 3.4.4 based on trauma exposure. These signs of maladjustment include elevated pain severity, depression, affective distress, disability, health care utilisation and dysfunctional coping. Classification based on PTSD rather
than trauma exposure alone and the similarity of findings across different pain populations, including heterogeneous pain complaints, orofacial pain, fibromyalgia, traumatic brain injury and hand pain, lead to greater confidence in the existence of the hypothesised relationship. Nonetheless, as discussed below, many of the studies continue to suffer from methodological limitations demanding further research is required.

Two studies have examined the relationships between posttraumatic stress, pain, affective disturbance and disability in patients of a multidisciplinary pain centre (Asmundson et al., 2000; Geisser, Roth, Bachman, & Eckert, 1996). Geisser et al.’s (1996) classified pain patients into three groups based on their trauma experience: patients reporting high posttraumatic symptoms after an accident, patients reporting low/no posttraumatic symptoms after an accident and patients whose pain was unrelated to an accident. Chronic pain patients reporting significant posttraumatic symptoms reported greater pain severity and more depression and general psychological distress than the other groups. Both accident groups reported greater disability in voluntary living tasks than the non-accident pain group and the high posttraumatic symptom group also reported greater disability in mandatory living tasks than the other two groups.

Two notable limitations stand out in this study that relied on a median split of scores on the Posttraumatic Chronic Pain Test (PCPT) (Muse & Frigola, 1986) to distinguish patients' level of reported posttraumatic symptoms. First, the scale itself is problematic, lacking adequate published data to support its validity as a single measure of PTSD. Aside from this study, no data has been published on the PCPT since the original publication, which reported adequate consistency and reliability but was based on two samples of 13 and 20 (only 10 of whom in total were diagnosed with PTSD). Claimed validity in the original study was based only on comparisons with a four-way classification of the degree of PTSD (none, mild, moderate, severe) rated by two interviewers. No comparisons with other psychometric measures were made and no normative data was provided. Second, Geisser et al.’s classification based on a median split (a score of 9) is concerning, given that a cut off of 3 correctly classified patients as

2 The published article refers to PTSD, however, see the comments, below, about problems with assessment measures.
having PTSD or not in the original PCPT study. Geisser et al.’s results therefore may be contaminated by an inappropriate classification between high and low/no PTSD.

Asmundson et al. (2000) examined the experience of PTSD in a sample of 115 chronic pain patients attending a multidisciplinary rehabilitation centre for treatment of work-related injuries. Seventy four patients were classified as either dysfunctional, interpersonally distressed or minimisers/adaptive copers according to their responses to the West Haven-Yale Multidimensional Pain Inventory (WHYMPI) (Kerns, Turk, & Rudy, 1985). Posttraumatic stress symptoms were assessed with the Modified PSSS, which indicated PTSD was relatively common in this sample (35% met criteria for full PTSD, 21% met criteria for partial PTSD, although “diagnosis” was based only on self-report). Further, PTSD was more prevalent amongst dysfunctional copers (71%) when compared to their interpersonally distressed (43%) or minimising/adaptive (21%) counterparts. Dysfunctional copers scored higher on an overall measure of PTSD (frequency and severity) and on each of the PTSD symptom clusters (i.e. B, C and D). In addition, although all three groups reported similar levels of general fears, global anxiety sensitivity and fear of somatic sensations, dysfunctional copers were more inclined to fear cognitive and emotional dyscontrol and reported more depressive symptoms than patients classified as either interpersonally distressed or minimisers/adaptive copers. This is consistent with the role of a sense of helplessness in the development of PTSD (Criterion A(2), DSM-IV TR). Although causal attributions are precluded by the cross-sectional nature of the study, patients with comorbid diagnoses of chronic pain and PTSD were clearly at risk of poor adaptation to chronic pain.

The prevalence and impact of PTSD (specifically diagnosed) on ratings of pain, affective distress, and other variables was examined, this time in patients presenting for treatment of facial pain (Sherman, 1997). Two hundred and fifty consecutive patients were approached, 180 consented to participate and 141 (56%) provided complete measures. Assessment using the SCID determined a lifetime diagnosis of PTSD in 23% of patients, 16 of whom currently met criteria for PTSD. Subsyndromal PTSD was detected in a further 12 patients, 6 of whom reported current symptoms. In light of findings that suggest an elevated risk of developing PTSD in females than in men (R. C. Kessler et al., 1995; Norris, 1992), the prevalence rate reported by Sherman (1997) may be inflated by the gender bias evident in his sample (158 females: 22 males), although it
is noted that the gender ratio in his study is consistent with a general acknowledgment that females are over-represented amongst pain patients.

Sherman (1997) classified patients into three groups: patients with current full or partial PTSD, patients with lifetime (but not current) full or partial PTSD and those without significant PTSD symptomatology. The results indicated that PTSD tends to affect the cognitive-affective aspects of pain rather than sensory aspects. Current PTSD symptomatology affected VAS ratings of pain severity and unpleasantness but had no influence on sensory descriptors chosen to describe pain. Further patients with PTSD received higher scores for affective distress (MPQ) than patients with lifetime or no experience of PTSD symptoms, whom generally did not differ from each other. They were also more likely to be identified as having a dysfunctional coping style, according to the WHYMPI, compared to those without PTSD. Patients with current or lifetime PTSD scored higher on WHYMPI affective distress compared to patients with no history of trauma. Lifetime PTSD was also associated with a perception of having less control over life, relative to those with an absence of PTSD symptoms. No differences were seen in the number of treatments sought by patients across groups, however patients with lifetime PTSD reported having more medical tests than those with no history of PTSD symptoms.

Although Sherman's (1997) categorisation is more suitable than the one used by Geisser et al., (1996), regression analyses in addition to analyses of variance (or covariance) are required to ascertain the nature of PTSD influence. It is possible the effect is responsive to symptom dose (i.e. PTSD severity) rather than a diagnostic (or threshold) effect. Further, the study did not distinguish whether current PTSD symptoms coincided with injury onset or were the result of an unrelated traumatic event (cf. Geisser et al., 1996). Indeed, only two participants suffered PTSD due to work-related industrial accidents; the remainder had symptoms secondary to the holocaust, domestic abuse or childhood physical or sexual abuse. This point demands clarification when considered in light of findings in the literature regarding specificity of responses in pain patients.

The effect of posttraumatic stress symptoms has also been considered in 93 consecutive patients with fibromyalgia (Sherman, Turk, & Okifuji, 2000). PTSD-like symptoms were relatively common amongst these fibromyalgia patients, with 56% of
the sample reporting clinically significant symptoms on a crime-related PTSD scale. Such patients reported greater levels of pain, depressive symptomatology, overall affective distress, life interference and disability than the patients who did not report significant posttraumatic stress symptoms. Significantly less patients reporting posttraumatic stress symptoms were classified as adaptive copers (15%) compared to those who did not report posttraumatic stress symptoms (48%), again suggesting that posttraumatic stress symptoms may impact on adaptation to chronic pain.

Another study examined the association between chronic pain and PTSD in 96 patients who had sustained a severe traumatic brain injury (TBI) (R. A. Bryant, Marosszeky, Crooks, Baguley, & Gurka, 1999). Six months after discharge from hospital, TBI patients were assessed for PTSD using the PTSD Interview and completed various outcome measures. More patients with chronic pain reported PTSD than did those without chronic pain. That is, 37% of the 62 patients with chronic pain met criteria for PTSD, compared to only 15% of the 38 patients without pain. Although pain severity did not differ between those with or without PTSD, greater severity of PTSD was associated with greater pain severity. In addition, PTSD severity played a mediating role in the relationship between pain severity and several variables. That is, once the effect of PTSD severity was partialled out, the relationship between pain severity and emotion-focused coping styles, depression, health problems, low community integration and satisfaction with life were no longer significant. Only the relationship between pain severity and avoidant coping remained significant. The correlational data precludes causal inference, making it impossible to tease out whether the associations reflect the influence of chronic pain on PTSD, the influence of PTSD on chronic pain, or a dynamic interdependence between the two. Cognitive deficits directly associated with the TBI, a potential confound, were not controlled.

An investigation of injury flashbacks one month post-injury also showed that subjective perceptions of injury severity rather than actual injury severity were important in determining physical outcomes of traumatic injury (Grunert, Devine, Matloub, Sanger, & Yousif, 1988). The sample comprised of 61 patients who were treated for serious hand injuries, including amputation, deformity, scarring and functional loss. Flashbacks were classified according to the nature of the image: replay flashbacks (replaying events as they happened immediately preceding the accident and up to the point of injury); appraisal flashbacks (seeing an image of the injured hand
immediately after the trauma) and projected flashbacks (seeing images of an injury worse than was actually sustained). The most common flashbacks were appraisal/projected (48%) whereby the person sees an image of a more severe injury. Replay flashbacks were the second most common (34%), while replay/projected (13.1%) flashbacks, whereby the person re-experiences the events leading up to the injury as well as an injury more severe than what was actually sustained were least common. Patients reporting only replay flashbacks required fewer sessions of psychological treatment and were most likely to return to previous work duties (95%) compared to those with replay/projected (56%) or appraisal/projected (10%) flashbacks. Indeed, the type of flashback predicted return to work better than the physical nature of the injury. According to Grunert et al. (1988), their results suggest that replay flashbacks are part of the normal process of recovery, whereas appraisal/projected and replay/projected flashbacks appear to be maladaptive responses to injury. This is consistent with Ehlers and her colleagues’ findings that individuals who attach dysfunctional meaning to intrusions are more likely to experience enduring PTSD symptoms (Ehlers & Steil, 1995; Steil & Ehlers, 2000). Grunert et al. (1988) argued that the distorted flashbacks foster a perceived lack of control and fear of reinjury that promotes avoidant behaviour, thus impacting upon recovery from injury. This is an important possibility to consider with traumatised chronic pain patients.

The only study to have investigated the impact of PTSD on chronic pain patients’ responses to acute pain experiences, using experimental control, reported mostly null results (Spertus, 2000). All participants were currently seeking treatment for chronic pain and had experienced a traumatic event that included pain. Pain duration was highly variable, ranging from 1-2 years up to more than 15 years. Participants were classified as having PTSD (n =10) or not (n = 17), using the SCID. Where there was more than one traumatic event, PTSD assessment focused on the most traumatic event. Thus, six individuals classified (for the purpose of the study) as not having PTSD actually met criteria for partial PTSD (n = 3) or criteria for PTSD to traumatic events that did not induce pain (n = 3). Only 14 patients attributed their current pain to their traumatic experience and the report did not specify the distribution of these patients between the two groups. Even when controlling for this problem of cloudy classification, no significant differences were recorded between the PTSD and no-PTSD groups on cardiovascular measures of physiological arousal (i.e. heart rate and blood pressure), self-reported distress or avoidance in response to pain-induced during a cold-pressor
task. Although these findings did not support a link between PTSD and responses to pain, this may have been affected by a number of limitations of the study, including the focus on experimentally induced pain, which was of an acute duration and non-specific to both the individual’s experience of trauma and the normal pain site(s). Questions about the effects of PTSD in relation to the acute experience of pain in chronic pain patients thus remain open, given findings of reactivity to individual, fear-specific (but not general) cues of pain-related threats (Flor, Birbaumer et al., 1992; Flor et al., 1985) and possibly confounded by the partial PTSD members of the control group.

3.5 Co-morbidity between Accidental Traumatic Injury and PTSD

This section highlights the higher rates of PTSD found in individuals assessed after an (objectively) traumatic injury.

Published estimates of the prevalence of PTSD following accidental or traumatic injury cover the full spectrum from 1 to 100%, the diversity obviously dependent on methodological differences. A comprehensive review is presented elsewhere (O'Donnell, Creamer, Bryant, Schnyder, & Shalev, 2003). Examples of issues with methodology include the timing of assessment, choice of assessment measures (e.g., self-report instruments versus clinical interview), reliance on formal diagnosis versus high symptomatology, changes in diagnostic criteria between DSM-III, DSM-III-R and DSM-IV (American Psychiatric Association, 1980, 1987, 1994) and failure to control for potential confounds (e.g., traumatic brain injury, use of narcotic analgesics). The population from which participants are drawn is also a relevant consideration. The more conservative estimates generally appear in community-based and prospective studies, less dependent on treatment seeking patients than cross-sectional studies of clinical patients.

3.5.1 Estimates Based on Consecutive Hospital Attendees

3.5.1.1 From 1 to 6 months post-injury

The prevalence of PTSD assessed between 1 and 6 months after accidental or traumatic injury ranges from 8% to 42%. Amongst a group of 48 patients who underwent surgery for fractured legs, the prevalence of PTSD at 6 weeks was 25% (based on a self-report checklist of DSM-III-R symptoms), declining to 15% at 6 months (Feinstein & Dolan, 1991). At 1 month, 30% of injured patients recruited from
the emergency room of a general hospital met diagnostic criteria for PTSD but only
18% had PTSD at 4 months (Shalev, Freedman et al., 1998). Three months after an
MVA leading to attendance at an emergency department, Ehlers et al. (1998) reported a
PTSD rate of 23% in consecutive attendees. Other research assessed severely injured
patients admitted to an Australian casualty ward after an MVA (M. M. Green,
McFarlane, Hunter, & Griggs, 1993). Structured interviews conducted after 3 months
revealed 2 of the 24 patients had PTSD (8%) and 7 had partial PTSD (29%). Notably,
the sample size was small and represents just over half of the eligible participants.

On the basis of a structured diagnostic interview, 26% of patients met DSM-III-R
criteria for PTSD at 6 months after presentation to an emergency department for
moderate injuries (Shalev, Peri, Canetti, & Schreiber, 1996). Notably, the development
of PTSD appeared unrelated to the type of traumatic event, across survivors of MVAs,
work and other accidents, terrorist acts and physical assaults. One study indicated that
the rate of PTSD was 24% 6-months after a traumatic brain injury (R. A. Bryant &
Harvey, 1998). Six months after admission to hospital for accidental injury, the rate of
PTSD was assessed as 42% (Michaels et al., 1999).

3.5.1.2 At 12 months or more post-injury

Twelve months or more after an injury the prevalence of PTSD is generally lower,
although estimates vary from 1% to 36%. The lowest rate arises from a prospective
study of patients who attended the casualty department of a Norwegian hospital after
accidental injury (Bendixen et al., 1994). Almost half of the injuries were due to an
MVA and most individuals had sustained mild to moderate injuries. The study relied on
clinical interviews and self-report questionnaires administered on initial presentation
and again around 28 months later (range 15 to 51 months). Only 1 of the 107 patients,
an MVA survivor, was diagnosed with PTSD. Three others were diagnosed with partial
PTSD. Self-reported symptomatology was much higher, with 21% and 44% endorsing
significant intrusions and avoidance, according to scores on the IES. Similarly, the rate
of PTSD was only 2% in a study conducted 12 months after severe injury requiring
admission to ICU (Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001a). Of the
patients followed up at 18 months by Green et al. (1993), 30% had developed full
PTSD, which had not been diagnosed or treated before. This study is thus unusual in
showing an increased rate of PTSD over time.
By 12-month follow-up, the rate of PTSD amongst Ehlers et al.’s (1998) injured patients had dropped to 16%. Several other studies indicated that approximately one third of individuals had PTSD after one year (Blanchard, Hickling, Mitnick et al., 1995; Koren, Arnon, & Klein, 1999; Zatzick et al., 2002) and 18 months (M. M. Green et al., 1993). The latter study is of interest as it represented an increase, rather than a decrease, in the rate of PTSD over time.

By comparison, rates of PTSD recorded following burn injuries, which cause severe pain and psychological distress, are relatively stable over the first year. Between 29% and 41% of patients reported clinically significant PTSD symptoms during hospitalisation for burns (Patterson, Carrigan, Robinson, & Questad, 1990; Perry et al., 1987) and a similar percentage of burn patients met diagnostic criteria for PTSD 12 months post injury (R. A. Bryant & Harvey, 1996; Difede & Barocas, 1999; Perry, Difede, Musngi, Frances, & Jacobsberg, 1992; Powers, Cruse, Daniels, & Stevens, 1994).

3.5.2 Estimates Based on Individuals Seeking Tertiary Treatment

Typically, higher rates of PTSD are reported when individuals are selected on the basis of their subsequent presentation for secondary or tertiary assessment or treatment. In one study, the entire sample of 30 clinical patients, referred for medico-legal opinion or treatment following an MVA injury, met DSM-III criteria for PTSD (Kuch, Swinson, & Kirby, 1985). Interviews conducted from 1.3 and 7 years post-MVA in a group of 56 people referred for psychiatric assessment, all of whom were litigating, supported a diagnosis of PTSD in 32% of cases (Dalal & Harrison, 1993).

Selection bias is typically blamed for the relatively inflated rates of PTSD reported by such studies. By contrast, there are indications that a primary pain presentation may cloud detection of PTSD after injury. For example, 29% of individuals who had been referred for treatment of chronic headaches after an accident were diagnosed with PTSD and a further 20% met partial criteria for PTSD (Chibnall & Duckro, 1994).

Importantly, none of these patients had been diagnosed previously with PTSD. It is possible that this reflected delayed onset PTSD, however, it is also plausible that other treatment providers, presumably the majority of whom were physically-oriented, had failed to detect PTSD symptoms.
3.5.3 Estimates Based on Community Samples

Widening the scope to a US community sample of 1000 adults, MVAs were found to be the third most common lifetime stressor, compared to nine other potential stressors such as fire, rape, assault and robbery (Norris, 1992). Moreover, MVAs resulting in injury were the most common causes of PTSD in the community. Based on a structured interview, 12% of respondents who reported experiencing an MVA in their lifetime currently met the criteria for PTSD. A Dutch study also recorded high rates of post-traumatic stress symptoms in a non-treatment-seeking sample (Brom, Kleber, & Hofman, 1993). This questionnaire-based study of individuals who had had an MVA, as identified from police records reported high IES scores for 22% of the sample at 1 month and 15% at 6 months. Although Brom et al. (1993) estimated a prevalence rate of 10% for PTSD, this can only be considered a rough estimate as the response rate for the study was very low (20% of eligible participants) and the IES is not diagnostic of PTSD.

3.5.4 Limits of Prevalence Data

Although simple prevalence data is useful in linking injury and PTSD, it overlooks vital information about the dynamic course of problems after traumatic injury and limits our understanding of any substantive relationship between PTSD and dynamic post-event factors, such as adjustment to injury and chronic pain. A prospective study in the UK, which considered the outcomes for MVA survivors over the course of 5 years, highlights this problem (Mayou, Bryant, & Duthie, 1993; Mayou, Tyndel, & Bryant, 1997).

Participants were 188 consecutive hospital attendees, seeking treatment for multiple injuries and whiplash and were included if they had been unconscious for less than 15 minutes after the accident. The initial assessment and follow-up at 3 and 12 months used the Present State Examination, a semi-structured interview based on DSM-III-R criteria (Mayou et al., 1993). Five years after their MVA, 111 participants completed a postal survey or telephone interview (Mayou et al., 1997). PTSD symptoms were assessed using an abridged version of the Posttraumatic Stress Symptom Severity scale (PSSS) (Foa, Riggs, Dancu, & Rothbaum, 1993).

3 This forms an interesting parallel with reports that MVAs are common causes of injuries that proceed to chronic pain.
The prevalence of PTSD assessed at 3 months, 12 months and 5 years was 8% on each occasion (i.e. 11, 8 and 9 participants, respectively) (Mayou et al., 1993). The apparent stability in the incidence of PTSD is misleading, however, masking stark variations in course (Mayou et al., 1997). Five of the 33 participants with acute symptoms were symptom free at 12 months, while 8 went on to develop full PTSD. Only 8 of 177 participants met PTSD criteria at both 3 and 12-month time points and only 1 male met criteria across all three time points. Five participants exhibited time-limited PTSD, meeting criteria at 3 months but not 12 months, while 6 participants exhibited delayed PTSD, meeting criteria at 12 but not 3 months. Four of the 5 participants diagnosed with PTSD at 12 months had improved by 5 years. Of the 9 participants with PTSD at 5 years, 8 had a delayed-onset of PTSD, not having previously met criteria for the disorder.

Although comparisons should be cautious as different measures were used to ascertain PTSD up to 12 months and at 5 years, the variation in course gives rise to questions about what factors might be involved in change over time. As will be discussed below, adjustment to injury and chronic pain may be two such factors.

3.6 The Unique Contribution of Injury versus Trauma Exposure to the Development of PTSD

Research conducted with survivors of disaster, victims of crime and combat veterans suggests that actually sustaining an injury during a traumatic event is related to the development of PTSD. At the most superficial level, elevated rates of PTSD are understandable in the context of injury, if one defines a traumatic stressor on the basis of it involving actual or threatened injury (as per DSM-IV TR). In other words, if all else is equal, it is reasonable to assume that an event involving actual injury has a greater chance of being perceived as traumatic than an event that does not involve injury.

Support for this claim is evident in research demonstrating increased rates of PTSD in injured compared to non-injured survivors of the Pittsburgh Regatta disaster (Martini, Ryan, Nakayama, & Ramenofsky, 1990). Higher rates of PTSD were also reported for victims of crime who were injured compared to those who were not injured (Kilpatrick
et al., 1989) and physical injury during rape predicted PTSD in 54 rape victims (Bownes, Gorman, & Sayers, 1991).

Further, the prevalence of PTSD in injured veterans (20%) was reported to be more than twice the rate found in uninjured veterans (9%) (R.K. Pitman et al., 1989) and, conversely, a group of veterans diagnosed with PTSD after exposure to Agent Orange were more likely to have had combat injuries than were controls who had been exposed to Agent Orange but did not develop PTSD (Solkoff, Gray, & Keill, 1986). In addition, a survey study of 489 Vietnam veterans seeking treatment for substance abuse found that injury predicted PTSD (McFall, Mackay, & Donovan, 1991). Physical injury also predicted PTSD in another survey study of 84 Vietnam veterans (Buydens-Branchey, Noumair, & Branchey, 1990).

A comprehensive matching procedure was used to isolate the unique contribution of physical injury to the development of PTSD in Israeli soldiers (Koren, Norman, Cohen, Berman, & Klein, 2005). Sixty injured soldiers were matched with 40 uninjured soldiers according to rank, military role, length of service, pre-injury medical status and importantly, involvement in the same combat event. Injury severity was assessed during initial hospitalisation using the Injury Severity Score (ISS) and clinical interviews took place on average 15.5 months post-injury. Based on the SCID, 10 of the injured soldiers (17%), but only 1 of the uninjured soldiers (2.5%) met diagnostic criteria for PTSD, representing an eight-fold increase in the risk of developing PTSD in the injured sample. A further 3 injured soldiers (5%) but none of the uninjured soldiers, exhibited partial PTSD. Based on the CAPS, injured soldiers exhibited higher levels of total posttraumatic stress symptoms (i.e. re-experiencing, avoidance and hyperarousal all combined) compared to uninjured soldiers and this effect was independent of PTSD status per se.

Of further interest is the study’s finding that the combination of injury and PTSD differentially affected the pattern of posttraumatic symptoms. Amongst those without PTSD, hyperarousal was relevant, with injured soldiers displaying significantly higher hyperarousal scores than uninjured soldiers. By contrast, amongst those with PTSD, re-experiencing was relevant, with injured soldiers displaying higher re-experiencing symptoms than uninjured soldiers. Koren et al. (2005) concluded from these results that
injury, first, increases hyperarousal irrespective of PTSD and, second, has an effect through re-experiencing symptoms for those who develop PTSD.

Overall, these studies suggest that injury makes a unique contribution to the development of PTSD, over and above the contribution of trauma exposure per se.

**3.7 THE RELATIONSHIP BETWEEN INJURY SEVERITY AND TRAUMA-RELATED SYMPTOMS OR PTSD**

Evidence of increased pain severity amongst patients with PTSD (see Section 3.4.2) may represent a real effect or, perhaps, the possibility that individuals with PTSD have more severe injuries. Prima facie, it is logical to reason that an individual is more likely to be traumatised following a severe injury rather than a mild or moderate injury. Researchers seeking to explore this possible effect have used a range of study designs including prospective, treatment outcome and cross-sectional studies. Perusal of the literature turns up conflicting evidence, however, for the existence of a relationship between injury severity and the severity of trauma reactions and outcomes. While some evidence supports a higher rate of PTSD in severely injured individuals, several researchers have found no such effect. First, it will be argued that one possible explanation for this disparity relates to the differing emphases on objective versus subjective indicators of the original injury. Second, it will be argued that subjective experiences of, and responses to, the ongoing consequences of injury (e.g., chronic pain) have been largely overlooked. Most researchers have tied injury to the original traumatic event, viewing it as a static peri-traumatic factor, and have directed little research toward how injury sequelae, such as chronic pain, might operate as post-event factors influencing the maintenance of, or recovery from, trauma (Ehlers & Steil, 1995).

Most studies assess injury severity at one time point (in the immediate aftermath of the accident), employing an objective measure, typically the Abbreviated Injury Scale (AIS) and its derivative, the Injury Severity Score (ISS), which are rated by medical practitioners according to a standardised scale. The ISS is based on the three most severely injured regions of the body, with each region objectively rated from 1 (minor) to 6 (untreatable, virtually unsurvivable), resulting in a maximum possible score of 18.
A score of 1-3 includes soft tissue injuries (e.g., bruising, abrasions, lacerations) up to minor fractures (e.g., broken finger). It will be argued here that using objective measures, such as the ISS and AIS, as indicators of injury severity is problematic given findings outlined in Chapter 2 about the lack of correlation between injury (i.e. tissue damage) and injury sequelae (i.e. pain, distress and impairment).

3.7.1 Objective Measures of Injury Severity and PTSD

The empirical evidence is quite divided as to whether a relationship exists between objective injury severity and posttraumatic symptom severity or PTSD. A large body of research has demonstrated a positive link, however, a similarly large body has shown no such effect.

3.7.1.1 Studies Demonstrating an Effect of ISS/AIS

As part of a large-scale project conducted in the UK (Epidemiology of Major Trauma in Mersey Region and North Wales Study) (Piccinelli, Patterson, Wilkinson, Boot, & Braithwaite, 1999), individuals with severe injuries were followed up 5 years after experiencing a traumatic event. The extent of injury was determined initially using the ISS, with 349 patients surviving “severe” traumatic injuries. Five years later, broad psychological data was obtained for one third of these individuals. Self-reported anxiety and depressive disorders were common and related to more severe injuries at baseline. The ISS was an inefficient predictor of anxiety and depressive disorders however, accounting for only 30% of variance in a logistic regression controlling for age and gender.

An epidemiological survey of survivors of terrorist bomb blasts in France demonstrated a connection between injury severity and the more stringent criteria of a PTSD diagnosis (Abenhaim, Dab, & Salmi, 1992). Typical injuries included “blast syndrome”, burns and coma, whereas amputations were less common. This study indicated that the severity of physical injury predicted the development of PTSD, with 31% of severely injured individuals reporting PTSD compared to only 9% of moderately injured victims and 10% of uninjured victims. Another study considered an array of accidental injuries and found a significant relationship between the severity of injury and the development of PTSD (Malt, Hoivik, & Blikra, 1993). The group who developed PTSD represented only a small proportion of participants (~5% of 192 MVA victims). Thus, although these results are interesting, it would be unwise to place too
much reliance on a study of only 10 cases. A dose-response effect was also highlighted in a large, representative sample of injured veterans, in which combat injuries requiring hospitalisation were found to significantly predict the presence of PTSD problems at a later age (Card, 1987).

A team of investigators, headed by Blanchard and Hickling, conducted a series of studies (the Albany Motor Vehicle Accident Research Project) investigating the psychological sequelae of MVAs in survivors who sought medical treatment soon after their accident (Barton, Blanchard, & Hickling, 1996; Blanchard, Hickling, Barton et al., 1996; Blanchard et al., 1997; Blanchard, Hickling, Mitnick et al., 1995; Blanchard, Hickling, Taylor et al., 1996; Blanchard, Hickling, Vollmer et al., 1995). The initial prevalence of PTSD and partial PTSD ranged from 39-41% and 26-29%, respectively, at 1-4 months post-accident (Blanchard, Hickling, Taylor et al., 1996; Blanchard, Hickling, Vollmer et al., 1995).

This group replicated a link between pain severity and PTSD across several studies. For the first study, significant correlations occurred between injury severity (AIS) and the development of posttraumatic stress symptoms and PTSD diagnosis (Blanchard, Hickling, Mitnick et al., 1995). Moreover, the researchers found a dose specific effect, whereby patients diagnosed with full PTSD obtained higher AIS ratings. In combination with ratings of fear of death, the AIS score was a modest predictor of posttraumatic stress symptom scores, predicting 12% of variance. Nonetheless, the AIS score showed only moderate sensitivity and specificity as a predictor. Individuals with an AIS score of 6 or more had a 62% chance of having PTSD, although 58% of those with PTSD scored 5 or less on the AIS. In another study, four variables (extent of physical injury, fear of dying, prior major depression, and a decision to pursue litigation) correctly predicted 70% of the patients as either having PTSD (57%) or not (78%) (Blanchard, Hickling, Taylor et al., 1996). The improved sensitivity and specificity may have occurred due to the inclusion of a broader range of injuries, as selection was based on presentation not admission to hospital. After litigation, the most important predictor of PTSD was the extent of objective physical injury (AIS), based on standardised regression weights.

Blanchard and his colleagues (Blanchard & Hickling, 1999; Blanchard, Hickling, Barton et al., 1996; Blanchard et al., 1997) reported a 55% remission rate for the PTSD
group and 67% for the sub-syndromal PTSD group over the next 6 months. Four variables seemed to predict remission, including the initial PTSD-symptom severity, initial injury severity (AIS score), relative physical recovery by 4 months and (absence of) subsequent family trauma during the intervening period. The physical recovery measure was obtained from self-ratings on a 4-point scale, ranging from completely remitted to remains the same (Blanchard et al., 1996, p.778). Here, the relevance of the AIS points to an association between extent of physical injury and the onset and maintenance of PTSD; whereas the physical recovery measure links physical injury sequelae (and potentially pain variables) to the maintenance of PTSD.

3.7.1.2 Studies Demonstrating a Weak or Absent Effect of ISS/AIS

By contrast, a range of other studies has failed to replicate a link between injury severity and trauma-related symptoms or PTSD. No relationship was found between injury severity (AIS) and trauma severity (IES) in a group of patients admitted to a hospital trauma unit after accidental injury (Landsman et al., 1990). Feinstein and Dolan (1991) also reported equivalent ISS across 48 orthopaedic surgery patients with and without PTSD, though this is not surprising given all patients selected had experienced a fractured femur, tibia or fibula, truncating the range of injury severity. The presence of PTSD also bore no relationship to ISS in US and Australian samples of hospitalised MVA survivors (Epstein, 1993; M. M. Green et al., 1993). The samples were again small (n = 15 and 24, respectively) and truncated by selecting only patients with severe injuries.

The same problem of restricting the sample to severely injured individuals may explain why a Swiss study of consecutive ICU admissions again identified no relationship between injury severity (ISS) and PTSD at 12 months post injury (Schnyder et al., 2001a; Schnyder, Moergeli, Trentz, Klaghofer, & Buddberg, 2001b). The initial and 12-month prevalence of PTSD of 4.7% and 2%, respectively, was also much lower than rates reported above. In addition, the initial ISS did not significantly correlate with subjective appraisals of the event in terms of the degree of death threat and accident severity. This might be due to the ceiling effect created by using only the severely injured or could reflect a real disparity between objective and subjective ratings.
Findings from a 12 month follow-up study in Israel also did not support a link between objective injury severity and PTSD at 1 year, though 32% of the traffic accident victims with mild to moderate physical injuries suffered from PTSD (Koren et al., 1999). The study can be distinguished on a number of levels. First, although the authors indicated that patients were eligible if they sustained mild to moderate injuries, patients were selected from those admitted to an orthopaedic ward for at least two days. Hospital admission speaks to a reasonably high severity of injury and is likely to exclude patients who may only sustain soft tissue injury. Similar problems are evident in the 3-year follow-up of this sample (Koren, Arnon, & Klein, 2001). Objective measures of injury severity (AIS ratings and yes/no classification of accident-related medical complications) were equivalent across those patients who continued to experience PTSD compared to those who had recovered.

A significant relationship between objective injury severity and a higher risk of PTSD at 1, 3 and 6 months post-accident was also absent in another study recruiting from a trauma centre and local police reports (Ursano et al., 1999). Nonetheless, the trend was in the expected direction as 29% of injured individuals had PTSD at 3 months, whereas no cases of PTSD were reported amongst non-injured individuals. The high refusal rate (50-75%) might have influenced the failure to reach significance.

Again, injury severity was not clearly correlated with trauma-related symptoms in a study of 109 individuals hospitalised in three urban trauma centres following moderate to serious injury (Richmond & Kauder, 2000). Levels of trauma-related symptoms were measured using the IES during acute hospitalisation and 3 months post-discharge. Initially, severe levels of psychological distress were reported by 32% of patients, while a further 15% reported moderate levels. By the time of follow-up, the numbers of patients reporting psychological distress increased to 50% at the severe level and 17% at the moderate level. ISS ratings of injury severity were again not predictive of subsequent IES scores. Two methodological limitations suggest caution in interpretation: a low participation rate (48% of eligible participants) and a staggered time frame for follow-up. Determined on time since discharge, differing lengths of hospitalisation meant follow-up varied from 3 to 6 months post-injury, a period during which remission is common.
In a study that recruited 101 survivors of MVAs or assaults admitted to the surgical ward of a University of California medical center, no link was found between initial injury severity and PTSD at 1, 4 and 12 months (Zatzick et al., 2002). The ISS and PTSD Checklist – Civilian Version were used to measure symptoms. Claims of representative selection are undermined in this study however, as 76% of the 397 patients initially considered declined to participate (n = 39) or were excluded or not approached because participation would be prevented by their lack of English, severity of injury or physical impairments, early discharge or transfer, surgery, participation in a pilot study or miscellaneous reasons (e.g., incarceration).

In a study matching injured and uninjured combat veterans, injury severity was not predictive of the development of PTSD (SCID) or the severity of posttraumatic symptoms (CAPS) (Koren et al., 2005). Notably, the response rate was only moderate (51%) and injured soldiers were disproportionally represented amongst those who refused participation.

3.7.1.3 Reasons for the Inconsistent Findings Regarding Injury Severity and PTSD

Aside from the methodological problems discussed in relation to specific studies, the apparent inconsistency in this area may derive no: so much from a tenuous link between injury and PTSD, as from a choice of variables mis guided by faulty assumptions. While it appears logical to assume that a more severe injury would be associated with greater threat and consequently more severe PTSD, a focus on the objective nature of injury severity is problematic as it is well recognised that the individual’s subjective appraisal is critical in the development of PTSD (American Psychiatric Association, 1994; Ehlers & Clarke, 2000; B. L. Green, 1994; March, 1993). The AIS and ISS are independent clinical ratings of severity, having regard to objective characteristics of the injury and knowledge of usual prognosis. From the patient’s point of view however, an assessment of injury severity may be made according to features readily accessible to them, such as visible ‘damage’ or pain. Another study found that PTSD is more likely to occur in cases where victims sustained highly visible injuries (e.g., bruises, lacerations, open wounds) or when irreparable damage was done to the vehicle (S. Taylor & Koch, 1995). Similarly, injury and pain are not isomorphic — pain does not necessarily bear a proportional relationship to the extent of structural or tissue damage detected (Melzack, 1990; see Chapter 2). Finally, traditional injury rating measures (such as AIS and ISS) have been shown to grossly under-predict the
consequences of orthopedic injury, such as bodily pain, physical function, physical and emotional role, mental health, social function and impact on work (Michaels, Madey, Krieg, & Long, 2001). Indeed, high levels of acute psychological distress have been recorded for emergency patients with mild physical injuries (Joy, Probert, Bisson, & Shepherd, 2000) and burn patients with objectively small burns (<1% of body surface area) (Tedstone & Tarrier, 1997). For these reasons, subjective and dynamic factors related to injury need to be considered with respect to PTSD.

3.7.2 Subjective Measures of Injury Severity, Pain and Physical Recovery
Self-rated measures of injury severity, pain and physical recovery may bear closer associations with the development and maintenance of trauma.

3.7.2.1 Motor vehicle accidents
Returning to Koren et al.’s (2001) study of accidental injuries, which found no evidence of a link between objectively rated injury severity and PTSD, patients who had not recovered from PTSD gave higher subjective ratings of accident-related pain (measured on a 101-point VAS) compared to recovered patients. Although the trend was not statistically significant, the power to detect a significant relationship was compromised by small cell sizes (10 and 9 patients), suggesting that further investigation is warranted.

Pain was implicated in a study of 107 patients with whiplash admitted consecutively to a Norwegian emergency department (Drottning, Staff, Levin, & Malt, 1995). Initial assessment occurred only hours after admission and a significant correlation was found between a VAS rating of acute pain and IES scores. The study’s strength lies in the high response rate of 93% at follow-up 4 weeks later, when 42% of patients reported significant persistent pain. Seventy per cent of the patients reporting high pain at 4 weeks had high IES scores at initial assessment, compared to only 26% of the low pain group, suggesting an interaction between trauma symptoms and the maintenance of pain (although the time-frame means such pain would still be regarded as acute rather than chronic). Interestingly, neither initial pain variables (neck pain or stiffness, headache, interscapular pain) nor the velocity of impact predicted the persistence of pain at 4 weeks.
In Australia, 56 individuals were assessed 12 months after they were hospitalised for MVA-related injuries (R. A. Bryant & Harvey, 1995). Objective measures of trauma severity (ISS and length of hospitalisation) were unrelated to the presence of trauma-related symptoms (IES), whereas subjective ratings of trauma severity and avoidant coping styles were associated with high ratings of intrusive symptoms at 12 months. Interestingly, participants who reported psychological trauma also reported more pain. The strength of these findings, however, is undermined by the low response rate (43%) perhaps reflecting avoidance itself (Weisaeth, 2001) and the use of a restricted sample, bound by a minimum of 24 hours hospitalisation, which possibly relates more to objective rather than subjective factors.

Differences between objective and subjective measures of injury and pain are borne out in a series of studies conducted by the Oxford group (Ehlers, Mayou, & Bryant, 1998; Mayou & Bryant, 2001, 2002; Mayou, Ehlers, & Bryant, 2002; Mayou et al., 1997). This project centred on a prospective study of 967 consecutive patients presenting to an Oxford hospital accident and emergency department after an MVA. Follow-up assessments were conducted with 888 patients at 3 months, 718 patients at 12 months and 546 patients at 3 years post accident. The severity of injuries in the sample ranged from bone injuries (20%) to soft tissue injuries (60%), so only 26% of participants required hospital admission and 20% of participants sustained no injury at all. Analyses of drop outs showed that patients who were admitted to hospital or who had bone injuries were more likely than their less seriously injured counterparts to participate for the duration of the study. Based on PSSS scores at 3 months, 23% of participants met DSM-IV criteria for PTSD. After 1 year, the rate declined to 16.5% of participants and 11% at 3 years. Approximately half of those diagnosed with PTSD at 3 months continued to suffer from the disorder at 1 year, with a similar proportion continuing to suffer from PTSD symptoms at 3 years.

The study investigated risk factors for the development of PTSD, including pre-morbid factors (i.e. gender, prior health status and emotional functioning) and peri-traumatic variables (i.e. injury severity, perception of threat during trauma, dissociation). In addition, the study sought to identify post-event factors that might promote the maintenance of symptoms. The study thus considered post-event psychological factors (negative interpretation of intrusions, rumination, thought
suppression and anger) and concurrent problems (persistent financial and medical problems, and litigation).

Objective measures of injury severity were not significant predictors of PTSD. By contrast, persistent medical problems, as rated by the patient, greatly increased the likelihood of a PTSD diagnosis and higher symptom severity. Indeed, 55% of patients who reported major medical problems at 3 months and 74% of patients who reported major medical problems at one year met criteria for PTSD. Similarly, at 1 year, persistent medical problems were associated with the delayed onset of PTSD that occurred in 6% of patients. Persistent medical problems at all three time points showed moderate correlations with PTSD diagnosis and symptom severity at 3 years. Stepwise regression analyses indicated that (self-rated) persistent medical problems at 1 year was the strongest predictor of PTSD severity 3 years after the accident. Persistent medical problems, in combination with negative interpretation of intrusions, rumination, thought suppression, anger cognitions, financial problems, dissociation and being female, accounted for 39% of variance. Furthermore, persistent health problems, litigation and negative interpretations of intrusions at 1 year, explained variance in PTSD severity at 3 years, over and above what could be explained by initial and 1 year PSS scores. While these findings support a relationship between PTSD and injury adjustment, the study relied on a single item. It would therefore be prudent to conduct a more detailed investigation of this effect. A more recent prospective study by the Oxford Group (Murray, Ehlers, & Mayou, 2002) replicated the finding that injury severity measured by the AIS was a poor predictor of PTSD severity, whereas patients’ own ratings of injury severity correlated significantly with PTSD severity at 4 weeks and 6 months.

Mayou et al., (2002) argued that persistent medical and financial problems are chronic stressors, which deplete coping resources as well as acting as reminders of the event. It is reasonable to expect that persistent medical problems are likely to contribute to financial problems (via work disability and treatment costs), moderating the relationship between financial problems and PTSD. In addition, persistent medical problems are likely to increase the frequency of reminders of the accident and reinforce negative interpretations such as ‘I will never get over it’ and ‘Others have harmed me’. Arguably, the perception of persistent medical problems would maintain a sense of current threat (Ehlers & Clarke, 2000; Mayou et al., 2002).
The covariation of physical problems and PTSD symptoms in Mayou et al.'s (1993, 1997) study is also worth reviewing to illuminate the role of subjective post-event variables. After 5 years, 49 participants (44%) reported a full physical recovery. None of these participants were diagnosed with PTSD. By contrast, at 5 years, 44% of current PTSD patients (4/9) reported major physical problems compared to 7% of patients (7/102) without PTSD. Patients with current PTSD at 5 years were also more likely to have had a poor medical outcome at 12 months (57%, 5/9) compared to those without PTSD (17%, 17/102). Conversely, patient reports of continuing medical problems at 5 years significantly predicted PTSD at 5 years. Moreover, there appeared to be a dose response effect. Of the 51 participants who reported ongoing minor physical problems, 9 (18%) had minor PTSD and 5 (10%) had major PTSD; whereas for the 11 participants reporting major physical problems, 1 had minor PTSD (9%) and 4 (36%) had major PTSD. This pattern of results is interesting as physical status was not predictive of PTSD at the 12-month assessment. Given the moderate response rate (64% at 12 months) and relatively small cell sizes, the relationship between ongoing physical problems and PTSD begs further investigation.

Another report on the same sample (Mayou & Bryant, 2001) elucidates further the need to consider subjective ratings of injury and recovery. Most injuries (61%) were recorded as minor on objective, clinical measures. Based on current medical knowledge and epidemiological data outlined in Chapter 2, one would expect that most participants would recover from their medical problems within 3 months. However, upon follow up, 45% of the sample reported continuing physical problems, 22% reported moderate to very severe pain and 17% reported moderate to extreme impairment of daily activities. Seriously injured patients (requiring at least three nights in hospital) had poorer physical, psychological and social outcomes, most notably higher rates of PTSD and pain and poorer physical recovery. However, the objective type of injury (none, soft tissue, bone) was largely unrelated to outcomes as, for example, a significant subset of patients with mild or no injuries reported continuing physical problems. On the other hand, ratings of physical recovery and psychological outcomes were significantly correlated. Further, an initial perception of threat was associated with both a greater risk of developing PTSD and a greater risk of reporting moderate to very severe pain at follow-up.
From these results, Mayou & B. Bryant (2001) concluded “the association between physical and psychological variables suggests that psychiatric complications affect perception of pain and impairment and that interim physical problems may maintain post-traumatic symptoms” (p. 533). The role of psychological factors in pain perception is further reinforced by the fact that the 23% of participants who suffered serious injury continued to have worse physical outcomes but did not report greater pain than those with minor injuries (or, conversely, people with minor injuries did not report any less pain than people with serious injuries) (Mayou & B. Bryant, 2002). Indeed, none of the baseline injury variables predicted pain levels at 3 years, reinforcing the point that injury is not isomorphic with pain (see Chapter 2) and should not be confused as a post-event variable.

The Bendigo Road Accident Research Project lends further support to the disparity between objective and subjective ratings of injury and links the latter with the development of PTSD (Jeavons, 1999, 2000; Jeavons, Greenwood, & De L. Horne, 2000). The Bendigo Project involved a prospective study of 72 MVA survivors who initially attended a regional hospital in Victoria, Australia. Participants were re-assessed at 3, 6 and 12 months. The sample differed from other studies by including the breadth of injury severity, from minor (61% self-rated using a 0-3 scale, 80% doctor-rated, taken from case notes), through moderate (28% self-rated, 10% doctor-rated) to severe (11% self-rated, 10% doctor-rated) injuries. Although the doctor and self-ratings were moderately correlated, the different proportions highlight a disparity between objective and subjective ratings, especially at the minor end of the scale. Although the frequency of injuries self-rated and doctor-rated as severe were similar, about 20% of people identified by their doctors as having only minor injuries actually viewed their injuries as moderate.

Trauma-related symptoms were measured in the project by the IES and the Posttraumatic Stress Disorder Interview (PTSD-I, based on DSM-III-R criteria). Using the PTSD-I, criteria for PTSD were met by 8% of individuals at 3, 6 and 12 months. It is not clear from the published data whether the same cases received a diagnosis of PTSD at each time point, although the researcher has since indicated that they were not always the same (Jeavons, 2001, personal communication). Survivors’ perception of life threat was significantly correlated with both the IES and PTSD-I scores at 3 and 6 months.
Mixed results were obtained regarding the effects of injury severity on posttraumatic symptoms. Consistent with research based on conventional injury severity indicators (above), doctor-ratings were not correlated with IES or PTSD-I scores at any time point. However, other indicators of injury severity appeared to have a delayed effect. The length of hospital admission was unrelated to PTSD symptoms until 12 months, when moderate correlations with trauma symptoms were detected. Interestingly, correlations between self-rated injury severity and 12-month IES and PTSD-I scores were moderate.

Additional analyses of predictors of PTSD were also restricted by sample size. Variables were initially entered in groups and re-entered in the final model if found to be significant. Risk factors differed between the IES and PTSD-I, across time and in the proportion of variance predicted. Perceived threat to life, expectation of injury and emotion-focused coping significantly accounted for 34% of variance in IES scores and 38% of PTSD-I scores at 3 months. At 6 months, self-rated injury, maladaptive coping (emotion-focused coping, avoidance in the form of social diversion), thinking one might die, distress at the time of the accident and an assumption of benevolence significantly accounted for 49% of variance in IES and 77% in PTSD-I scores. Self-rated injury did not feature in predictive models at 12 months. Rather, length of hospital admission, fear during the accident and emotion-focused coping accounted for 26-45% of IES variance, whereas emotion-focused coping, length of hospital admission, accident distress and whether or not the patient was a driver or passenger during the accident accounted for 44-58% of variance in PTSD-I.

The inconsistency with other studies showing positive relationships might also relate to the range of injury severity included in studies, with relationships less apparent when samples are restricted to severe injuries or objective ratings. In addition, Jeavons (2000) argued that inconsistent evidence regarding the role of injury severity may be due to the research time frame, suggesting that injury severity may become more important in the longer term. While this possibility was reflected in correlations between self-rated injury and trauma scores at 12 months, injury severity did not enter a regression model predicting either IES or PTSD-I. Nonetheless, the argument is supported by other research on two samples of MVA survivors (Steil & Ehlers, 2000). This found that the perceived probability of death or severe injury due to the accident,
as rated from the present perspective (an average of 6.1 years post-accident), was more strongly correlated with PTSD severity and intrusion distress than the probability of death or severe injury perceived at the actual time of the accident. Certainly, enduring problems with pain may contribute to interpretations that the injury sustained in the accident was severe.

The impact of injury severity and pain on the development of ASD has also drawn attention (Fuglsang, Moergeli, Hepp-Beg, & Schnyder, 2002). The research involved a cross-sectional study of a randomised sample of accident victims admitted to a Swiss trauma surgery ward for more than two nights. The 323 patients who participated were interviewed and completed questionnaire measures, on average, 5 days after their accident. A low incidence of ASD was found, relative to other studies. Using DSM-IV criteria, 4% (13 patients) exhibited ASD, while a further 10% met a sub-syndromal diagnosis (inconclusive on only one criterion). As with other studies, a prior history of psychiatric problems and a measure of stress resilience and coping predicted ASD, as well as two objective accident-related variables indicative of injury severity, involvement in a traffic accident and stay in intensive care. In line with recent findings (Mayou & Bryant, 2001, 2002; Mayou et al., 2002; Murray et al., 2002), subjective perceptions and cognitions of not only the accident (consistent with the A(2) stressor criterion) but also its sequelae were important predictors, more so than pre-accident variables. The relevant factors included the patient’s self-ratings of the threat of death, accident severity, others’ ability to prevent the accident and future ability to recover physically, as well as a VAS rating of pain.

3.7.2.2 Combat veterans

Although case reports have identified instances of PTSD arising from the experience of severe, uncontrolled pain (Cundy, 1995; Schreiber & Galai-Gat, 1993), pain as a variable itself has been largely overlooked in studies following up veterans after traumatic injury. An 8-year follow up study, recruited 44 war-wounded refugees, during their initial hospitalisation (Hermansson, Thyberg, Timpka, & Gerdle, 2001). Seventy three percent reported chronic pain 8 years later and the mean VAS ratings of the intensity of pain was significantly correlated with the frequency of psychiatric symptoms.
3.7.2.3 Burn injuries

Comparatively more research has been conducted on the relationship between acute pain and trauma in the context of burn injuries, however it is not clear whether findings can be extrapolated to chronic pain. In a study of 43 hospitalised burn patients, ratings of prior adjustment suppressed the relationship between pain and PTSD, measured using a DSM-III-R checklist (Ptacek, Patterson, Montgomery, & Heimbach, 1995). The partial correlation between pain and PTSD only became significant after prior mental health adjustment was taken into account (Ptacek et al., 1995). This finding suggests there may be a common underlying factor to explain the association between acute pain and PTSD.

The findings of one group were less supportive of a relationship between pain and PTSD, however, in their investigation of the effect of morphine on the development of PTSD in a sample of 24 children with burns (Saxe et al., 2001). Morphine doses (averaged over the course of hospitalisation) were significantly correlated with changes in PTSD at 6 months follow-up, such that higher morphine doses were related to a reduction in PTSD symptoms. One explanation of these results could be that morphine controlled pain, causing a reduction in PTSD symptoms. Two findings were raised to indicate that this was not the case, however. First, pain reported during acute assessment (2-26 days post-admission) was unrelated to change in PTSD at 6 months. Second, the partial correlations between morphine dose and change in PTSD remained significant, when controlling for the percentage of body surface area burned (injury severity) and pain. Saxe et al. (2001) offered an alternative explanatory pathway for the effects of morphine: that morphine affected the operation of norepinephrine on the amygdala, thus countering the facilitative effects of the neurotransmitter on fear conditioning and memory consolidation. Rigorous, experimental investigation would be required to further explore these effects and overcome the study’s methodological flaws, however, before firm conclusions are drawn. Most notably, morphine dosages were determined according to usual treatment practices and were not subject to experimental manipulation. Presumably, reported pain levels would be one factor in the clinical decision of how much morphine to administer, yet pain was assessed at only one time point for the study, the timing of which was not constant across patients. Further, it would arguably have been better to enter change in pain over time rather than using a static measure of pain to test whether pain or morphine moderated the effect.
Finally, as the participants were children it is not clear how well the results would extrapolate to an adult population.

The limitations of correlational research were circumvented in a time series analysis of burn patients (Taal & Faber, 1998). Patients’ ratings of pain were recorded five times a day across the first two weeks of hospitalisation (i.e. acute pain). Strong rank correlations were found between ratings of acute pain and scores on the IES and the Burn Specific Pain Anxiety Scale (BSPAS), which measured anxiety related to acute pain and treatment procedures, none of which were correlated with burn size. However, a fourth-order partial rho between pain perception in the second week and post-traumatic stress was close to zero once the effects of age, burn size, BSPAS and pain in the first week were partialled out. Taal and Faber (1998) concluded that the relationship between pain and PTSD is indirect, moderated by the degree of anxiety measured by the BSPAS.

Overall, the reviewed studies are equivocal, at best, regarding the impact of objective injury severity on the development and maintenance of PTSD. By contrast, the bulk of studies investigating the role of the individual’s subjective perception of physical injury, pain and recovery in the development and maintenance of trauma-related symptoms and PTSD are more consistent and suggest a promising direction for further research. Indeed, greater exploration of pain-related constructs, as reviewed in Chapter 2, is necessary to elucidate whether such constructs mediate the relationship between traumatic injury and PTSD.

3.8 Effects of Comorbidity on Treatment Outcome

Further support for exploring subjective, long term characteristics of physical injury, such as chronic pain, arise from the few treatment outcome studies that have addressed the effects of injury status and chronic pain on individuals who undergo treatment for PTSD after a traumatic injury. Similarly, the presence of PTSD also appears to have an effect on treatment outcomes for those undergoing treatment for chronic pain.
One such study relied on a sample of 81 MVA survivors, comprised of 48 seeking treatment at a rehabilitation centre for chronic pain and 33 seeking treatment for PTSD induced by the collision (Fedoroff, Taylor, Asmundson, & Koch, 2000). Analysis of data obtained from the combined sample indicated that pain severity and anxiety sensitivity significantly predicted scores on the PTSD Symptom Severity Scale. Further investigation of the 28 patients who completed a 12-week CBT treatment program for PTSD, revealed that reductions in anxiety sensitivity, pain severity and belief in the dangerousness of road travel significantly predicted a reduction in post-treatment PTSD symptoms severity. These findings are consistent with earlier case studies indicating that pain interferes with the success of CBT treatment for PTSD (Koch & Taylor, 1995). The mechanism and direction of causality underlying the link between reduced pain severity and reduced PTSD are not clear.

An earlier report on the same sample discussed the relationship between post-MVA stressors and PTSD treatment outcome (Koch, Shercliffe, Fedoroff, Iverson, & Taylor, 1999). Preliminary data obtained from 28 participants found moderate correlations between mean PTSD scores, based on the Penn PTSD scale and a self-report Posttraumatic Stress Scale, and several injury-related variables, including slow physical recovery, pessimism about physical recovery, and self-reported number of health care appointments. As Koch et al. (1999) argued, their data promotes the prominence of ongoing injury- and pain-related variables as needs to be addressed in treatment. These post-event variables made a unique contribution to the maintenance of trauma symptoms; a contribution over-looked by the narrow, conventional research focus on event variables (e.g., initial injury severity) and pre-disposing factors (e.g., prior psychological problems), and clinical focus on treating one problem over another or sequentially.

A further study explored the factors associated with different patterns of treatment outcome in 50 MVA survivors with PTSD (S. Taylor et al., 2001). Patients recruited by advertisement or physician referral completed 12 weeks of group CBT. Only 8% of participants were diagnosed with Pain Disorder, however 94% had current MVA-related injuries and “most” reported some form of recurrent pain. Cluster analyses revealed two groups of patients: responders and partial responders. Compared to responders, partial responders reported significantly greater pain severity, were more likely to have comorbid major depression, and had lower global levels of functioning at pre-treatment.
Pain severity and major depression each accounted for 17% of the variance in treatment outcomes between groups. There was also a non-significant trend (p value between .01 and .05) for partial responders to report greater pain-related interference in daily activities (an indication of pain-related disability), which accounted for 9% of variance in outcomes. As Taylor et al. (2001) pointed out, depression and pain-related variables appear related to achieving only a partial response to treatment for PTSD, although the design of this study does not allow causal attributions to be made. The results therefore beg further exploration of the nature of the relationship between chronic pain and PTSD.

Gillespie and colleagues reported on the effects of individual, community-based CBT treatment following a car bombing (Gillespie, Duffy, Hackmann, & Clark, 2002). This research is important as significant treatment effects here are of greater importance in the absence of the typically selective criteria for inclusion in experimental clinical trials. The 91 participants included individuals who were physically injured in the bombing, individuals who were present at the bombing but not injured, and emergency service workers involved in the aftermath. The three groups did not differ on initial ratings of PTSD symptoms (Posttraumatic Stress Diagnostic Scale, PDS), (Fo, Cashman, Jaycox, & Perry, 1997), however differences emerged in the degree of improvement following treatment. Physically injured individuals improved significantly less than individuals who were not injured and emergency workers. The median improvement for each group was 59%, 77% and 85%, respectively. Gillespie et al. suggested that ongoing physical problems and medical interventions probably served as reminders of the negative effect of the bombing, resulting in negative interpretations which in turn maintain symptoms of PTSD.

Findings from a randomised, controlled study of psychological debriefing in consecutive road traffic accident victims are more difficult to interpret in terms of the influence of pain on treatment for PTSD (Mayou, Ehlers, & Hobbs, 2000). First, some difficulties arise in generalising the results as the intervention and non-intervention groups differed with respect to baseline extremity injury severity and length of hospital stay. Once these factors were controlled, however, patients who received psychological debriefing reported more pain and less physical recovery at 3 years than patients who did not receive the intervention. As initial IES scores had no effect on either pain or physical recovery, Mayou et al. (2000) suggested that something about the process of
intervention per se must have directly contributed to the maintenance of these problems. However, the intervention group sustained not only high levels of pain but also high IES scores, whereas these were reduced in the non-intervention group. Although the intervention may have contributed to this, it is also arguable that the effects are due to an interaction between pain, physical recovery and trauma symptoms, resulting in the maintenance of symptoms.

A connection between childhood abuse and responses to chronic low back pain also appears in a study that evaluated 473 consecutive patients who completed a functional restoration program for chronic pain (McMahon, Gatchel, Polatin, & Mayer, 1997). History of childhood abuse, determined using a structured interview, was not related to patients’ initial response to treatment, based on measures of physical capacity and successful completion of the program. Patients with such a history, however, reported greater psychological distress, were subsequently less likely to maintain at work and more likely to have more post-program surgery to the injured site, when compared to patients who did not reveal a history of childhood abuse. McMahon et al. (1997) suggested a history of abuse increases the likelihood of relapse as maladaptive behaviours are more resilient to change and are likely to be resurrected.

An interaction between PTSD and treatment for chronic pain is also illustrated by two early case series (Hickling, Blanchard, Silverman, & Schwartz, 1992; Muse, 1986). Hickling et al. (1992) discussed the treatment of 20 patients with post-traumatic headache, 10 of whom also had PTSD. For the headache only group, patients responded well to cognitive behavioural treatment, which included training in relaxation, biofeedback and exposure techniques. Patients with both headache and PTSD needed significantly longer treatment, and responded to headache treatment only after undergoing treatment for PTSD, suggesting that PTSD is causal in at least the maintenance of chronic pain. Muse’s (1986) case series also highlighted the need for additional treatment to resolve patients’ PTSD after completion of treatment for pain.

On the contrary, the influence of PTSD on treatment outcomes was examined in a group of chronic low back pain patients with quite different results (Bovasso, 1993). Three hundred and thirty four patients attending a 3-week functional restoration program were assessed using the SCID (DSM-III-R). Females were more likely to receive a diagnosis of PTSD than were males, and females with PTSD had a higher
lifetime prevalence of other Axis I and II disorders. Despite their more complicated clinical presentation, however, functional indicators did not appear to differ in females with PTSD relative to other females. At discharge, no differences were found between groups on levels of strength, range of motion, nor on self-ratings of pain and disability. Follow-ups conducted 6 months later found patients with PTSD were, however, more likely to have sought treatment for their injury in the intervening period than were patients without PTSD. No association was found, however, between PTSD and return to work, reinjury, post-discharge surgery or use of addictive medication.

### 3.9 Summary

Although findings are not always consistent, the overall weight of evidence reviewed above is persuasive of a relationship between chronic pain and PTSD.

At the basic level of comorbidity, chronic pain patients are more likely than the general population to have a history of trauma or PTSD, regardless of whether research is conducted within a treatment-seeking setting or the wider community. Conversely, PTSD is more prevalent amongst individuals injured during a traumatic event than amongst those individuals who were not injured.

A history of trauma, particularly abuse, has been associated with greater pain intensity and reduced pain threshold in some cross-sectional studies, although other studies have found no such effect. While prospective studies also support a lack of association between objectively defined abuse and pain severity, when the research instead considered subjective reports of victimisation the link re-appeared. The inconsistencies may be due to methodological issues and questionable assumptions of cause in cross-sectional studies, however, the role of subjective interpretation and specific posttraumatic reactions actually may be central to the effect. More consistent findings of a positive relationship between pain severity and PTSD (cf. trauma) bolster this claim. Further, all three of the symptom clusters have been shown to correlate with pain severity, but the pattern is not consistent across the predominantly survey-based studies. In fact, there is a paucity of research examining the direct effect of PTSD on chronic pain in real time. These difficulties mean discussion of results and underlying mechanisms has fallen mostly to speculation.
The existing body of research further suggests that trauma exposure and PTSD may have a greater influence on cognitive-affective responses to pain and issues associated with behavioural adjustment rather than directly influencing the sensory characteristics of pain itself. That is, as reviewed above, there is strong evidence that individuals with comorbid PTSD and chronic pain are more distressed, depressed and disabled and prone to dysfunctional coping than their non-traumatised counterparts.

Conversely, research operating from the standpoint of PTSD has attempted to draw links between injury severity to aid the prediction of PTSD development and long-term maintenance. Objectively defining injury severity produces equivocal results to the extent that there are as many studies supporting the link as rejecting it. Self-rated injury severity, pain and estimates of physical recovery show more promise in predicting the maintenance of PTSD and the severity of symptoms, particularly over the longer term. The data typical stem from large-scale prospective studies that necessitate a narrow conception of these factors. There is thus a need to explore the connection in a context permitting a deeper, more focused analysis of subjective injury sequelae, such as chronic pain. Experimental manipulation would thus be a useful alternative to cross-sectional or prospective research to the extent that it facilitates improved control and depth of analysis.

Finally, individuals with comorbid chronic pain and PTSD respond less well to treatment (particularly if it is unimodal in its focus) and are more likely to relapse after treatment. The complex interaction of symptoms could be one reason for poor treatment outcomes. Another reason could be that existing treatments are ineffective due to their failure to address the synergy of the conditions. Most of the research on treatment has been generated by evaluations of specific treatment protocols, however, very little is known about the general level of understanding amongst health professionals about comorbidity or how pain patients with PTSD are currently treated under circumstances of usual care.

Whilst the current literature provides interesting insights, an absence of theoretically driven studies limits firm conclusions about the nature of the relationship between PTSD and chronic pain, especially when considering the range of methodological issues raised in review. The next chapter thus sets out theoretical models that will be used to guide subsequent empirical studies.
CHAPTER 4

THEORETICAL EXPLANATIONS OF THE RELATIONSHIP BETWEEN CHRONIC PAIN AND PTSD

Chronic pain and PTSD frequently co-occur, resulting in a complex clinical presentation and negative outcomes. As already outlined in Chapter 3, the concurrence of the two conditions has been associated with reports of pain severity, PTSD severity, affective distress (i.e. general negative affect, depression), pain-related anxiety, disability, dysfunctional coping and health care utilisation, all elevated above levels reported by individuals with the singular presentation of PTSD or chronic pain. Many of the investigations providing these findings have been essentially exploratory, designed without reference to a clear theoretical framework. In the absence of a unifying theory, the data are often correlational, not systematic or poorly controlled and the discussion of underlying processes post-hoc and speculative. This impediment to clinical understanding restricts the development of effective treatments and the opportunity for early intervention to divert a chronic course.

Several theoretical models have been proposed to account for the nature of the relationship between chronic pain and PTSD, addressing both aetiological and maintaining factors. Although biological theories are acknowledged for their valuable insights into possible mechanisms (van der Kolk & Saporta, 1993), the following section emphasises recent psychological models (Asmundson et al., 2002; Sharp & Harvey, 2001).

4.1 Mutual Maintenance Model

Sharp and Harvey (2001) proposed a “mutual maintenance” model to explain the relationship between chronic pain and PTSD. According to this model, the two conditions could perpetuate each other through several layers of overlapping and or reciprocal processes. In particular, Sharp and Harvey hypothesised seven specific mechanisms by which mutual maintenance could occur:

1. Attentional bias: Individuals with chronic pain may have cognitive biases towards painful and threatening stimuli (as reviewed in Chapter 2) and
individuals with PTSD may have attentional biases towards threatening stimuli (R. A. Bryant & Harvey, 1995a; Cassiday, McNally, & Zeitlin, 1992; R. McNally, Kaspi, Riemann, & Zeitlin, 1990). Therefore, individuals with PTSD may have an attentional bias to pain sensations as threat-related cues, amplifying the pain experience.

2. Anxiety sensitivity: Anxiety sensitivity – evident in individuals with chronic pain (see Chapter 2) and individuals with PTSD (S. Taylor, Koch, & McNally, 1992) – may predispose individuals to misinterpret or catastrophise about bodily sensations associated with pain and arousal.

3. Reminders: Pain may be a persistent reminder of the traumatic event, triggering an arousal response. This may lead to avoidance of the cause of pain and any memories of the trauma. Through this process of mutual maintenance, distress and disability escalate.

4. Avoidance: Avoidance may be adopted as a means to minimise pain and anxiety in both chronic pain (see Chapter 2) and PTSD (R. A. Bryant & Harvey, 1995). Initially this may be adaptive, however, prolonged avoidance may contribute to the maintenance of symptoms through either physical deconditioning and disability or failure to activate, process and resolve the networked representation of the trauma stored in memory.

5. Depression: Fatigue, lethargy and physical inactivity associated with depression may contribute to the maintenance of chronic pain (through increased disability) and PTSD (limited exposure to trauma-related stimuli, necessary for activating, processing and resolving the fear network).

6. Anxiety: As an anxiety disorder, PTSD may directly affect an individual's perception of pain severity, which is known to increase under conditions of anxiety. Higher perceived pain may lead to reduced activity levels, increased disability and distress.

7. Limited cognitive capacity: Cognitive symptoms of pain (e.g., catastrophising) and PTSD (e.g., re-experiencing symptoms) may drain the individual's cognitive capacity, thereby limiting resources available to initiate and sustain adaptive coping strategies.

These ideas are not new, however, the model is important for advancing clear and testable hypotheses under the concept of mutual maintenance. Asmundson et al. (2002) criticised the model as overly simplistic, however, and suggested that not all of the
multiple dimensions of PTSD and chronic pain (cognitive, affective, behavioural, and physiological) may be causally related.

Although they do not do so themselves, it is feasible to parse Sharp and Harvey's predictions of mutual maintenance into four types of causal relationships. The true nature of the relationship between PTSD and chronic pain could involve the combination of all four pathways, as Sharp and Harvey suggest. Alternatively, only one or some might be involved (cf. Asmundson et al., 2002). The four causal relationships are as follows:

4.1.1 A Unidirectional Effect of PTSD on Pain

From hypotheses 1 and 6, PTSD could be a unidirectional causal factor increasing the perception of pain, via hypervigilance and hyperarousal, respectively. Two separate longitudinal studies support at least the direction of this causal link between pain and posttraumatic symptoms, finding that initial scores on the IES predicted subsequent VAS ratings of pain at 1 month Drottning et al. (1995) and moderate to severe whiplash symptoms at 6 months (Sterling et al. 2005). A small experimental study indicated that PTSD symptoms can result in analgesia during subsequent episodes of induced pain (Pitman et al., 1990), although the ability to generalise from this study is questionable as the participants (veterans) were otherwise apparently pain-free. The same proposed mechanisms of effect have been raised by a number of researchers (Andreski et al., 1998; Scarinci et al., 1994; Sherman, 1997; Turk & Okifuji, 1996), however, existing data does not permit conclusions about cause, due to methodological issues (e.g., study of singular not comorbid presentation, cross-sectional/correlational data). Experimental studies showing that stress-induced physiological reactivity produces hyperalgiesia in response to cold pressor and ischaemia tasks are peripheral, too, in that they have not involved the specific group of chronic pain patients with PTSD (Bruehl, Carlson, & McCubbin, 1992; Caceres & Burns, 1997; France & Stewart, 1995; Peckerman et al., 1991).

4.1.2 A Unidirectional Effect of Pain on PTSD

Based on hypothesis 3, pain could be a unidirectional causal factor maintaining PTSD, through its operation as a threat-related cue and reminder of the traumatic event. Similar arguments have been advanced by others (Beckham et al., 1997; Koch & Taylor, 1995; Koren et al., 2005; McFarlane et al., 1994; Spertus, 2000; White &
Faustman, 1989). The reasoning draws upon the work of Foa and her colleagues (Foa & Riggs, 1993; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989), who explained the development and maintenance of PTSD in terms of the formation of a fear network. After exposure to a traumatic event, they proposed, information about what is threatening and to be avoided is stored in memory as an associative network. Activation of this fear network occurs via cued-recall, resulting in strong cognitive, affective, behavioural and physiological responses, which mirror responses that occurred at the time of the event. Over time, the cues may generalise according to behavioural principles and an attentional bias that predisposes ready activation of the fear network (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988). Foa and her colleagues argued that for recovery to occur, all aspects of the fear network need to be activated and modified during “emotional processing” to weaken associations between stimulus and response and reduce the intensity and magnitude of fear (see also (Foa & Kozak, 1986; B. Litz & Keane, 1989). When the individual engages avoidance to manage distress, avoiding thoughts and situations related to the traumatic event, the fear network is less likely to be activated fully, restricting opportunities to appropriately challenge threat-related beliefs and coping strategies (Creamer, Burgess, & Pattison, 1992). The fear network is also dependent on the subjective meaning attached to the event and subsequent sequelae (Foa et al., 1989).

It has been proposed that a painful sensation may trigger a flashback to the original injury via activation of the amygdala (R. A. Bryant & Harvey, 1995a). Although the injury may subsequently resolve, bodily movements might then continue to trigger flashbacks, which include pain memories from the original injury that are nonetheless experienced as real, current sensations. Such flashbacks or reminders of the event cued by pain are believed to result in affective distress, hyperarousal and avoidance.

The causal direction of this proposed mechanism (i.e. that pain affects PTSD) is broadly supported over a longer time-frame, by a study, which demonstrated that self-rated persistent medical problems at 1 year was the strongest predictor of PTSD diagnosis and symptom severity at 3 years (Mayou et al., 2002). Specific to the mechanism itself, PTSD re-experiencing symptoms have been linked with pain severity, although the findings are cross-sectional and cannot be used to attribute cause (Beckham et al., 1997; Koren et al., 2005; McFarlane et al., 1994). For example, the pattern of results obtained by Koren et al. (2005) supported an effect of injury via re-
experiencing in those who develop PTSD, irrespective of the hyperarousal promoted by injury. On the other hand, Spertus' (2000) study did not support the proposed causal mechanism. She found no evidence that (experimentally induced) pain specifically activated a fear-network in individuals with chronic pain and PTSD, as this group's affective, behavioural and physiological reactivity in response to pain was equivalent to the group with chronic pain only. A number of methodological limitations suggest, however, that replication is required before accepting the null result.

4.1.3 Bi-directional Relationship Between Pain and PTSD

Dynamic bi-directional relationships are proposed in hypotheses 4 and 7, whereby avoidant coping and cognitive symptoms (such as catastrophising) limit recovery opportunities in a self-perpetuating and reciprocal manner that maintains symptoms, distress and disability related to both conditions. In this case, the question of which disorder has primacy is irrelevant; rather, the focus is on current interdependence. Numerous cross-sectional studies have revealed high levels of affective distress, disability and broad dysfunctional coping amongst chronic pain patients with PTSD or a history of trauma (Asmundson et al., 2000; R. Bryant et al., 1999; Geisser et al., 1996; Sherman, 1997; Sherman et al., 2000; Spertus et al., 1999; Turk & Okifuji, 1996), however, there is a relative paucity of data regarding the interdependence of these with processes such as catastrophising, pain-related anxiety, fear of injury and avoidance.

4.1.4 Relationship Between Pain and PTSD due to a Third Variable

Finally, drawing on hypotheses 2 and 5, the apparent relationship between the two disorders may be spurious and actually dependent on a third variable (anxiety sensitivity, depression). However, Asmundson et al. (2002) argued that Sharp and Harvey’s model “confuses” the role of anxiety sensitivity by failing to distinguish that it represents a shared vulnerability relevant to onset, distinct from any subsequent capacity it has to mutually maintain conditions (p. 933; see below).

4.2 Shared Vulnerability Model

While acknowledging the possibility of mutually maintaining symptoms, Asmundson et al. (2002) specifically advanced a “shared vulnerability” model to explain the apparent interdependence of chronic pain and PTSD. As discussed earlier, in Chapter 2, anxiety sensitivity is the term given to the tendency to respond fearfully to
anxiety-related bodily sensations, based on the belief that these sensations are harmful. Asmundson et al. argued that individuals who display a high level of anxiety sensitivity are likely to respond more fearfully to traumatic stressors and or painful injuries, both of which automatically elicit physiological reactivity. In both cases, the fear of physiological arousal leads to further affective and physiological reactivity, as well as avoidance, maintaining the problem and facilitating the development of PTSD and or chronic pain. When a traumatic stressor and painful injury co-occur, the vulnerability becomes pronounced. By contrast, they argued that individuals with low anxiety sensitivity were unlikely to develop PTSD or chronic pain, as they are less likely to fear the reactivity generated by acute pain or exposure to a traumatic stressor.

Support for this model is mostly indirect, stemming from studies of the disorders as independent conditions or cross-sectional studies of injured individuals. As reviewed in Chapter 2, chronic pain patients with high anxiety sensitivity reported greater pain-related fear, negative affect and avoidance of activity than those with low anxiety sensitivity (Asmundson & Norton, 1995). Further, anxiety sensitivity predicted pain-related fear, which in turn predicted avoidance (Asmundson & Norton, 1995; Zvolensky et al., 2001) and also predicted perceptions of vitality, mental health and social functioning and use of analgesia (Plehn et al., 1998). Important to the causal placement of anxiety sensitivity as a vulnerability factor, evidence indicates that anxiety sensitivity is not dependent on the current experience of chronic pain, as it predicted pain-anxiety symptoms even in healthy individuals (Keogh & Mansoor, 2001; Muris, Vlaeyen, & Meesters, 2001). In a comparison of the DSM IV anxiety disorders, individuals with PTSD scored highly on the anxiety sensitivity index (only those with panic disorder scored more highly) (S. Taylor et al., 1992). Anxiety sensitivity also predicted PTSD symptoms in a group of women exposed to domestic violence (A. J. Lang, Kennedy, & Stein, 2002) and was associated with the severity of PTSD symptoms (Fedoroff et al., 2000). Evidence of the effect in the specific case of patients with comorbid pain and PTSD is limited to one study, which found that change in anxiety sensitivity was implicated as one of two mechanisms by which CBT reduced PTSD symptoms in individuals injured in a road accident (Fedoroff et al., 2000).

In light of the existing literature, the shared vulnerability model has logical merit, however, as yet, there is no prospective evidence of the impact of anxiety sensitivity on the development of chronic pain and or PTSD. In addition, an obvious limitation of the
model is its emphasis on quite a narrow construct, fear of anxiety-related bodily sensations, which may not address the breadth of problems observed in those with chronic pain and PTSD.

4.3 Perception of Current Threat: A Unifying Concept?

One criticism of Sharp and Harvey’s (2001) model is that the predictions are not framed within a unifying theory. It is argued that a recent theoretical model from the field of PTSD could provide this framework.

With respect to the independent literature on PTSD, Ehlers and Clark (2000) proposed a cognitive model of PTSD in which they argued that:

[P]ersistent PTSD occurs only if individuals process the traumatic event and/or its sequelae in a way which produces a sense of serious current threat ... Once activated, the perception of current threat is accompanied by intrusions and other re-experiencing symptoms, symptoms of arousal, anxiety and other emotional responses. The perceived threat also motivates a series of behavioural and cognitive responses that are intended to reduce perceived threat and distress in the short-term, but have the consequence of preventing cognitive change and therefore maintaining the disorder. (p.320)

Also important to this cognitive theory is the notion that retrieval from memory is cue-driven and unintentional, such that the individual is not always aware of the activating trigger (cf. the unexpected onset of pain). That is, PTSD symptomatology is characterised by data-driven processing rather than conceptual-processing of information that permits appropriate integration of memory, new information and meaning. Support for Ehlers and Clark’s contention that cognitive appraisal underpins PTSD has been accumulating (Brewin & Holmes, 2003; B. Bryant, Mayou, Wiggs, Ehlers, & Stores, 2004; Dunmore, Clark, & Ehlers, 1997, 1999; Dunmore, Clark, & Elhers, 2001; Ehlers, Maercker, & Boos, 2000; Ehlers, Mayou, & Bryant, 2003; Ehlers et al., 1998; Ehlers & Steil, 1995; Halligan, Michael, Clark, & Ehlers, 2003).

Applying this theory and drawing on the primacy of fear-avoidance constructs in the maintenance of chronic pain (see Chapter 2), it is argued that the relationship
between chronic pain and PTSD can be viewed within a hierarchical framework headed by a perception of current threat. The currency of threat then has a cascading effect on cognitive-affective, behavioural and physiological systems. That is, difficulties arise when patients with PTSD experience pain as an indication of current harm to self. Complex and reverberating processes across the different systems, extrapolating from Sharp and Harvey (2001), may then lead to the persistence of pain and PTSD over the longer term. Vulnerability factors, including but not limited to anxiety sensitivity, may contribute to the perception of current threat. Therefore to investigate the comorbidity of chronic pain and PTSD, an estimation of the individual current perception of threat needs to be obtained while investigating the interaction of trauma symptoms and chronic pain factors (including pain severity and disability).
CHAPTER 5

A CROSS-SECTIONAL STUDY OF DIFFERENCES BETWEEN CHRONIC PAIN PATIENTS WITH AND WITHOUT PTSD

The volume of research reviewed in previous chapters, at the very least, establishes an association between physical injury and PTSD symptoms, unsurprising given psychological trauma often involves injury. To a lesser extent, there is also evidence of an association between pain and PTSD symptoms. However, the nature and strength of these associations, beyond simple concurrence, is less clear. Compared to our understanding of the comorbidity and influence of depression, anxiety and fear on chronic pain, current knowledge of the impact of PTSD in this area is limited. Very little is known about the degree of overlap between these disorders and whether or not they are interdependent.

Although basic comorbidity data has its utility, it is worthwhile to investigate the possibility of interdependence between chronic pain and PTSD. Rachman outlined an approach to conducting psychological comorbidity research, in which he emphasised exploring the connectedness of the two conditions. He distinguished between cases of static and dynamic associations between conditions. Taking time to distinguish static and dynamic effects, for example, facilitates treatment planning and accurate prognoses, by answering the question "if problem A is treated successfully, will problem B disappear, or persist?" (p.462) (Rachman, 1991). Similarly, an argument has been mounted against taking a “Kraepelininan” classification of disorders (whereby the disorders are viewed as mutually exclusive and exhaustive) in the context of chronic pain and depression (de C. Williams, 1998). de Williams instead advocated for a phenomenological approach to related multiple experiences and overlapping symptomatology and strongly rejected continued pursuit of gross conceptions of causal sequences\(^4\) and comorbidity.

While the chronic pain and PTSD literatures have independently identified factors associated with the maintenance of each singular condition, very little is known about the functional interrelationships between the two disorders. It is likely that the

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\(^4\) An example of this question would be whether pain causes PTSD or PTSD causes pain.
relationship between chronic pain and PTSD is complex, reciprocal and contingent, with the possibility of "mutual maintenance" (Sharp & Harvey, 2001) occurring over the course of time. Contemporary literature hints at the biological and cognitive-behavioural links between pain and PTSD. Research needs to explore these connections more directly. Study of such interrelationships and effects on long-term outcomes may provide a better understanding of the specific processes responsible for the maintenance and exacerbation of chronic pain comorbid with PTSD, shedding light on interventions useful in modifying risk factors and reciprocal interactions and, ultimately, reducing the incidence, severity and duration of symptomatology.

Although the comorbidity of chronic pain and PTSD has attracted growing interest, there remains an empirical hiatus due to the methodological problems found in prior studies and the scarcity of attention to factors contributing to comorbidity. A review relevant to studies of injury and PTSD is presented elsewhere (O'Donnell et al., 2003). Existing literature in the area includes several case studies, which are interesting but may not generalise to the wider population (Lebovits, Yarmush, & Lefkowitz, 1990; Muse, 1985, 1986; Pilowsky, 1985; Schreiber & Galai-Gat, 1993). Sampling biases and methodological issues have also afflicted quantitative studies. Some are restricted by very small sample sizes (Hickling & Blanchard, 1992) and many involve selection bias, for example, recruiting only treatment-seeking individuals. In others the gender distribution is not representative (Asmundson et al., 2004; O'Donnell et al., 2003). A focus on narrow pain conditions, such as headache (Muse, 1985), orofacial pain (Sherman, 1997) and fibromyalgia (Sherman et al., 2000) or the inclusion of potential confounds, such as traumatic brain injury (R. A. Bryant et al., 1999; O'Donnell et al., 2003) also limits the generalisation of results. Still others make questionable assumptions regarding the effects of potential confounds such as litigation, in excluding individuals involved in litigation from analyses (Turk & Okifuji, 1996). Arguably, it would be useful to incorporate litigation status as a covariate in analyses, rather than creating an exclusive sample.

In addition, the causal interpretation of many studies is questionable as the link between the index trauma and the onset of pain is not clear (e.g., due to long intervening periods between the index trauma and pain onset, Geisser et al., 1996; Sherman, 1997). Therefore, it is necessary to tighten the parameters of study and, for example, consider the specific case where the index trauma and chronic pain arise from the same event.
With respect to assessment procedures, often those studies that comprehensively assessed a range of pain-related factors are limited by reliance on inadequate measures or classification of PTSD (Drottning et al., 1995; Geisser et al., 1996; Muse & Frigola, 1986; Scarinci et al., 1994; Spertus, 2000; Spertus, Burns, Glenn, Lofland, & McCracken, 1999; Toomey, Seville, Abashian, Finkel, & Mann, 1994; Turk, Okifuji, & Starz, 1996), while others that adequately assessed PTSD using accepted diagnostic criteria measured pain in only a very basic way, relying on a single or few non-standardised items which do not reflect the complexity of the pain experience (Asmundson et al., 2004; Ehlers et al., 1998; Mayou & Bryant, 2001, 2002; Mayou et al., 2002; Mayou et al., 1997). Many studies have also relied exclusively on self-report data. For all of these reasons, prevalence data varies widely and it is questionable whether one can generalise patterns of results regarding associated factors and mechanisms.

5.1 Study 1

The purpose of this study therefore was to compare and contrast differences between chronic pain patients with and without PTSD. In order to address some of the methodological problems discussed above, participants were compared in a cross-sectional analysis across multiple variables, using validated and, where possible, clinical measures. The study aimed, first, to tighten the parameters of study by exploring the relationship between PTSD and chronic pain in the specific case where they arise from the same index event. Consideration of such cases is also warranted given the high prevalence of single events leading to PTSD and chronic pain and their high associated costs. Second, the study aimed to replicate results of earlier studies that found elevated pain symptoms, health care utilisation, depression and disability in pain patients with PTSD relative to pain patients without PTSD. Third, the study aimed to determine whether PTSD is related to different responses to pain, such as elevated pain-related fear and anxiety and specific coping strategies. Fourth, the study aimed to explore the relationships between potential independent and dependent variables for chronic pain problems in the presence and absence of PTSD.
5.2 Method

5.2.1 Participants

Patients were considered eligible for the study if they were aged above 18 and had experienced a chronic pain condition for at least six months. Although the International Association for the Study of Pain defines chronic pain as requiring a duration of three months or greater, a cutoff of six months was chosen here to encompass the definition of chronic PTSD proposed by DSM-IV TR (American Psychiatric Association, 2000). Patients were excluded from the study if they (1) had serious head injury or burns; (2) major psychiatric disorder (e.g., psychosis or bipolar disorder); (3) predominant drug and alcohol abuse; (4) were pregnant; or (5) did not have sufficient comprehension of English to allow them to complete measures.

One hundred and twenty voluntary participants were recruited from patients with heterogeneous chronic pain complaints referred to multidisciplinary cognitive-behavioural pain management programs, physiotherapists and rehabilitation providers in the Australian Capital Territory\(^5\). In particular, participants were recruited from the Canberra Injury Management Centre, a private multidisciplinary pain clinic (CIMC: \(n = 61\)), the Pain Unit operated by the Department of Anesthesiology and Pain at The Canberra Hospital, a publicly funded hospital (TCH: \(n = 25\)), Canberra Physiotherapy Centre, a large independently run physiotherapy practice (CPC: \(n = 20\)) and several rehabilitation providers employed in the private sector (\(n = 19\)).

Attempts were made to obtain a sample of consecutive patients, however, it was not possible to determine the precise number of patients who were not informed about the study by staff at referring agencies, nor how many individuals declined to participate. Where known, the reasons cited for declining included being too busy, in too much pain, and (unfounded) concerns that participation might jeopardise compensation claims or pending litigation. It was considered reasonable to exclude individuals on the latter ground anyway, as predominant concerns about compensation may have influenced the veracity of self-report.\(^6\)

\(^5\) The ACT is a small territory surrounding the city of Canberra, the national capital of Australia. The population is approximately 320,000 people, with a comparatively high level of education and income, according to ABS census statistics.

\(^6\) Even if the individual has a genuine complaint, there is a risk that they may over-state their presentation to “prove” the veracity of their symptoms and impairment, particularly under circumstances
The total sample consisted of 83 females, 36 males and 1 participant who did not specify gender. Participants’ ages ranged from 19 to 73 years, although they were predominantly middle aged ($M = 43.32$ years, $SD = 11.68$ years). Other general demographic data are presented in Table 1. In summary, the majority was non-

<table>
<thead>
<tr>
<th>Demographic (N = 120)</th>
<th>Percentage Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
</tr>
<tr>
<td>non-indigenous Australian</td>
<td>76%</td>
</tr>
<tr>
<td>European</td>
<td>18%</td>
</tr>
<tr>
<td>African</td>
<td>3%</td>
</tr>
<tr>
<td>Asian</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>South American</td>
<td>&lt;1%</td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
</tr>
<tr>
<td>married</td>
<td>62%</td>
</tr>
<tr>
<td>single</td>
<td>15%</td>
</tr>
<tr>
<td>separated or divorced</td>
<td>11%</td>
</tr>
<tr>
<td>de facto relationship</td>
<td>5%</td>
</tr>
<tr>
<td>other</td>
<td>7%</td>
</tr>
<tr>
<td><strong>Living arrangements</strong></td>
<td></td>
</tr>
<tr>
<td>with partner</td>
<td>73%</td>
</tr>
<tr>
<td>alone</td>
<td>12%</td>
</tr>
<tr>
<td>with parents or relatives</td>
<td>8%</td>
</tr>
<tr>
<td>with children</td>
<td>5%</td>
</tr>
<tr>
<td>with friends or flatmates</td>
<td>3%</td>
</tr>
<tr>
<td><strong>Highest educational qualification</strong></td>
<td></td>
</tr>
<tr>
<td>less than Year 9</td>
<td>3%</td>
</tr>
<tr>
<td>Year 9</td>
<td>10%</td>
</tr>
<tr>
<td>Year 10</td>
<td>13%</td>
</tr>
<tr>
<td>Year 11</td>
<td>4%</td>
</tr>
<tr>
<td>Year 12</td>
<td>23%</td>
</tr>
<tr>
<td>TAFE/trade qualification</td>
<td>23%</td>
</tr>
<tr>
<td>undergraduate qualification</td>
<td>9%</td>
</tr>
<tr>
<td>post-graduate qualification</td>
<td>14%</td>
</tr>
<tr>
<td><strong>Employment level</strong></td>
<td></td>
</tr>
<tr>
<td>manager, professional or associated professional</td>
<td>58%</td>
</tr>
<tr>
<td>tradesperson</td>
<td>8%</td>
</tr>
<tr>
<td>higher administrative or clerical workers</td>
<td>23%</td>
</tr>
<tr>
<td>production, transport, low level administration or labouring</td>
<td>11%</td>
</tr>
</tbody>
</table>

where they perceive that others do not believe them.
indigenous Australian, married, living with a partner and well-educated professionals. Those born overseas were long term residents (range = 10 to 47 years, $M = 28.46$ years, $SD = 9.73$ years) and only 12% were from non-English speaking backgrounds.

The mean duration of pain for the sample was 54.58 months ($SD = 70.39$ months) and 83% were taking medication for pain and or psychological symptoms. Table 2 illustrates the heterogeneity of participants’ pain complaints and attributed source of injury/pain. Less than half (46%) were currently working and one third of these (34%) were on restricted duties due to their pain. Seventy percent of the entire sample were receiving or seeking compensation for injury/pain. Lifetime exposure to a traumatic event was reported by 72% of participants and the mean number of different types of prior trauma (e.g., natural disaster, accident, assault etc) was 1.84 ($SD = 1.81$).

**Table 2. Classification of pain sites and sources of injury for entire sample**

<table>
<thead>
<tr>
<th>Pain Site and Source</th>
<th>Percentage Frequency</th>
<th>$N = 120$</th>
</tr>
</thead>
<tbody>
<tr>
<td>IASP pain site coding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>more than three sites</td>
<td>44%</td>
<td></td>
</tr>
<tr>
<td>lower back</td>
<td>21%</td>
<td></td>
</tr>
<tr>
<td>shoulders and upper limbs</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td>neck</td>
<td>6%</td>
<td></td>
</tr>
<tr>
<td>lower limbs</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>middle back</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>head and face</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>abdominal/pelvic region</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Attributed source of injury/pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>work-related accidents</td>
<td>39%</td>
<td></td>
</tr>
<tr>
<td>non-accidental work injuries</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>(i.e. gradual onset)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>motor vehicle accidents</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>no apparent cause</td>
<td>6%</td>
<td></td>
</tr>
<tr>
<td>sporting injuries</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>illness</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>surgery</td>
<td>&lt;1%</td>
<td></td>
</tr>
<tr>
<td>accidents at home</td>
<td>&lt;1%</td>
<td></td>
</tr>
</tbody>
</table>
5.2.2 Measures

The complete questionnaire (see Appendix A) included the following measures:

5.2.2.1 Background Information and Pain Characteristics Questionnaire

Participants completed a questionnaire devised to collect information regarding pain characteristics and demographic background (age, sex, nationality, language, marital status, living arrangements, educational level, and occupation). The cause of injury/pain was selected from a list and participants gave open-ended responses to questions about diagnosis and pain localisation. Participants were asked to specify pain duration in months and select the frequency of pain from 1 (constant – always present, always the same intensity) to 7 (rarely present – I have pain every now and then, with days or weeks in between). Pain intensity was assessed using an 11 point numerical rating scale, anchored at 0 (no pain) and 10 (worst pain imaginable) for current pain, average pain, minimum and maximum pain. Numerical rating scales are considered valid measures of pain intensity (Jensen & Karoly, 1992). Expectations of prognosis were rated as either: get worse, no change, improve, totally recover.

The next series of questions inquired about treatment history. Participants were asked to list the medications taken in the past week and specify the number of days on which the medication was taken. Participants were also asked to select from a list which professional groups they had consulted and to specify any not on the list. Professional groups listed were: acupuncturist, anaesthetist, chiropractor, homeopath, hypnotherapist, massage therapist, naturopath, neurologist, neurosurgeon, occupational therapist, orthopaedic surgeon, osteopath, physiotherapist, psychologist, psychiatrist, rheumatologist, pain clinic and general practitioner. Participants were further asked how many consultations in total they had in the previous three months: 0-3 consultations, 4-6 consultations, 7-12 consultations, 13-24 consultations.

Participants were asked to indicate their current work status, primary source of income, whether or not they were receiving or seeking compensation and how able they believed they were to currently do their normal job (1 = as well as I could before my injury/pain, 2 = only with reduced hours and or restricted duties, 3 = not at all in my current condition). For those who were participating in the paid workforce, they were asked to indicate whether they were on restricted duties and how much time off work they had had in the past 12 months. For those who were not working, they were asked
to indicate how long it had been since they had last worked, whether or not they had attempted to return to work and how much time they had had at work in the past 12 months. No reliability checks were performed for these measures, unless data were missing.

5.2.2.2 Posttraumatic Diagnostic Scale

Participants completed a modified version of the Posttraumatic Stress Diagnostic Scale (PDS) (Foa et al., 1997), 49-item self-report instrument designed to mirror DSM-IV diagnostic criteria and to quantify the severity of PTSD symptoms. In the original version, the first section comprises 12 items assessing exposure (yes/no) to different types of traumatic events, including an ‘other’ category. These are totalled to form an index of the total number of traumatic exposures. With reference to the most disturbing of these events, the individual then rates the frequency in the past month (ranging from 0 (never) to 3 (5 times per week or more/very severe/nearly always) of 17 symptoms specified by the DSM-IV. A continuous measure of overall symptom severity is obtained by summing symptom item ratings (maximum score = 51). Due to the direct correspondence with DSM-IV criteria, the PDS can also be used as a dichotomous measure to determine whether the individual meets symptom cluster criteria for diagnosis of PTSD (PTSD+/PTSD-). Thus, PTSD is determined according to satisfaction of DSM-IV criteria B (one or more re-experiencing symptoms corresponding to items 1 to 5), C (three or more avoidance or numbing symptoms, corresponding to items 6 to 12) and D (two or more hyperarousal symptoms, corresponding to items 13 to 17). In addition, the presence or absence of impairment due to PTSD symptoms is subjectively rated across nine areas of functioning.

A slight modification therefore was made to the PDS administration. After completing the checklist reflecting their experience of other traumatic stressors, participants were asked to complete the PTSD symptom and impairment questions with respect to the circumstances of their injury/pain (rather than the most disturbing traumatic event as in the original). This modification was made to narrow the scope of investigation to the specific case where PTSD and chronic pain arise from the same index event. It was considered that standard administration of the PDS could potentially confound interpretation of the results by blurring different causal pathways and mechanisms.
According to the manual, the scale has good test-retest reliability \((r = .74)\) and excellent internal consistency \((\alpha = .92)\) and offers good overall agreement with PTSD diagnoses obtained by use of the “gold standard” assessment tool, the Structured Clinical Interview for DSM-III-R (SCID) (R. L. Spitzer, Williams, Gibbon, & First, 1990). Specifically, “a kappa of .59 between the PDS and the SCID was obtained, with 79.4% agreement between the two measures” (Manual, p.15). Further, the PDS demonstrated high sensitivity (82.0%) and specificity (76.7%) (Manual, p. 15-16). Foa et al. demonstrated that higher PDS scores also correlate with higher scores on subjective measures of depression (BDI), anxiety (STAI) and intrusion and avoidance symptoms (IES) (Foa et al., 1997; Foa & Riggs, 1993; Foa et al., 1993).

5.2.2.3 Anxiety Sensitivity Index

Evidence indicates that fear of pain and subsequent pain-related avoidance may be mediated by a general tendency to fear the somatic and affective arousal associated with pain (anxiety sensitivity) (Asmundson & Norton, 1995; Asmundson, Norton, & Veloso, 1999; Asmundson & Taylor, 1996). Similarly, anxiety sensitivity appears to mediate the presence of a diagnosis of PTSD (A. J. Lang et al., 2002; S. Taylor et al., 1992) and PTSD symptom severity (Asmundson et al., 1998; Fedoroff et al., 2000; A. J. Lang et al., 2002).

Thus to assess this construct, the widely used Anxiety Sensitivity Index (ASI, Reiss, Peterson, Gursky, & McNally, 1986) was administered. Each of the 16 items is rated on a 5-point scale, ranging from 0 (very little) to 4 (much), indicating the degree to which an individual is concerned about possible negative consequences of anxiety-related sensations. The ASI has been shown to be a reliable and stable measure of the fear of anxiety with Cronbach’s \(\alpha\) above .88 and test-retest correlations above .70, even over a period of three years (Peterson & Heilbronner, 1987; Reiss, 1991; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004; Sandin, Chorot, & McNally, 2001). Studies have shown that the ASI can be explained by three lower order-factors (physical concerns, mental incapacitation concerns and social concerns), which all load onto a single second-order factor of general AS (Rodriguez et al., 2004; Zinbarg, Barlow, & Brown, 1997).
5.2.2.4 Pain Anxiety Symptoms Scale

Participants also completed the Pain Anxiety Symptoms Scale (PASS) (McCracken et al., 1992). The PASS is a 40-item self-report measure of fear and anxiety associated with pain. In addition to a total score, there are four sub-scales assessing cognitive anxiety symptoms, escape and avoidant behaviours, fearful appraisals of pain and physiological anxiety symptoms related to pain. In the present study, the PASS total score and sub-scale scores were used to assess anxiety responses to pain. Each item is rated from 0 (never) to 5 (always) as to how often the individual has the thought or does the behaviour.

Initial construction and validation of the PASS (McCracken et al., 1992) indicated good internal consistency for the total scale (Cronbach’s $\alpha = .94$) and each of the subscales (ranging from .81 to .89). There were also meaningful inter-scale correlations ($r = .45$ to .70) that did not supercede the individual scales’ abilities to provide unique information. The validity of scores from the PASS has been supported by significant positive correlations with other measures of anxiety, depression, pain and disability, such as the MPQ, the STAI, the WHYMPI, the Catastrophising subscale of the CSQ, the BDI and the Pain Disability Index (PDI) (McCracken et al., 1993; McCracken et al., 1992; Rosenstiel & Keefe, 1983).

5.2.2.5 Coping Strategies Questionnaire

The Coping Strategies Questionnaire (CSQ) (Rosenstiel & Keefe, 1983) contains 44 items covering eight types of strategies (five cognitive, three behavioural) for coping with pain: reinterpret pain sensations, coping self-statements, ignore sensations, diverting attention, praying/hoping, catastrophising, increased behavioural activities, and pain behaviours. Items are rated from 0 (never) to 6 (always). The perceived effectiveness of the coping efforts is rated on two items, using a 7-point scale. The ‘control over pain’ scale is anchored at 0 (no control), 3 (some control) and 6 (complete control). The ‘ability to decrease pain’ scale is anchored at 0 (can’t decrease it at all), 3 (can decrease it somewhat) and 6 (can decrease it completely).

Based on its original data, the subscales of the CSQ have good internal consistency (Cronbach’s $\alpha = .71$ to .85) except for increasing pain behaviours, which is poor (.28) (Rosenstiel & Keefe, 1983). Internal consistency coefficients for the CSQ in subsequent studies have ranged from adequate to very good (.57 to .89) (Keefe et al.,
Numerous studies support the concurrent validity of the CSQ, which has explained significant variance in adjustment and distress (Main & Waddell, 1991; Rosenstiel & Keefe, 1983).

5.2.2.6 The Chronic Pain Coping Inventory

The Chronic Pain Coping Inventory (CPCI) (Jensen et al., 1995) consists of 65 items forming 11 subscales that measure different strategies patients might use to cope with their pain. The CPCI was specifically designed to capture primarily behavioural strategies that are actively discouraged or encouraged during multidisciplinary pain management programs (Jensen et al., 1995). The subscales are divided into three categories: illness-focussed (guarding, resting, asking for assistance and medication use (opioid, nonsteroid, sedative-hypnotic)), wellness-focussed strategies (relaxation, task persistence, exercise/stretch and coping self-statements) and the strategy of seeking social support. Items are rated according to the number of days in the past week that the individual utilised the strategy (i.e. 0 to 7 days).

Initial validation and cross validation studies conducted by Jensen et al. (1995) demonstrated adequate to excellent test-retest reliability (.65 to .90) and internal consistency (.74 to .91). The CPCI also exhibited validity in its correlation with spouse-ratings of patient functioning. Moderate to strong correlations ($r > |+.30|$) existed between patient ratings of guarding, resting, asking for assistance and sedative-hypnotic medication use and spouse ratings of disability and activity level. Slightly weaker but still significant correlations ($|+.20| < r > |+.30|$) also existed between coping self-statements and seeking social support and spouse ratings of disability. The CPCI has also been used to predict several adjustment variables, including pain severity, interference and social support (Hadjistavropoulos, MacLeod, & Asmundson, 1999).

5.2.2.7 Pain Disability Index

The Pain Disability Index (PDI) (Tait, Chibnall, & Krause, 1990) is a seven-item inventory, which was used to assess the degree of interference caused by pain in seven voluntary and mandatory areas of functioning: family/home responsibilities, recreation, social activity, occupation, sexual behaviour, self care and life-support activity. Overall impact of pain on each area of function is rated on an 11 point scale, ranging from 0 (no disability) to 10 (total disability).
Research with the PDI has shown support for it as a valid and reliable measure of pain-related disability (Tait et al., 1990). It has high internal consistency (.86), though test-retest reliability over a 2-month period was significant but low (.44). The PDI has also displayed concurrent validity with patient reports of psychological distress, pain severity, and other items measuring pain-related disability. Disability scores determined by the PDI also have been predicted by relevant constructs such as interference with activities, pain symptoms (extent, severity, duration), satisfaction with life activities, employment and education.

5.2.2.8 Zung Self-Rating Depression Scale

Depression is commonly comorbid with both chronic pain (Dworkin & Gitlin, 1991) and PTSD (Breslau et al., 2000). With respect to chronic pain patients, significant depressive symptoms are reported by 50% of individuals (Romano & Turner, 1985) with 30% satisfying DSM-IV criteria for Major Depressive Disorder (Wilson, Eriksson, & D'Eon, 2002). Fifty two percent of males and 65% of females with PTSD met criteria for a Major Depressive Episode according to DSM-IV in an Australian epidemiological sample (Creamer et al., 2001). Rates of depression for chronic pain patients (Magni, Marchetti, Moreschi, & et al., 1993) and for individuals with PTSD (Creamer et al., 2001) are also higher than the general community. For these reasons, measurement of depression was included in the present study.

Participants were thus administered the modified version of the Zung Self-Rating Depression Scale (SDS), as described by Main & Waddell (1984). The SDS assesses the frequency of various symptoms of depression, for this version, rated on a scale ranging from rarely or none of the time or < 1 day per week to most of the time or 5-7 days per week. Good concurrent validity, sensitivity and specificity has been demonstrated for the SDS in chronic pain samples in comparison to the BDI, MMPI Depression Scale and diagnosis based on DSM-III criteria (Turner & Romano, 1984). The SDS involves calculating an index (SDS index), derived by summing the raw scores for the 20 items on the original version, dividing by the maximum possible score of 80 and multiplying by 100. For this study, calculations were adjusted to maintain an index out of 100.
5.2.3 Procedure

Patients from the THC, CIMC, CPC and rehabilitation providers were advised of the existence of the study during attendance at the unit/centre and it was clearly explained that participation was voluntary. After written informed consent was obtained, participants completed the questionnaire in their own time and returned it in a sealed envelope via treating staff or to the researcher directly by mail. No treatment was conducted as part of this study. Participants were aware that treating practitioners would not be given access to questionnaires nor provided with feedback unless express consent was granted. The informed consent forms and information sheets that were given to participants are reproduced in Appendix B.

5.2.4 Ethics Approval

The Human Research Ethics Committee of the Australian National University and ACT Department of Health and Community Care approved the protocol for this study. The ACT Department of Health and Community Care Survey Resource Group endorsed the questionnaire prior to ethics approval. The approval letters are reproduced in Appendix C.

5.2.5 Data Analyses

Prior to data analysis, data were screened for accuracy of data entry, missing values, univariate and multivariate outliers and fit between their distributions and the assumptions of multivariate analysis (normality, linearity, homoscedasticity, multicollinearity and singularity). Three cases were discarded due to insufficient completion of published instruments (more than 2 missing items per subscale or scale). Of the remaining 120 questionnaires, missing single item data for published instruments was minimal. The pattern appeared random and was replaced by the mean of that individual’s subscale or total scale score for that instrument. This method of replacing missing data was chosen to enable retention of a sufficient sample size. No univariate outliers were identified nor were there any multivariate outliers, using Mahalanobis distance with \( p < .001 \).

Significant skewness and kurtosis (\( z > 3.29 \)) were evident in the distribution of maximum pain ratings, PTSD disability scores and number of painful body sites. A distribution with negative skew and kurtosis for maximum pain ratings is an expected result given the nature of the scale so raw scores were retained for analysis. The PTSD
disability variable was deleted from analysis as it showed significant negative skew and kurtosis and was an unreliable measure of disability. The yes/no format of items encouraged an affirmative response in most participants. Over half of the participants obtained scores of 7 or above (maximum = 9), irrespective of PTSD diagnosis or symptom severity, suggesting responses may have been tainted by ratings of disability due to non-PTSD symptoms. Square root and logarithmic transformations reduced the skew of number of body parts identified as painful, but did not improve kurtosis. For this reason and given the more straightforward interpretation of non-transformed variables, raw scores were retained for analysis.

Independent sample t-tests and chi square analyses were conducted to determine whether PTSD and No PTSD groups differed according to PTSD severity and on the primary demographic variables of age, gender, educational background and prior traumatic exposures. Calculation of t was based on the assumption of equal variances unless otherwise specified. The reliability of each published scale was calculated using Cronbach’s alpha consistency coefficient (the standardised item alpha is reported). Subscales as well as total scores were evaluated for internal consistency in this way. Multiple analyses of variance (MANOVA) were then conducted to examine differences between PTSD and No PTSD groups on measures of (1) outcome (pain severity, health care utilisation, pain-related disability and depression); (2) pain-related anxiety; (3) pain coping strategies; and (4) anxiety sensitivity. The alpha level was set at .05 and Bonferroni corrections were made to maintain this level of Type I error in the context of multiple comparisons. Given the survey design, a sequential adjustment was chosen to deal with nonorthogonality in MANOVAs (Tabachnick & Fidell, 2001). Bivariate correlations were also calculated to highlight relationships between variables within each patient group. Three hierarchical regression analyses were conducted on the dependent variables, pain severity index, pain-related disability and depression in order to determine the unique contribution each block of independent variables made to predicting these.

SPSS for Windows (Version 10) was the software used for all analyses. SPSS calculates alpha to three decimal places, therefore all “.000” output values are reported here as $p < .001$. 

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5.3 Results

5.3.1 Classification and Severity of PTSD

Participants were classified into two groups, based on whether or not they met DSM-IV TR criteria for diagnosis of PTSD, according to their responses on the PDS. That is, to classify participants into the PTSD group, they must have: (1) indicated that their pain arose from a work-related accident, motor vehicle accident or other sudden injury or event (A1); (2) perceived that their own or someone else’s life was in danger and felt helpless and or terrified (A2) and (3) endorsed at least one cluster B symptom; three cluster C symptoms and two cluster D symptoms.

As PTSD is a multidimensional construct comprising three symptom clusters (re-experiencing, avoidance/numbing, and hyperarousal), all three clusters measured by the PDS as well as the total score were evaluated for internal consistency. The full scale and re-experiencing subscale both demonstrated excellent reliability (Cronbach’s $\alpha = .94$ and .92, respectively), while the avoidance/numbing and hyperarousal subscales exhibited high reliability (both Cronbach’s $\alpha = .85$).

On the basis of their PDS scores, 57 participants met full criteria for comorbid onset PTSD and 63 participants did not meet full criteria for comorbid onset PTSD. It must be noted that three of the individuals included in the No PTSD group met criteria for partial PTSD (i.e. met the requisite stressor and re-experiencing criteria and either C or D criteria).

Table 3 presents PDS total and subscale scores for the PTSD and No PTSD group. Some of the No PTSD group did not complete the PDS as their pain did not result from a specific incident or injury and were excluded from the following analysis. As would be expected, independent t-tests showed that, compared to the No PTSD group, the PTSD group scored significantly higher on re-experiencing symptoms ($t(89^7) = 9.18$, $p<.001$, $d = 1.82$), avoidance and numbing symptoms ($t(97) = 8.13$, $p<.001$, $d = 1.65$), hyperarousal ($t(97) = 8.00$, $p<.001$, $d = 1.62$) and PDS total ($t(97) = 9.77$, $p<.001$, $d = 2.07$). These were large effects. The non-zero scores of the No PTSD group, however, suggest two possibilities: the potential for overlapping symptoms from chronic pain

---

7 Degrees of freedom were reduced in this case as calculation of $t$ did not assume equal variance (Levene’s test was significant).
problems to artificially inflate scores on this scale or that a dimensional rather than a categorical approach may better represent posttraumatic symptoms.

Table 3. Mean Posttraumatic Stress Diagnostic Scale (PDS) scores for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>PDS Scale</th>
<th>PTSD (n=57)</th>
<th>No PTSD (n=42)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Re-experiencing (B) symptoms</td>
<td>7.88</td>
<td>4.45</td>
</tr>
<tr>
<td>Avoidance &amp; numbing (C) symptoms</td>
<td>11.54</td>
<td>4.46</td>
</tr>
<tr>
<td>Hyperarousal (D) symptoms</td>
<td>10.61</td>
<td>3.44</td>
</tr>
<tr>
<td>Total</td>
<td>30.04</td>
<td>10.48</td>
</tr>
</tbody>
</table>

5.3.2 Demographic Analyses

Descriptive statistics for the demographic variables for the PTSD and No PTSD groups are presented in Table 4. In general, the two groups were quite similar with respect to demographic background. There were no significant differences between the groups in terms of gender ($\chi^2(2) = 5.20, p = .07, \text{power} = .69$) or educational level ($\chi^2(7) = 5.39, p = .61$), nor in terms of age ($t(118) = .03, p = .98, \text{power} = .05$) or reported number of prior traumatic exposures ($t(118) = .20, p = .84, \text{power} = .08$), however, the low power to detect a significant effect for the latter two is noted. With respect to the nature of prior trauma, the PTSD group only differed from the No PTSD group in terms of exposure to military combat ($\chi^2(1) = 16.11, p < .001$) and sexual contact before the age of 18 years with someone more than five years older ($\chi^2(1) = 8.07, p < .01$). That is, the PTSD group was more likely to have been exposed to military combat. By contrast, the No PTSD group was more likely than the PTSD group to have been exposed to early sexual contact. Exposure to all other specified traumas was equivalent (all $p > .05$).
Table 4. Mean and percentage frequency demographic data for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Demographic</th>
<th>PTSD (n = 57)</th>
<th>No PTSD (n = 63)</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Females</td>
<td>60%</td>
<td>78%</td>
</tr>
<tr>
<td>Mean age in yearsa</td>
<td>43.28 (11.08)</td>
<td>43.35 (12.29)</td>
</tr>
<tr>
<td>% reaching each educational level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>less than Year 9</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>Year 9</td>
<td>12%</td>
<td>8%</td>
</tr>
<tr>
<td>Year 10</td>
<td>14%</td>
<td>12%</td>
</tr>
<tr>
<td>Year 11</td>
<td>5%</td>
<td>3%</td>
</tr>
<tr>
<td>Year 12</td>
<td>16%</td>
<td>30%</td>
</tr>
<tr>
<td>TAFE/trade qualification</td>
<td>12%</td>
<td>21%</td>
</tr>
<tr>
<td>undergraduate qualification</td>
<td>6%</td>
<td>7%</td>
</tr>
<tr>
<td>post-graduate qualification</td>
<td>14%</td>
<td>15%</td>
</tr>
<tr>
<td>Mean number of prior traumatic exposuresa</td>
<td>1.88 (1.70)</td>
<td>1.81 (1.91)</td>
</tr>
<tr>
<td>% Exposed to:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>accident, fire or explosion</td>
<td>46%</td>
<td>33%</td>
</tr>
<tr>
<td>natural disaster</td>
<td>19%</td>
<td>13%</td>
</tr>
<tr>
<td>phys. assault, known perpetrator</td>
<td>21%</td>
<td>22%</td>
</tr>
<tr>
<td>phys. assault, unknown perpetrator</td>
<td>18%</td>
<td>16%</td>
</tr>
<tr>
<td>sex assault, known perpetrator</td>
<td>9%</td>
<td>13%</td>
</tr>
<tr>
<td>sex assault, unknown perpetrator</td>
<td>5%</td>
<td>10%</td>
</tr>
<tr>
<td>military combat</td>
<td>23%</td>
<td>0%</td>
</tr>
<tr>
<td>sexual contact (&lt;18yrs), with person &gt;5yrs older</td>
<td>5%</td>
<td>24%</td>
</tr>
<tr>
<td>imprisonment</td>
<td>4%</td>
<td>5%</td>
</tr>
<tr>
<td>torture</td>
<td>0%</td>
<td>3%</td>
</tr>
<tr>
<td>life-threatening illness</td>
<td>19%</td>
<td>19%</td>
</tr>
<tr>
<td>other trauma</td>
<td>19%</td>
<td>18%</td>
</tr>
</tbody>
</table>

*a SD is presented in parentheses.

5.3.3 Analyses for Pain Severity, Health Care Utilisation, Disability and Depression

Descriptive statistics for pain severity, health care utilisation, disability and depression variables for the PTSD and No PTSD groups are presented in Table 5. The pain severity index is a four-item composite, which was calculated for each participant by averaging his or her current, average, minimum and maximum pain intensity ratings. A discussion of the validity and utility of this measure is presented elsewhere (Jensen et al., 1999). The pain severity index was chosen for analysis in the present study as it showed improved distribution compared to the individual rating scales (which violated
assumptions of homogeneity of variance and covariance). In addition, it showed high internal consistency (Cronbach’s α = .84). The PTSD group showed a trend toward

Table 5. Means and percentage frequencies for pain severity, health care utilisation, depression and disability variables for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>PTSD (n = 57)</th>
<th>No PTSD (n = 63)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pain severity variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current pain intensity</td>
<td>5.36</td>
<td>4.68</td>
</tr>
<tr>
<td>Average pain intensity</td>
<td>5.79</td>
<td>5.58</td>
</tr>
<tr>
<td>Minimum pain intensity</td>
<td>3.69</td>
<td>3.02</td>
</tr>
<tr>
<td>Maximum pain intensity</td>
<td>8.32</td>
<td>7.79</td>
</tr>
<tr>
<td>Pain Severity Index</td>
<td>5.79</td>
<td>5.27</td>
</tr>
<tr>
<td>Duration of chronic pain (months)</td>
<td>54.91</td>
<td>54.27</td>
</tr>
<tr>
<td>Number of painful body locations</td>
<td>5.65</td>
<td>6.37</td>
</tr>
<tr>
<td><strong>Health care utilisation variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of medications</td>
<td>2.74</td>
<td>1.90</td>
</tr>
<tr>
<td>Number of professions consulted</td>
<td>7.67</td>
<td>6.16</td>
</tr>
<tr>
<td>% Frequency of consultations in the past 3 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>once per month</td>
<td>12%</td>
<td>16%</td>
</tr>
<tr>
<td>once per fortnight</td>
<td>26%</td>
<td>30%</td>
</tr>
<tr>
<td>once per week</td>
<td>26%</td>
<td>32%</td>
</tr>
<tr>
<td>twice per week</td>
<td>35%</td>
<td>22%</td>
</tr>
<tr>
<td><strong>Disability variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain Disability Index score</td>
<td>42.06</td>
<td>30.10</td>
</tr>
<tr>
<td>Number of days off in past 12 months</td>
<td>145.91</td>
<td>110.48</td>
</tr>
<tr>
<td>% Currently working</td>
<td>40%</td>
<td>52%</td>
</tr>
<tr>
<td>% Perceived ability to work as before</td>
<td>2%</td>
<td>15%</td>
</tr>
<tr>
<td>with reduced hours/duties</td>
<td>42%</td>
<td>44%</td>
</tr>
<tr>
<td>not at all in current condition</td>
<td>51%</td>
<td>24%</td>
</tr>
<tr>
<td>% Perceived prognosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>get worse</td>
<td>35%</td>
<td>10%</td>
</tr>
<tr>
<td>no change</td>
<td>18%</td>
<td>22%</td>
</tr>
<tr>
<td>improve</td>
<td>46%</td>
<td>56%</td>
</tr>
<tr>
<td>totally recover</td>
<td>2%</td>
<td>11%</td>
</tr>
<tr>
<td>% Receiving compensation or litigating</td>
<td>88%</td>
<td>54%</td>
</tr>
<tr>
<td><strong>Depression variable:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-rating Depression Scale index score</td>
<td>55.59</td>
<td>43.26</td>
</tr>
<tr>
<td></td>
<td>12.43</td>
<td>14.29</td>
</tr>
</tbody>
</table>
higher scores on the pain severity index than the No PTSD group. An independent samples t-test indicated that this difference was not significant, however the power was less than ideal ($t(118) = 1.89, p = .06$, power = .60). There were also no significant differences between groups in terms of duration of pain ($t(118) = .05, p = .96$, power = .06) or number of painful body locations ($t(118) = .98, p = .33$, power = 1.0).

A MANOVA indicated a significant, though small effect of PTSD on the two health care utilisation variables (number of different professions consulted and number of medications taken) (Wilks’ lambda = .92, $F(1,118) = 4.98, p = .008$, $\eta_p^2 = .09$, power = .80). Univariate ANOVAs showed that the PTSD group had consulted more types of treatment providers ($F(1,118) = 6.74, p = .011$, $\eta_p^2 = .06$) and were taking more medications ($F(1,118) = 7.06, p = .009$, $\eta_p^2 = .05$) than the No PTSD group. Chi-square analysis revealed no significant difference between groups in terms of the frequency of consulting treatment providers in the previous 3 months ($\chi^2(3) = 2.48, p = .48$). Compared to the No PTSD group, more of the PTSD group used non-steroidal anti-inflammatories (NSAIDS; 51% vs 30%), simple analgesics (47% vs 33%), sedatives (21% vs 10%), anticonvulsants (25% vs 11%) and antidepressants (51% vs 32%). Analysis of odds ratios showed the PTSD group were more than twice as likely than the No PTSD group to use both NSAIDS (OR = 2.40, 95% CI [1.14, 5.07]) and antidepressants (OR = 2.23, 95% CI [1.06, 4.68]), however, they were no more likely to use other medications.

Reliability analyses showed the PDI had high internal consistency (Cronbach’s $\alpha = .86$). An independent t-test showed that the PTSD group obtained significantly higher PDI scores than the No PTSD group, representing a large effect ($t(118) = -5.48, p < .001$, $d = 1.00$). Chi-square analysis revealed that the PTSD group expected a significantly different prognosis to the No PTSD group ($\chi^2(4) = 14.77, p = .005$). In particular, odds ratios showed the PTSD group were five times more likely than the No PTSD group to rate their most likely prognosis as get worse (OR = .20, 95% CI [.07, .54]). Further, the No PTSD group were twice as likely than the PTSD group to expect a positive prognosis rather than a negative prognosis (OR = .43, 95% CI [.20, .90]).

8 A positive prognosis referred to expectations of improvement or total recovery. A negative prognosis referred to an expectation of no change or to get worse, as the maintenance of a clinical disorder (i.e. absence of improvement over time) is considered a negative outcome.
Fewer of the PTSD group were currently working compared to the No PTSD group, although this difference was not significantly different ($\chi^2(3) = 1.74, p = .19$). Further, the PTSD and No PTSD groups significantly differed in their beliefs about their current ability to work ($\chi^2(3) = 15.45, p = .001$). The PTSD group were three times more likely than the No PTSD group to believe they could not work at all (OR = 3.33, 95% CI [1.51, 7.36]). Conversely, the No PTSD group were ten times more likely than the PTSD group to believe they could work as well as they could before their injury (OR = .10, 95% CI [.01, .81]). The PTSD group were also more likely than the No PTSD group to be receiving compensation or undertaking litigation ($\chi^2(1) = 16.23, p < .001$). Indeed, PTSD diagnosis was the only variable significantly correlated with receiving compensation or undertaking litigation, although the association was weak and accounted for only 14% of variance ($r^2 = .37, p < .001$).

The SDS demonstrated excellent internal consistency (Cronbach’s α = .90). The PTSD group obtained higher SDS index scores than the No PTSD group, indicating that they were significantly more depressed than the No PTSD group ($t(118) = 5.02, p < .001, d = .92$).

5.3.4 Analyses of Anxiety Sensitivity

Based on ASI scores, the PTSD group demonstrated greater anxiety sensitivity ($M = 22.51, SD = 10.94$) than the No PTSD group ($M = 14.05, SD = 10.32$). According to an independent samples t-test, this difference was significant and represented a large effect ($t(118) = 4.36, p < .001, d = .80$). The ASI also demonstrated excellent internal consistency (Cronbach’s α = .92).

5.3.5 Analyses for Pain-related Anxiety

Reliability analyses indicated that the PASS had high to excellent reliability, for the total score (Cronbach’s α = .96), the Cognitive subscale (Cronbach’s α = .92), the Fearful Appraisal subscale (Cronbach’s α = .86), the Physiological Symptoms subscale (Cronbach’s α = .82) and the Avoidance subscale (Cronbach’s α = .92).

As can be seen from Table 6, the PTSD group obtained higher PASS scores than the No PTSD group. An independent sample $t$ test indicated that this difference was
significant ($t(118) = 6.22, p < .001, d = 1.13$). A MANOVA conducted on the four subscales of the PASS also showed a significant, though small effect (Wilk’s lambda = .421, $F(4,115) = 9.64, p < .001, \eta_p^2 = .25$). Univariate analyses revealed that the PTSD group reported higher levels of Cognitive anxiety symptoms ($F(1) = 24.50, p < .001, \eta_p^2 = .17$), Escape and Avoidance Behaviours ($F(1)=28.06, p<.001, \eta_p^2 = .19$), Fearful Appraisals of pain ($F(1) = 27.67, p < .001, \eta_p^2 = .19$) and Physiological anxiety symptoms related to pain ($F(1) = 33.65, p < .001, \eta_p^2 = .22$) than the No PTSD group.

### Table 6. Mean Pain Anxiety Stress Scale (PASS) scores for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>PASS subscale</th>
<th>PTSD</th>
<th>No PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$n = 57$</td>
<td>$n = 63$</td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Cognitive anxiety symptoms</td>
<td>33.09</td>
<td>10.19</td>
</tr>
<tr>
<td>Avoidance</td>
<td>26.41</td>
<td>8.87</td>
</tr>
<tr>
<td>Fearful Appraisal</td>
<td>20.68</td>
<td>10.11</td>
</tr>
<tr>
<td>Physiological anxiety symptoms</td>
<td>23.59</td>
<td>11.48</td>
</tr>
<tr>
<td>Total</td>
<td>103.77</td>
<td>34.85</td>
</tr>
</tbody>
</table>

5.3.6 Analyses for Pain-related Coping Strategies

Results of reliability analyses, presented in Table 7, indicated that the CSQ\(^9\) and CPCI full scales and subscales were all of high to excellent internal consistency. Descriptive statistics for the two measures of pain-related coping strategies (CSQ and CPCI) are shown in Table 8. Separate MANOVAs were conducted upon the individual subscales within each test to determine if there were any differences between scores for individuals with and without PTSD.

For the CSQ, the combined subscales were significantly affected by a diagnosis of PTSD (Wilk’s lambda = .756, $F(7,112) = 5.16, p < .001, \eta_p^2 = .24$). Univariate analyses indicated that the PTSD group reported greater reliance on praying/hoping ($F(1) = 9.14$, $p = .003, \eta_p^2 = .07$), were more likely to engage in catastrophising ($F(1) = 36.26$, $p < .001, \eta_p^2 = .21$).

\(^9\) The pain behaviours subscale had been included in the questionnaire administered to participants, however, the results of this subscale were not analysed due to overlap with items better assessed by the CPCI.
Table 7. Results of internal consistency analyses for the Coping Strategies Questionnaire (CSQ) and Chronic Pain Coping Inventory (CPCI)

<table>
<thead>
<tr>
<th>Scale (N = 120)</th>
<th>Cronbach’s Standardised Item α</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CSQ</strong></td>
<td></td>
</tr>
<tr>
<td>Diverting attention</td>
<td>.80</td>
</tr>
<tr>
<td>Reinterpret pain sensations</td>
<td>.78</td>
</tr>
<tr>
<td>Coping self-statements</td>
<td>.80</td>
</tr>
<tr>
<td>Ignoring sensations</td>
<td>.81</td>
</tr>
<tr>
<td>Praying/hoping</td>
<td>.71</td>
</tr>
<tr>
<td>Catastrophising</td>
<td>.90</td>
</tr>
<tr>
<td>Increased behavioural activity</td>
<td>.74</td>
</tr>
<tr>
<td>Total</td>
<td>.88</td>
</tr>
<tr>
<td><strong>CPCI</strong></td>
<td></td>
</tr>
<tr>
<td>Guarding</td>
<td>.81</td>
</tr>
<tr>
<td>Resting</td>
<td>.83</td>
</tr>
<tr>
<td>Asking for assistance</td>
<td>.89</td>
</tr>
<tr>
<td>Relax</td>
<td>.77</td>
</tr>
<tr>
<td>Task persistence</td>
<td>.85</td>
</tr>
<tr>
<td>Exercise/stretch</td>
<td>.90</td>
</tr>
<tr>
<td>Coping self-statements</td>
<td>.87</td>
</tr>
<tr>
<td>Seeking social support</td>
<td>.83</td>
</tr>
<tr>
<td>Total</td>
<td>.92</td>
</tr>
</tbody>
</table>

$p < .001, \eta^2_p = .24$) and were less likely to use coping self-statements $(F(1) = 7.70, p = .006, \eta^2_p = .06)$. There were no significant differences between the PTSD and No PTSD groups in the use of diverting attention, reinterpreting or ignoring pain sensations or increasing behavioural activity to cope with pain, although the statistical power for these analyses was very low (power = .06, .05, .31, .11, respectively).

For the CPCI, the combined subscales were significantly affected by a diagnosis of PTSD (Wilk’s lambda = .79, $F(8,111) = 3.74, p = .001, \eta^2_p = .21$). Univariate analyses indicated that the PTSD group were more likely to engage in guarding $(F(1) = 12.56, p = .001, \eta^2_p = .10)$ and resting behaviours $(F(1) = 9.33, p = .003, \eta^2_p = .07$) to cope with pain. Although they also appeared less likely than the No PTSD group to persist with tasks, this variable was no longer significant following the Bonferroni correction to $p > .006$ (power = .68). There were no significant differences between groups in their use of relaxation, asking for assistance, exercise/stretching, coping self-statements or seeking social support (all $p > .006$), but again the ability to detect significant effects was low to very low (power = .27, .24, .05, .05, .35).
Table 8. Mean Coping Scale Questionnaire (CSQ) and Chronic Pain Coping Inventory (CPCI) scores for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Scale</th>
<th>PTSD (n = 57)</th>
<th>No PTSD (n = 63)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>CSQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diverting attention</td>
<td>13.14</td>
<td>7.51</td>
</tr>
<tr>
<td>Reinterpret pain sensations</td>
<td>7.78</td>
<td>5.87</td>
</tr>
<tr>
<td>Coping self-statements</td>
<td>18.23</td>
<td>7.22</td>
</tr>
<tr>
<td>Ignoring sensations</td>
<td>12.14</td>
<td>7.75</td>
</tr>
<tr>
<td>Praying/hoping</td>
<td>15.09</td>
<td>8.65</td>
</tr>
<tr>
<td>Catastrophising</td>
<td>20.80</td>
<td>8.98</td>
</tr>
<tr>
<td>Increased behavioural activity</td>
<td>14.90</td>
<td>6.53</td>
</tr>
<tr>
<td>Total</td>
<td>102.15</td>
<td>28.06</td>
</tr>
<tr>
<td>CPCI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guarding</td>
<td>32.20</td>
<td>15.13</td>
</tr>
<tr>
<td>Resting</td>
<td>27.65</td>
<td>11.84</td>
</tr>
<tr>
<td>Asking for assistance</td>
<td>11.89</td>
<td>8.74</td>
</tr>
<tr>
<td>Relax</td>
<td>16.26</td>
<td>11.37</td>
</tr>
<tr>
<td>Task persistence</td>
<td>21.32</td>
<td>10.87</td>
</tr>
<tr>
<td>Exercise/stretch</td>
<td>44.72</td>
<td>22.39</td>
</tr>
<tr>
<td>Coping self-statements</td>
<td>31.43</td>
<td>17.76</td>
</tr>
<tr>
<td>Seeking social support</td>
<td>14.09</td>
<td>11.73</td>
</tr>
<tr>
<td>Total</td>
<td>205.06</td>
<td>70.39</td>
</tr>
</tbody>
</table>

5.3.7 Correlational Analyses

To address the second purpose of this study, which was to determine discernable patterns of association between variables for the two patient groups, a correlational analysis was performed. The key measures of PTSD severity, pain-related anxiety, coping strategies and ASI were treated as independent variables and their association with dependent variables (i.e. health care utilisation, disability and depression) was examined. Given the large number of variables, only variables that showed significant differences between groups (above) were included and the significance level was set at
This correlational data is presented below in Tables 9 and 10. Correlational data for the entire sample are provided in Table D.1 in Appendix D.

5.3.7.1 PTSD group

As can be seen in Table 9, for the PTSD group, the pain severity index showed significant moderate correlations with the PASS Physiological Symptoms, CPCI Resting and PDS-C symptom severity subscales. The pain severity index was significantly and moderately correlated with only one dependent variable, the PDI. The PDI was found to have significant moderate positive correlations with PASS Total, Fear Appraisal, Physiological Symptoms and Escape/Avoidance subscales. By contrast, the other two disability measures, perceived work ability ratings and compensation status were not significantly correlated with the PDI, or any of the independent or other dependent variables. Similarly, neither of the health care utilisation variables, number of providers or number of medications, were significantly correlated with any of the independent or other dependent variables.

Viewing depression as a dependent variable, the SDS (see Table 9) demonstrated strong positive correlations with PASS Total, Cognitive Symptoms and Fear Appraisal subscales and CSQ Catastrophising subscales (all $p < .001$). Significant moderate positive correlations were also demonstrated between the SDS and the following variables: PASS Physiological Symptoms and Escape/Avoidance subscales, CPCI Guarding and Resting subscales, ASI and PDS-C, PDS-D and PDS-Total symptom severity.

Amongst the independent variables, PDS-B severity was not significantly correlated with any other non-PTSD independent variable. PDS-C severity showed significant strong positive correlations with PASS Total and Physiological Symptoms subscales and significant moderate positive correlations with PASS Cognitive Symptoms, Fearful Appraisal and Escape/Avoidance, CSQ Catastrophising, CPCI Resting subscales and ASI. PDS-D severity demonstrated significant moderate positive correlations with ASI, PASS Cognitive Symptoms and CSQ Catastrophising subscales. PDS-Total symptom severity had significant strong positive correlations with the PASS Physiological Symptoms subscale and significant moderate positive correlations with the PASS Total, PASS Cognitive Symptoms, CSQ Catastrophising, CPCI Resting subscales and the ASI. In addition, the ASI demonstrated strong positive correlations with PASS Total, Fearful
Table 9. Zero-order correlations ($r$) for predictor and outcome variables for the PTSD group ($n = 57$)

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$r = r_{pb}$ when correlated with a continuous variable or $r_p$ when correlated with another dichotomous variable.

* $p < .05$, ** $p < .01$, *** $p < .001$
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\[ r = r_{pb} \text{ when correlated with a continuous variable or } r = r_{p} \text{ when correlated with another dichotomous variable.} \]

* \( p < .05 \), ** \( p < .01 \), *** \( p < .001 \)
Appraisal and Physiological Symptoms subscales, moderate positive correlations with the PASS Cognitive Symptoms, Escape/Avoidance and CPCI Resting subscales and a moderate negative correlation with the CSQ Coping Self-Statements subscale (all \( p < .001 \)).

5.3.7.2 No PTSD group

As can be seen in Table 10, a different pattern of relationships was evident amongst the No PTSD group. For this group, the only independent variable significantly correlated with the pain severity index was CSQ Catastrophising. Pain severity was also significantly, strongly and positively correlated with the SDS. The PDI was found to have significant moderate positive correlations with PASS Total, Fear Appraisal and Physiological Symptoms subscales and a strong positive correlation with the SDS. By contrast, the other two disability measures, perceived work ability ratings and compensation status were not significantly correlated with any of the independent or other dependent variables. With respect to the health care utilisation variables, there was a significant moderate positive correlation between number of providers and the PASS Total scale and Physiological Symptoms subscale and between number of medications and the PASS Cognitive Symptoms subscale. Depression (SDS) was strongly and positively correlated with PASS Total, Cognitive Symptoms and Physiological Symptoms subscales, CSQ Catastrophising subscale, PDS- D severity and PDS-Total severity (all \( p < .001 \)). Significant moderate positive correlations were also demonstrated between the SDS and PASS Fearful Appraisals, ASI and PDS-C, PDS-D and PDS-Total severity scores.

Turning to associations between independent variables, neither the PDS-B nor PDS-D severity was significantly correlated with any non-PTSD independent variable. Both the PDS-C and PDS-Total severity only showed significant moderate positive correlations with the ASI. The ASI demonstrated strong positive correlations with PASS Total, Fearful Appraisal, Physiological Symptoms subscales (all \( p < .001 \)) and moderate positive correlations with PASS Cognitive Symptoms, Escape/Avoidance, CSQ Catastrophising and CPCI Guarding subscales (all \( p < .001 \)).
Table 10. Zero-order correlations (*r*) for predictor and outcome variables for the No PTSD group (*n* = 63)

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<td>.53***</td>
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1. *n* = 42 for correlations with PDS. Some participants in the No PTSD group did not complete this scale.
2. *r* = *r* < sub>pb</sub> when correlated with a continuous variable or *r* < sub>b</sub> when correlated with another dichotomous variable.
3. *p* < .05, ** *p* < .01, *** *p* < .001
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$r = r_{PC}$ when correlated with a continuous variable or $r_{DC}$ when correlated with another dichotomous variable.

* $p < .05$, ** $p < .01$, *** $p < .001$
Three separate hierarchical regression analyses were performed to determine the degree to which each group of independent variables improved prediction of pain severity, pain-related disability and depression across the entire sample. The focus was narrowed to these three dependent variables, based on the absence of significant correlations between the other dependent variables and independent variables (see above, Section 5.3.7). Pain severity was included as an independent variable in the regression analysis for pain disability, given the logical and theoretical links between the two. Although the possibility of reciprocal interactions between pain severity and depression and between depression and pain disability is noted, the distinction of these as dependent variables was otherwise maintained for the purpose of clarity. In this way, neither pain disability nor depression served as independent variables in regression analyses. That is, depression was not used to predict pain-related disability nor was pain-related disability used to predict depression.

Entry of independent variables was ordered according to logical and theoretical considerations. Pain severity was given primacy in predicting pain-related disability. Otherwise, ASI was entered first as it represents a potential vulnerability factor, occurring chronologically before other symptoms develop. As a primary Axis I disorder, PTSD diagnosis was entered next (dummy coded), followed by PDS-B, C and D severity scores, in order to determine the relative importance of a diagnosis of PTSD compared to the presence of PTSD-like symptoms. Pain-related anxiety, followed by cognitive coping strategies (CSQ Catastrophising, Praying/Hoping) and then behavioural coping strategies (CPCI guarding and resting) were entered, given theoretical arguments and prior evidence that PASS predicted catastrophising and avoidance behaviours, to determine their contribution beyond ASI and PTSD variables.

Table 11 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $\Delta R^2$ change and $F$ change after entry of each block of IVs regressed on pain severity. $R$ was significantly different from zero at the end of each step. After step 6, with all IVs in the equation, $R = .55$, $F (13, 85) = 2.84$, $p = .002$. Thus, these variables together accounted for a total of 20% (adjusted) of the variability in pain severity.
As can be seen from Table 11, the ASI, the PDS severity scores (entered as a block) and the PASS subscales (entered as a block) all resulted in a significant increment in $R^2$, indicating that each of these blocks of variables uniquely contributed to the prediction of pain severity. The effect sizes, however, were small (addition of PDS and PASS, $f^2 = .11$ and .06, respectively). By contrast, PTSD diagnosis did not reliably improve the prediction of pain severity, after controlling for ASI, however this could be attributed to inadequate power (.46). Similarly, the CSQ subscales and the CPCI subscales (entered as blocks) did not reliably improve prediction of pain severity beyond what could be predicted from the variables entered previously, though limited power is again noted (power = .48 and .54, respectively). Inspection of the coefficients from the final model revealed the PASS Physiological as the best predictor of pain severity.
Table 12 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on the PDI. $R$ was significantly different from zero at the end of each step (all $p < .001$). After step 7, with all IVs in the equation, $R = .77$, $F (14, 84) = 8.48$, $p < .001$. Together these variables accounted for a total of 52% (adjusted) of the variability in the PDI.

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<thead>
<tr>
<th>Variables (n = 98)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj. $R^2$</th>
<th>Chg $R^2$</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ chg</th>
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<td>.31</td>
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<td>.06</td>
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Note. ASI = Anxiety Sensitivity Index, PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptom Scale, CSQ = Coping Strategies Questionnaire, CPCI = Chronic Pain Coping Inventory.

**$p < .01$, ***$p < .001$.

From Table 12, it can be seen that pain severity, the ASI, PTSD diagnosis and the PASS subscales (entered as a block) all resulted in a significant increment in $R^2$, indicating that each of these variables uniquely contributed to the prediction of PDI. The effect sizes ranged from large to small ($f^2 = .45, .08, .12, .14$, respectively). PDS-B, -C and -D severity subscales, the CSQ subscales and the CPCI subscales (entered as
blocks) did not reliably improve prediction of PDI scores, after controlling for the other variables ($r^2 = .02, < .01, .02$, respectively). Notably, the power to detect significant effects for these variables was low (power $= .33, .48, .50$, respectively). Inspection of coefficients from the final model suggested pain severity was the strongest predictor of pain disability.

Table 13 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R, R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on the SDS. $R$ was significantly different from zero at the end of each step (all $p < .001$). After step 6, with all IVs in the equation, $R = .83, F (13, 85) = 14.81, p < .001$. Together these variables accounted for a total of 65% (adjusted) of the variability in the SDS.

Table 13. Sequential Regression of IVs on Self-rating Depression Scale (SDS)

<table>
<thead>
<tr>
<th>Variables (n = 98)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj.</th>
<th>Chg</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ chg</th>
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</table>

Note. ASI = Anxiety Sensitivity Index, PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptom Scale, CSQ = Coping Strategies Questionnaire, CPCI = Chronic Pain Coping Inventory.

*p < .05, **p < .01, *** p < .001
Inspection of Table 13 indicates that the ASI, PTSD diagnosis, the PDS-B, -C and-D severity scores (entered as a block), the PASS subscales (entered as a block) and the CSQ subscales (entered as a block) all resulted in a significant increment in $R^2$, indicating that each of these blocks of variables uniquely contributed to the prediction of scores on the SDS. The effect sizes were large for the ASI and PDS subscales ($f^2 = .56$ and .34, respectively), medium for the PASS subscales ($f^2 = .16$) and small for the CSQ ($f^2 = .09$). The CPCI subscales (entered as blocks) were the only variables to not reliably improve prediction of scores on the SDS, after controlling for other variables. The power for detecting a possible significant effect was low, however, for this variable (power = .15). Based on their coefficients in the final model, PDS-D severity (hyperarousal) and CSQ Catastrophising were the strongest predictors of SDS scores.

5.4 Discussion

This study had several aims in investigating a group of patients with heterogenous chronic pain complaints. First, it set out to examine whether pain patients with PTSD differed from pain patients without PTSD on a range of pre-injury variables (age, gender, education level and exposure to prior trauma). Moreover, the study set out to replicate and extend prior research by considering differences between pain patients with and without comorbid onset PTSD on several measures canvassing a range of characteristics of chronic pain problems. Thus, in conjunction with measures of adjustment that have been typically assessed (e.g., pain severity, health care utilisation, disability, depression) the study also considered differences in levels of pain-related anxiety, anxiety sensitivity and use of coping strategies. Finally the study set out to explore the pattern of relationships between these variables amongst pain patients with and without PTSD.

The demographic characteristics of the overall sample were similar to those reported by other researchers who have dealt with individuals experiencing chronic pain following a traumatic injury (Turk et al., 1996). That is, the typical individual was female, middle aged, married and reasonably well-educated (finished high school) and had experienced pain for several years, which had negatively affected her employment status.

The present study revealed no differences in age, gender or education between those chronic pain patients in the PTSD group and those in the No PTSD group. Although
some studies have found a positive relationship between these demographic factors and the onset of PTSD, several review articles (Brewin et al., 2000; B. L. Green et al., 2000; Yehuda, 1999) have concluded that the overall evidence of their status as risk factors is equivocal, given several contradictory or negative findings by other studies.

According to the present data, there was also no difference between groups in terms of the number of prior traumatic experiences to which individuals had been exposed. This is unusual, given that prior exposure to trauma has been consistently linked to the development of PTSD (Brewin et al., 2000; B. L. Green et al., 2000; Yehuda, 1999). One explanation may be that there was a ceiling effect, as the lifetime prevalence of exposure to a traumatic event for the entire sample in this study was quite high at 72%. This is somewhat higher than the lifetime prevalence rates of being exposed to a traumatic stressor (57%) or a life-threatening accident (21%) that was reported in a national epidemiological study of Australians (Rosenman, 2002). Nevertheless, it is consistent with general findings of a high frequency of prior trauma in chronic pain samples compared to the general population, as reviewed in Chapter 3. More specifically, the findings here regarding the reported frequency of early sexual contact with an older person and sexual assault by a known or unknown perpetrator (15%, 11% and 8% of the total sample, respectively) were certainly within the range, if not a little lower, than the rates reviewed in Chapter 3; up to 48% amongst those with heterogenous pain complaints and up to 67% amongst those with specific chronic pain conditions. The prevalence of exposure to other types of traumatic stressors is not known amongst the population of pain patients. It would be reasonable to conclude, however, that if exposure to a more comprehensive range of traumatic stressors were assessed (as they were in this study), that the overall rate of prior trauma exposure would be higher still.

Importantly, previous research has indicated that a prior diagnosis of PTSD, rather than simply being exposed to trauma, may be a risk factor for developing PTSD in response to a future event (B. L. Green et al., 2000). It is possible that individuals in the No PTSD group may have had either a current or lifetime diagnosis of PTSD, tainting the group classification and potentially their responses to chronic pain. This is particularly relevant in light of the finding that the No PTSD group were more likely than the PTSD group to report a history of early sexual contact with an older person.
Unfortunately, it is only possible to speculate about this prospect as the present study did not include assessment of pre-injury or non-injury PTSD.

The large body of evidence reviewed in Chapter 3 indicated that individuals with PTSD were more likely to have or develop chronic pain. Several researchers have further shown that individuals who experience PTSD after a traumatic injury also report higher levels of chronic pain than patients who have low or no symptoms of PTSD (Geisser et al., 1996; McFarlane et al., 1994; Sherman, 1997; Sherman et al., 2000; Smith et al., 2002; Toomey et al., 1994) and that the severity of PTSD symptoms is related to the severity of pain (Beckham et al., 1997; R. A. Bryant et al., 1999; McFarlane et al., 1994).

The present data did not strongly support such findings, as there was only a non-significant trend toward greater pain intensity in the PTSD group compared to the No PTSD group. Further, the groups did not differ in terms of the perceived number of body locations affected by pain or with respect to pain duration. The latter result is similar to the findings of Asmundson et al. (1998), who also found no difference in pain duration between patients with and without PTSD following a work-related injury. Additional analyses using multiple regression showed that PTSD diagnosis was not a significant predictor of pain severity, although the severity of PTSD hyperarousal symptoms did contribute to a multiple regression model that predicted 20% of the variance in pain severity.

One point of distinction between the present study and many of the studies that reported a positive relationship between PTSD and pain severity is the intervening period since the traumatic stressor. In the present study, the majority of participants sustained their traumatic injury less than five years ago. By contrast, participants in other studies often had a comparatively long interval since their index trauma, potentially fifty years or more, where it related to their experience of the Holocaust or childhood physical or sexual abuse (Geisser et al., 1996; Sherman, 1997). Further, for the PTSD group in the present study, chronic pain followed directly from the index trauma, whereas this connection was not explicit in prior studies, where pain may have first developed years after the traumatic event (Geisser et al., 1996; Sherman, 1997). The period of time since injury may also be important given indications that self-rated medical problems/pain was a better independent predictor of PTSD at three years than
three months post-injury (Mayou & Bryant, 2002; Mayou et al., 2002). As argued by Spertus (2000), over the course of time pain could become attached to the trauma network much later via processes of higher-order conditioning and stimulus generalisation. Alternatively, physiological mechanisms (e.g., central hypersensitivity) may require time to develop. Thus, if the PDS had not been modified to force a focus on the index injury, it is possible that stronger support for a link between PTSD and pain variables may have been found.

Even though ratings of pain severity were not clearly different, a number of differences emerged in this study in terms of the way in which patients with and without PTSD reacted to their pain. Patients with PTSD had higher health care utilisation in consulting with more treatment providers and taking more medications than their non-traumatised counterparts. While the additional treatment could be for PTSD-related symptoms, the PTSD group were not only more likely to be prescribed antidepressants, they were also more likely to be taking non-steroidal anti-inflammatories. Because some anti-inflammatories are available as over-the-counter medications (e.g., Nurofen, Ibuprofen), it is not clear whether the increased use of anti-inflammatories by patients with PTSD is an individual coping decision (e.g., using medication as an attempt to alleviate or avoid pain), an iatrogenic effect or a true reflection of altered biological processes (e.g., increased inflammation as a function of PTSD). The latter is possible as increased inflammatory processes have been linked to chronic stress (Butler & Moseley, 2003). Further, the increased use of antidepressants in the PTSD group may explain the lack of significant difference in pain severity between the two groups, as tricyclic antidepressants (e.g., amitryptaline) have neurolytic effects (which may decrease pain via desensitisation of the central nervous system) and operate synergistically with analgesics (G. Griffiths, 2005, personal communication).

With respect to other indicators of adjustment, the current results demonstrated that patients with PTSD perceived greater overall disability, were more negative in their ratings of prognosis and ability to work, were more depressed and more likely to be receiving or seeking compensation than patients who did not have PTSD. Indeed, the multiple regression showed that PTSD diagnosis explained a significant proportion of variance in pain disability, and both PTSD diagnosis and severity explained a significant proportion of variance in depression.
The level of depression reported in the PTSD group ($M = 55.59$) approximated levels reported previously on the SDS by a chronic pain sample ($M = 58.29$) and are substantially higher than the levels reported within the general population ($M = 42.45$) (R. Taylor, Lovibond, Nicholas, Cayley, & Wilson, 2005). By contrast, the No PTSD group’s reported level of depression ($M = 43.26$) was more in line with the general population and certainly lower than would be expected given they are suffering from chronic pain. It is not clear why this pattern occurred, however, it is possible that Taylor et al.’s (2005) normative sample also contained a large number of individuals with comorbid PTSD, inflating the rate of depression across chronic pain patients overall, given the greater risk of depression in this group.

The finding of greater depression associated with PTSD compared to No PTSD is interesting in light of findings of Beckham et al. (1997), who found no significant difference in depression between PTSD patients with and without chronic pain. Taken together the two studies could suggest that depression is more strongly associated with PTSD than with chronic pain. On the other hand, in Beckham et al.’s study both groups demonstrated high levels of depression (90% scored higher than 70T on the MMPI), which may have produced a ceiling effect, occluding real differences. Another possibility is that the link between PTSD and depression is inflated by overlapping symptoms/ shared method variance. This is plausible as multiple regression analyses showed SDS scores were best predicted by PDS-D scores (a scale that includes irritability, sleep disturbance and impaired concentration, which overlap with depressive symptoms).

As a diagnosis of PTSD was the only measured variable significantly correlated with compensation status, it is possible that positive compensation status negatively influenced ratings of disability and prognosis in this group. Alternatively, it is possible that individuals with PTSD are more severely affected by their injury and therefore more inclined to be involved with or seek compensation. Given the cross-sectional nature of this study, it is not possible to distinguish here between such causal explanations. The small magnitude of the effect size suggests, however, that factors other than PTSD are more likely to account for whether or not an individual receives or seeks compensation. On the other hand, combining those who were receiving compensation with those who were in the process of seeking compensation could have diluted the strength of the relationship between PTSD and compensation (Turk &
Okifuji, 1996). These possibilities could be more thoroughly examined in future research via prospective studies.

In terms of coping strategies and reactions to pain, the PTSD group in the present study reported higher levels of anxiety sensitivity and pain-related anxiety, including more self-reported cognitive symptoms, fearful appraisals, physiological arousal and escape/avoidance behaviours in response to pain, compared to the No PTSD group. They were also more likely to engage in maladaptive or passive coping strategies (e.g., catastrophising and praying/hoping) and less likely to use the adaptive cognitive strategy of coping self-statements. The PTSD group were also more likely than the No PTSD group to use avoidant coping strategies such as guarding and resting, although they did not differ on constructive behavioural strategies (e.g., relaxation, exercise/stretching). Overall, these findings point to a strong tendency toward negative appraisal and maladaptive pain coping in the PTSD group. Indeed, PTSD severity correlated with the degree of pain-related anxiety (particularly cognitive and physiological symptoms), resting behaviours, depression, catastrophising and anxiety sensitivity. Some of these associations are not surprising given the potential overlap of the variables with classic PTSD symptoms of avoidance, numbing and hyperarousal. Due to the reliance on self-report measures it is not possible to determine how much of this is simply due to shared-method variance.

Nevertheless, PTSD diagnosis and severity, anxiety sensitivity, pain-related anxiety and maladaptive coping strategies, in combination with pain severity, together explained over half of the variance in pain-related disability. In addition, the same factors (excluding pain severity) significantly predicted depression, explaining 65% of the variance in scores. This is consistent with other research that tends to highlight that the presence of maladaptive coping strategies, rather than a lack of adaptive strategies, differentiates between chronic pain patients with high or low disability and depression (Jensen et al., 1991a). Overall, the variables measured in this study could account for much more of the variance in pain-related disability and depression than in pain severity. This suggests a difference in predicting chronic (disability and depression) as opposed to acute issues (pain severity) related to persistent pain and supports the notion that the impact of PTSD on chronic pain problems is served through difficulties with adjustment and management rather than the pain severity itself.
It has been argued that the apparent link between PTSD and chronic pain problems is due to the vulnerability towards anxiety sensitivity that the conditions share (Asmundson et al., 2002). The present pattern of results provides only partial support for this proposition. First, as expected, chronic pain patients with PTSD reported higher levels of anxiety sensitivity than pain patients without PTSD. Second, and consistent with Asmundson et al.’s prediction, anxiety sensitivity, when entered first into multiple hierarchical regression models, made a significant contribution to the prediction of pain severity, pain-related disability and depression. Notably, however, PTSD diagnosis and/or PTSD symptom severity, when entered subsequently, explained additional variance in these variables, beyond that already explained by anxiety sensitivity. Thus, while anxiety sensitivity may indeed underlie both PTSD and chronic pain, the latter result indicates that the relationship between the two conditions involves more than just a shared vulnerability to negatively interpret anxiety/somatic sensations.

A number of researchers have speculated in their discussion of results or the development of theoretical models that each of the PTSD symptom clusters could have distinct associations with chronic pain resulting in mutual maintenance of the conditions (Asmundson et al., 2002; Sharp & Harvey, 2001). For example, it has been argued that the presence of chronic pain could operate as either a somatosensory intrusion or a reminder of the traumatic event that provokes emotional and physiological reactivity (see Chapter 2). Although several researchers have demonstrated a relationship between re-experiencing symptoms and pain severity (Beckham et al., 1997; Koren et al., 2005) or physical symptoms generally (McFarlane et al., 1994), the present data do not support such a relationship. That is, contrary to those earlier studies, re-experiencing symptoms was not predictive of pain severity in this study. Although PASS physiological symptoms did predict pain severity, there was no significant relationship between re-experiencing symptoms and PASS physiological symptoms, further undermining the proposed link with PTSD.

Alternatively, it has been argued that attempts to avoid painful reminders of the traumatic event (e.g., situations, activities or movements) may result over the longer term in increased pain, depression and disability, as well as the maintenance of PTSD by limiting opportunities for emotional processing or desensitisation of trauma-related stimuli (Ehlers & Clarke, 2000; Sharp & Harvey, 2001; Spertus, 2000). This is discussed further in Chapter 4. Indeed, in support of this, PTSD re-experiencing and
avoidance and numbing (B and C cluster) severity significantly contributed to the prediction of both pain-related disability and depression. Interestingly, C cluster severity was significantly correlated with all aspects of pain-related anxiety, although it was more strongly correlated with pain-related physiological arousal than with pain-related avoidance (PASS Escape/Avoidance, CPCI Resting) and negative appraisal (PASS Fearful Appraisal, ASI, Catastrophising). One possible interpretation of this pattern is that PTSD and chronic pain maintain each other via respondent conditioning (Turk & Okifuji, 1996). Physiological arousal that occurs in response to pain (as a trauma-related cue) may lead to avoidance of activities and situations associated with the original injury, especially where the arousal is misconstrued or misattributed to injury or harm rather than fear or anxiety. In turn, this avoidance could maintain the conditioned response and prevent the interpretation that physiological arousal is an indicator of harm from being extinguished (Norton & Asmundson, 2003; Philips, 1987; Sharp, 2001).

A third hypothesis is that general hyperarousal due to PTSD contributes to pain severity via chronic elevation of muscular tension and autonomic activation (Burns et al., 1997; Sharp & Harvey, 2001). Data from this study provided some support for this link, as the significant contribution of PTSD severity to prediction of pain severity was primarily due to PDS Hyperarousal scores. On the other hand, this symptom cluster was also significantly correlated with depression, anxiety sensitivity, catastrophising and pain-anxiety cognitive symptoms. Arguably, the common thread strung between these variables is a bias of negative interpretation. It is therefore possible that the relationship between PDS Hyperarousal and pain is driven by a perception of threat rather than the physiological arousal itself. That is, the identification of hyperarousal as harmful and threatening might be more important in maintaining the problem (both PTSD and chronic pain) than the direct effect of arousal in lowering pain threshold or tolerance. Alternatively, the relationship between PTSD hyperarousal severity and other variables could have simply reflected overlapping symptoms, as discussed above (e.g., impaired concentration: PASS Cognitive and SDS; irritability and sleep disturbance: SDS).

Finally, low scores on the ignoring pain or diverting attention subscales of the CSQ could be interpreted as a sign of hypervigilance to pain. Importantly, there was no difference in the two groups’ self-reported use of these cognitive strategies. This is
contrary to a fourth hypothesis regarding increased hypervigilance to chronic pain in the context of PTSD. It would be unwise to reject, however, the hypotheses on the basis of self-report alone, which is likely to be a less reliable measure of hypervigilance than objective measures borrowed from cognitive psychology (e.g., Stroop test, dot-probe test) (Crombez, Van Damme, & Eccleston, 2005).

5.4.1 Limitations and Future Directions

Although this study improved upon previous studies by utilising standardised and comprehensive measures of pain and PTSD and examining a heterogenous sample of pain patients, it has its limitations. First, the study relates to a self-selected population and sample. Participants were both patients presenting for treatment (self-selecting population) and volunteers responding to an invitation to participate in research (self-selecting sample). Indeed, given higher rates of health care utilisation in the PTSD group found here and in other studies (Calhoun et al., 2002; Deykin et al., 2001) problems due to sampling bias are certainly possible. To this end, it would be beneficial for future investigations to consider non-treatment seeking individuals and consecutive attendees for treatment to permit better generalisation. An interesting problem occurred during data collection, however, whereby many treatment providers became strict “gatekeepers”, restricting access to potential participants for various reasons including an assumption that their patients would not want to participate. Interestingly, anecdotal evidence gathered during testing suggested that many participants found the experience beneficial, particularly those for whom psychological factors had been neglected in the course of treatment.

A further limitation is that the study relied solely on self-report measures, raising questions of diagnostic and symptom reliability. Most importantly, there is difficulty distinguishing shared symptomatology from symptomatology due solely to or better accounted for by one disorder alone. For example, it was not possible to establish whether symptoms such as sleep disturbance, concentration difficulties, irritability and activity changes were due to pain, PTSD or both (O'Donnell et al., 2003). Ideally, it would be beneficial to adopt multiple measurement methods to address possible reporting bias and confirm PTSD diagnosis, for example, by using structured clinical interviews, file review, physiological measures of arousal and behavioural tests. Finally, because the study was a cross-sectional design in which data were gathered at one time
point, it is not possible to draw conclusions regarding the causal direction of associations. The observed relationships can however form the basis for subsequent experimental studies.

In summary, the present data supported the overarching hypothesis that when chronic pain coincides with and arises from the same event as PTSD the emergent clinical presentation is distinct from and more complex than simple chronic pain. The correlational nature of this data, however, leaves the causal nature of the relationships ambiguous. Thus it is not entirely clear what it is about simultaneously experiencing PTSD and chronic pain that leads individuals to seek out more health care, perceive greater disability, catastrophise and show greater fear and anxiety about pain, adopt more avoidant coping and feel more depressed than individuals who experience chronic pain alone. Nonetheless, the data alludes to cognitive, behavioural and physiological links between PTSD and pain. The strongest support was for the notion that PTSD impacts most on pain coping and adjustment rather than pain severity itself, although there was some support for an association between pain severity and the inherent hyperarousal of PTSD and for the existence of an underlying interpretation bias. The studies presented in subsequent chapters were designed to explore these differences and connections more directly, using broader methodology including experimental manipulation to improve validity and reliability of results.
CHAPTER 6

FURTHER EXAMINATION OF PAIN-RELATED ANXIETY, FEAR-AVOIDANCE AND INDICATORS OF ADJUSTMENT IN PATIENTS WITH CHRONIC PAIN AND PTSD

Evidence from Study 1 suggested that a diagnosis of comorbid onset PTSD does impact on the way in which an individual adjusts to and manages chronic pain. Underlying anxiety sensitivity, pain-related anxiety, catastrophising and fear-avoidant coping were higher amongst individuals who experienced PTSD comorbid with chronic pain. In addition, indicators of adjustment suggested a much poorer outcome for these individuals. That is, these individuals consulted more professions, took more medication, were less likely to be working and more likely to be seeking or receiving compensation than their non-traumatised counterparts. Not surprisingly, therefore, they reported greater pain-related disability and depression compared to patients with chronic pain alone. There was also some evidence of greater pain severity in chronic pain patients with PTSD, which, as a non-significant trend, warrants further investigation.

A primary limitation of Study 1, however, was the sole reliance on self-report measures taken at cross-section. This may have inflated the effects between variables, due to shared method variance. Use of self-report also compromised the study’s ability to properly identify, differentiate and exclude symptoms, potentially confounding the diagnosis of PTSD. The following study was therefore designed to further explore the effects of PTSD found in Study 1, with the specific purpose of examining whether they would be upheld in the face of more rigorous methodology.

6.1 Study 2

The primary purpose of Study 2 thus was to replicate and extend findings of Study 1 and earlier research highlighting the negative impact of PTSD on chronic pain management. In doing so, the study sought to use more rigorous methodology, including diagnostic clinical interviews and contemporaneous measures of affect and physiological reactivity, to address some of the limitations of prior research.
6.1.1 Diagnosis and Classification of PTSD

In contrast to Study 1, which classified individuals into PTSD and No PTSD groups on the basis of their self-report on the PDS, the present study aimed to improve confidence in the diagnostic classification through use of the PTSD-module of the Structured Clinical Interview for DSM-IV (SCID) (R. L. Spitzer et al., 1990). Application of clinical judgment provides greater assurance that symptoms meet criterion specification (e.g., intrusive thoughts rather than just voluntary ruminative thought about an injury) and the ability to distinguish symptoms due to physical rather than psychological problems (e.g., loss of interest due only to post-injury physical limitations).

In addition, the study sought to address a confounding influence in previous research by differentiating (on the basis of a client’s self-reported history) the origin of symptoms of PTSD. That is, patients were assessed as to whether their PTSD symptoms arose from (1) a traumatic injurious event, (2) a prior traumatic event, temporally unrelated to the onset of their current pain episode and or (3) post-event factors, including severe/uncontrolled pain (Schreiber & Galai-Gat, 1993).

6.1.2 Assessment of Distress

In Study 1, distress was operationalised as depression and measured by the Zung SDS. This self-report questionnaire is problematic in the context of chronic pain, however, as it includes a range of somatic symptoms (e.g., insomnia, fatigue, changes in appetite and weight, loss of libido). Although somatic symptoms have been traditionally associated with depression, they may be attributable to a cause other than depression, such as physical illness (de C. Williams & Richardson, 1993; Gamsa, 1994b; Pincus, Callahan, Bradley, & al., 1886; R. Taylor et al., 2005). As Taylor et al. (2005) pointed out, “in pain patients, somatic symptoms could result from a specific injury, the unique experience of pain, the physical limitations imposed by pain, or the effects of medication, as well as from depression” (p.92). Indeed, somatic symptoms show a stronger relationship with pain severity than other depressive items on the SDS for patients with chronic pain (Estlander, Takala, & Verkasalo, 1995). Patients with chronic pain also endorsed somatic symptoms on the SDS in preference to other symptom items and rated them more strongly than did individuals attending a psychology clinic, indicating that the symptoms were more likely due to pain-related factors than depression (R. Taylor et al., 2005). In particular, items relating to
psychomotor retardation, disturbed sleep, constipation, irritability and fatigue were scored or ranked higher than other items by chronic pain patients.

Two of the options proposed to overcome this problem — omitting or substituting for the somatic items from self-report questionnaires (Wilson et al., 2002) or distinguishing symptoms using clinical judgment (Geisser, Roth, & Robinson, 1997) — have met with criticism (Geisser et al., 1997; R. Taylor et al., 2005). Taylor et al. (2005) instead argued for the use of an instrument which empirically discriminates between different aspects of distress, such as the Depression Anxiety Stress Scales (DASS) (Lovibond & Lovibond, 1995), to guard against over-inflated estimates of depression in chronic pain samples. Another advantage of using a scale, such as the DASS, in the present study, is that it also provides a broader measure of distress by assessing symptoms of anxiety and stress, in addition to depression (cf. Study 1).

6.1.3 Assessment of Fear of Movement/Reinjury

Theoretical models of the maintenance of chronic pain propose a primary role for fear of reinjury, movements and activities associated with pain (Lethem et al., 1983a; Vlaeyen et al., 1995a; Vlaeyen et al., 1995b; Vlaeyen & Linton, 2000). Arguably, fear associated with the original injury primes a traumatic reaction and the development of PTSD (Criterion A, DSM-IV TR). In addition, if pain is a reminder of trauma, then patients with comorbid PTSD may be more fearful of pain and movements believed to trigger acute pain sensations than individuals without PTSD. If present, it is expected that this fear and associated avoidance will directly impact on functional limitations and disability. It may also prevent activation of the fear network, which in turn will maintain symptoms over time.

It is thus of interest to determine whether individuals with PTSD demonstrate higher levels of fear of movement/reinjury in conjunction with their experience of chronic pain. Subjective perceptions have received significant attention in the literature on PTSD (e.g., March, 1993), thus it is important to consider measures of fear of movement/reinjury, both in general and specific terms. It is possible that measures examining responses to all exercise or general physical activity may be too broad, missing idiosyncratic fears detected by reference to more specific movements and activities of daily living. Prior research investigating fear of movement/reinjury has also failed to assess whether ratings of harmfulness for specific movements correspond with
the frequency with which the specific movement is performed during daily life. That is, it is argued that exposure to the movement will decrease ratings of harm (Vlaeyen et al., 2001), however, it is possible that individuals are performing the activity and that ratings of harm are driven by the pain experienced (even if that pain does not reflect actual damage).

6.1.4 Psychophysiological Reactivity Associated with PTSD and Chronic Pain

Much of the research regarding pain-related fear and anxiety has focused on self-report of physiological responses to pain (McCracken et al., 1996). However, it is common to detect asynchrony between self-reported and actual physiological responses (P. J. Lang et al., 1983), with most of the variance in correlations between the two unexplained. It is therefore possible to argue the need for determining how well self-report measures of pain-related anxiety and fear correspond with physiological reactivity, generally and in response to pain-related cues. In turn, it is necessary to investigate whether actual physiological reactivity predicts pain severity, disability and distress, given theoretical claims that physiological reactivity is the mechanism through which fear impacts on these factors (Norton & Asmundson, 2003). Further, as autonomic hyperarousal is a prominent feature of the clinical presentation of PTSD, it is worth investigating if there is a differential impact of physiological arousal in chronic pain patients with and without PTSD.

6.1.4.1 Psychophysiological reactivity and PTSD

Research indicates that the degree of cardiovascular reactivity initially experienced by an individual soon after a traumatic event strongly predicts the development of chronic PTSD (Shalev & Rogel-Fuchs, 1993; Shalev, Sahar et al., 1998). PTSD has also been associated with increased sympathetic activity (elevated heart rate and low frequency components of heart rate variability, LF-HRV) at baseline compared to individuals with panic disorder and normal controls (Cohen et al., 2000). In addition, individuals with PTSD show abnormal physiological responses, such as elevated heart rate, galvanic skin response and blood pressure, in response to stimulation across a range of studies, which differentiates them from individuals without PTSD (Shalev & Rogel-Fuchs, 1993). It has been argued that it is the timeless quality of such physiological reactivity (R.K. Pitman, Orr, & Shalev, 1993) that creates the perception of current threat (Ehlers & Clarke, 2000).
In their early critical review, Shalev and Rogel-Fuchs (1993) suggested physiological studies of PTSD can be broadly grouped into three, based on their method of eliciting a psychophysiological response: (1) studies that employ externally presented stimuli, (2) studies that rely on evoked internal stimuli (typically imagery) and (3) those that evaluate the acoustic startle reflex (ASR). Notably, many of the studies described rely on small sample sizes ($n = 5$ to $13$ per cell).

As highlighted by Shalev and Rogel-Fuchs, the first kind of study has produced findings that veterans with PTSD exhibit marked elevation across a range of psychophysiological measures, such as heart rate, respiration rate, blood pressure, skin conductance and norepinephrine, in response to visual and auditory presentations of reminder cues of combat (e.g., artillery sounds, combat films) (Blanchard, Kolb, & Pallmeyer, 1982; Dobbs & Wilson, 1960; Malloy, Fairbank, & Keane, 1983; McFall, Murburg, & Ko, 1990; Pallmeyer, Blanchard, & Kolb, 1986). These elevations have been demonstrated in comparison to various control groups, including combat veterans without PTSD (Dobbs & Wilson, 1960; Malloy et al., 1983; McFall et al., 1990), veterans and non-veterans with other psychiatric disorders (Pallmeyer et al., 1986) and healthy controls (Blanchard et al., 1982; Dobbs & Wilson, 1960). There are some contradictory findings, for example, one study found that military-trained controls demonstrated a greater skin conductance response to affectively charged slides than did individuals exposed to combat (Najstrom & Hogman, 2003). This effect occurred irrespective of the mode of presentation, either subliminal or supraliminal. One explanation for this contradictory effect is the distinction between trauma exposure and actual diagnosis of PTSD. Another reason, however, for the lack of reactivity may have been that the study used generic slides, which may have lacked personal relevance (see below).

The attraction of this first group of studies is their use of external stimuli, which can be standardised and manipulated across participants. The findings have been used to argue that PTSD is a disorder of conditioned responding (Shalev & Rogel-Fuchs, 1993; van der Kolk, 1996). That is, natural fear and physiological arousal (unconditioned response, UCR) in response to the original traumatic event (unconditioned stimulus, UCS) is followed by similar responses (conditioned responses, CR) to subsequent external cues (conditioned stimuli, CS). In this way, pain and or movements may
operate as either an unconditioned or conditioned stimulus that provokes significant physiological arousal.

The second kind of study has shown that personalised imagery of trauma, rather than standardised cues, elicits large heart rate, skin conductance and EMG responses in veterans with PTSD but not in their non-traumatised counterparts (Orr, Pitman, Lasko, & Herz, 1993; R. K. Pitman et al., 1990). These responses are also specific to PTSD rather than anxiety disorders per se (Pitman et al., 1990). Using this approach, similar findings for heart rate and EMG also have been demonstrated for survivors of childhood sexual abuse (Orr, Lasko et al., 1998) motor vehicle accidents (Blanchard et al., 1994) and heterogenous non-combat events, such as rape, road accidents and terrorism (Orr et al., 1993; Shalev, Orr, & Pitman, 1993). The hyperresponsiveness appears specific to trauma-related material and does not generalise to other stressors, such as mental arithmetic (Blanchard, Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986; Orr, Meyerhoff, Edwards, & Pitman, 1998), imagery of personal stressful life events (Orr et al., 1993; R. K. Pitman, Orr, Forgue, de Jong, & Clairborn, 1987; Shalev et al., 1993), postural change or cold pressor challenge (Orr, Meyerhoff et al., 1998; Spertus, 2000). Several studies have found contradictory psychophysiological responses, suggesting inhibited reactivity in the face of stressors. However, during recall of trauma or stressful situations, individuals with PTSD failed to respond with an increase in heart rate or LF-HRV, unlike individuals with panic and controls who demonstrated such an increase.

The benefit of this second group of studies is their ability to access the subjective experiences of individuals, although this is dependent on the individual’s ability to generate and control the imagery according to instruction (Shalev & Rogel-Fuchs, 1993). The studies follow the work of Lang (P. J. Lang, 1977; P. J. Lang et al., 1983) and the idea that memories are stored as “associative networks”, including sensory input, interpretative meaning and physiological responses. In this way, traumatic imagery may act as a cognitive conditioned stimulus that reinforces conditioning in the absence of external stimuli (Shalev & Rogel-Fuchs, 1993). Similarly, “remembered” pain may become part of this associated network, reinforced by physiological reactivity, whether conditioned or unconditioned.

Greater physiological reactivity and impaired habituation has been well illustrated for individuals with PTSD by the third approach, investigating the acoustic startle reflex
Veterans with PTSD display an amplified muscular startle response to noise bursts, for example, in contrast to veterans without PTSD (Shalev et al., 2000). A study of 57 women with current or lifetime PTSD related to childhood sexual abuse found that those with current PTSD displayed higher resting heart rates compared to those who had no history of PTSD (Metzger et al., 1999). In addition, compared to people who had never experienced PTSD, individuals with current and lifetime PTSD displayed larger accelerative heart rates and impeded habituation of skin conductance responses when exposed to startling tones (Metzger et al., 1999) and trauma-related stimuli (Orr, Lasko et al., 1998).

The similarity of responses across individuals with current and past PTSD suggests that elevations in reflexive startle mark a PTSD trait rather than a state (Shalev & Rogel-Fuchs, 1993; van der Kolk, 1996). Because the stimuli are “elemental”, non-specific to trauma experiences and trigger a reflexive reaction driven by limbic circuitry, some have argued that PTSD results in altered CNS responsiveness, in addition to conditioned emotional responses (Shalev et al., 1996; van der Kolk, 1996). This altered CNS responsiveness may affect reactivity to pain. Further, individuals with PTSD appear to have difficulty discriminating intense but irrelevant stimuli and regulating their affective and physiological response to such stimuli (Shalev et al., 1996; van der Kolk, 1996). This may be particularly problematic for individuals with comorbid pain, which is intense and ever present, but not biologically threatening.

6.1.4.2 Psychophysiological reactivity and acute pain

Acute pain induced in healthy controls is associated with a range of measures of physiological reactivity. An interesting interaction between blood pressure and pain has been established whereby the correlation between blood pressure and pain is negative amongst hypertensive individuals but positive amongst normotensive individuals. Further, when elevated blood pressure is experimentally induced, borderline hypertensive individuals experience an analgesic effect, whereas normotensive individuals show a hyperalgesic effect (Flor, Miltner, & Birbaumer, 1992).

Al’Absi, Petersen and Wittmers (2002) found both systolic and diastolic blood pressure increased in healthy controls during a 90-second, pain inducing cold pressor task. High resting systolic blood pressure was correlated with lower reported pain intensity, consistent with prior studies involving hypertensive individuals that
demonstrated a reduced sensitivity to acute pain, however, the effect was only present in females. On the other hand, blood pressure recorded during the cold pressor task was not correlated with reported pain intensity. By contrast, Caceres and Burns (1997) found that healthy controls did not display any significant increase in mean blood pressure or heart rate from resting baseline to a cold pressor tolerance task that lasted up to 5 minutes duration (Caceres & Burns, 1997). Further, there were no significant correlations between the two resting physiological measures and pain threshold or tolerance during the task. However, when a general stressor (i.e. mental arithmetic) preceded the task, participants who demonstrated high mean blood pressure and heart rate reactivity in response to the stressor, subsequently demonstrated lower pain threshold and tolerance. Despite this, subjective reports did not show evidence of any differences in perceived pain severity.

Finally, other studies have shown that although heart rate tends to increase during acute pain, it is more strongly correlated with subjective ratings than objective features of pain (Flor, Fydrich, & Turk, 1992; Hampf, 1990; Moltner, Holzl, & Strian, 1990). Acute pain is also associated with a rise in skin conductance (Dowling, 1982). One possible explanation for this is activation of the “fight/flight” response where pain is interpreted as an indication of threat to self.

6.1.4.3 Psychophysiological reactivity in chronic pain patients

As reviewed in Chapter 2, several studies have directed attention to the impact of “stress” on physiological responding in chronic pain patients. In a series of studies, Flor, Turk and Birbaumer (1985) found similar heart rates and skin conductance between patients with chronic low back pain and healthy controls during resting baselines and a range of stressors, such as a brief cold pressor task, mental arithmetic, discussion of personally stressful events and pain episodes (Flor et al., 1985). Lower heart rate variability was found for chronic pain patients compared to controls, however, during a longer cold pressor task (Arntz et al., 1991) and in response to personally relevant stressful images (Flor, Fydrich et al., 1992). By contrast, site-specific responding on EMG measures, suggested that the effect of stress manifests in muscular rather than autonomic reactivity in chronic pain patients (Flor, Fydrich et al., 1992).
6.1.4.4 Psychophysiological reactivity and pain-related fear

Vlaeyen and Linton (2000) specifically predicted from their model that “pain-related fear will be associated with increased psychophysiological reactivity, when the individual is confronted with situations that are appraised as ‘dangerous’” (p. 319). So far, however, minimal attention has been directed toward the specific association of psychophysiological reactivity and pain-related anxiety and fear of movement/reinjury in chronic pain patients and, in the few studies that have addressed this issue, the results have not revealed a strong autonomic effect.

McCracken et al. (1996) found self-reported physiological arousal (PASS – Physiological symptoms) predicted greater pain severity and more physical complaints in a heterogenous sample of chronic pain patients (McCracken, Goetsch, & Semenchuk, 1998; McCracken et al., 1996). Similarly, in a longitudinal study of 95 patients with rheumatoid arthritis, one group found that four items extracted from the PASS – Physiological symptoms subscale explained a significant portion of the increase in pain severity after 12 months, even after controlling for initial pain levels, demographic factors, disease activity and neuroticism (Evers et al., 2001). It cannot be assumed, however, that self-report provides an accurate reference of actual physiological responding. This point is reinforced by Evers et al.’s (2001) comment that only 40% of their participants reported at least “sometimes” experiencing physiological reactions to pain. Indeed, high reports of physiological reactivity may simply reflect an interpretation bias or hypervigilance to somatic symptoms, rather than any real difference in reactivity. Alternatively, false negative self-reported reactivity (i.e. low self-report in the face of high responding) might be attributable to other factors, such as poor insight, avoidance or dissociation. Another possibility, of course, is that the systems are differentially activated by stimuli, such that one system could be activated without activation of the others (P. J. Lang et al., 1983).

Three studies have specifically investigated the relationship between actual physiological reactivity and fear of movement/reinjury. Vlaeyen et al. (1995, Study 2) investigated the relationship between heart rate, skin conductance and fear of movement/reinjury during a physical task (lifting a 5.5kg bag). They found no significant correlation between the two autonomic measures and self-reported fear of movement/reinjury (measured by the TSK). Further, there was no obvious reactivity between baseline, anticipation or performance of the task. Vlaeyen et al. offered little in
the way of an explanation of the null findings, speculating only that behavioural avoidance might have been instigated before the onset of physiological arousal.

In another experimental study, Vlaeyen et al. (1999) exposed patients with chronic low back pain to a 6-minute video of a confederate performing vigorous activity. To enhance the threat potential, the participants were told they would subsequently perform the activity. This time the focus of interest was EMG reactivity from baseline (watching a neutral documentary) to exposure. Although participants reported subjective increases in tension, muscular reactivity actually decreased, albeit less so in fearful participants. Vlaeyen et al. (1999) concluded that the experimental context had created increased muscle tension at baseline, which abated more readily in the non-fearful participants. Consistent with the findings of Flor et al. (1992), fear of movement/reinjury only predicted reactivity of the spinal musculature, reflecting symptom-specificity, except in patients with high negative affect, who also displayed reactivity in non-specific musculature. There was also a trend towards larger ASR in the high fearful patients when noise bursts were delivered in the period between the imaginal exposure and actual performance (Beisiegel, 1997, as cited in Vlaeyen & Linton, 2000).

The relative absence of data regarding the relationship between autonomic arousal, pain-related anxiety and fear of movement/reinjury thus demands further investigation amongst individuals with chronic pain.

6.1.4.5 Psychophysiological reactivity in individuals with PTSD and chronic pain

Because of the possibility of asynchrony between the cognitive-affective, behavioural and physiological response systems associated with pain-related anxiety and fear, it is possible that not all systems are activated and or that different factors might maintain activation of the different systems. Studying the relationships between the different response systems amongst chronic pain patients with and without PTSD could thus provide improved understanding of the specific mechanisms underlying the comorbid presentations.

In summary, the findings reviewed above raise several potential problems for individuals with PTSD who also have chronic pain. As a reminder cue, pain may result in elevated physiological responding (Section 6.1.4.1), beyond the autonomic arousal
typically associated with the pain experience (Section 6.1.4.2). Alternatively, re-experiencing symptoms (as stressors) and the general hyperresponsiveness in individuals with PTSD may impact on pain perception (Sections 6.1.4.1 and 6.1.4.3). Finally, pain perception may be influenced via higher levels of pain-related anxiety and fear of movement/reinjury, if these are associated with altered physiological reactivity. As yet, no systematic study of these possibilities has been conducted.

6.1.5 Aims and Hypotheses

The aim of Study 2 was thus to replicate findings of Study 1, with improved methodology, and to extend the focus to consider whether chronic pain patients with and without PTSD exhibit different patterns of physiological reactivity. First, it was expected that physiological hyperarousal during description of personal trauma during clinical interview would be higher in patients with comorbid onset PTSD than patients without PTSD. Second, it was expected that chronic pain patients with a concurrent diagnosis of PTSD would exhibit higher pain-related anxiety and fear of movement/reinjury (general and specific) than chronic pain patients who do not meet a diagnosis of PTSD. Third, it was anticipated that self-report measures of pain-related anxiety and fear of movement/reinjury would be correlated not only with each other, but also with the degree of physiological hyperarousal upon exposure to the activity, and the frequency with which, such activities are performed. Finally, it was expected that the PTSD group would report more problems adjusting to chronic pain than non-traumatised pain patients, as reflected in adjustment indicators such as pain severity, health care utilisation, pain-related disability and distress, and that these would be influenced via different aspects of pain-related anxiety and fear.

6.2 Method

6.2.1 Participants

Patients were considered eligible for the study if they were aged between 18 and 65 and had experienced persistent pain for at least six months. As in Study 1, the criterion pain duration was six months to encompass the DSM IV TR definition of chronic Pain Disorder and PTSD. Exclusion criteria given to referral sources included (1) serious head injury or burns; (2) current major psychiatric disorder (e.g., psychosis or bipolar disorder), (3) predominant substance abuse; (4) pregnancy; (5) use of antihypertensives and (6) insufficient comprehension of English to permit completion of self-report measures. None of the individuals referred to the study met these exclusion criteria.
A convenience sample of 33 voluntary participants were recruited from patients with heterogeneous chronic pain complaints referred to a private multidisciplinary cognitive-behavioural pain management program (CIMC, \( n = 19 \)), private psychologists \( (n = 7) \) and physiotherapists \( (n = 7) \) in the Australian Capital Territory. Four individuals who initially agreed to participate withdrew from the study prior to their first attendance, citing illness or work or family commitments, leaving a total of 29 participants.

Referring agents were unable to or did not provide a record of the number of individuals who were eligible for the study but were not informed or the number of individuals who were informed but declined to participate. The recruitment process was slower than expected due to various factors, including workloads of referral sources and a 50% reduction of MVAs in the ACT over the course of recruitment.\(^{10}\)

Demographic data for the entire sample is presented in Table 14. The total sample consisted of 18 females and 11 males. Participants' ages ranged from 19 to 61 years, although they were predominantly middle aged. The majority were non-indigenous Australians and all were long term residents (>17 years) of Australia. Only 17% were from non-English speaking backgrounds. Most were in a relationship and lived with others. Consistent with the highly educated population base in the ACT, all but 10% had finished high school and many held tertiary qualifications. Although one quarter were working full time, over one third remained off work due to pain. Seventy six percent of the entire sample was currently receiving or seeking compensation for injury/pain. The figure for compensation rose to 86% when individuals who had already received a lump sum settlement were included.

The mean duration of pain for the sample was 37.48 months (SD = 39.52 months) and 66% were taking medication for pain and or psychological symptoms. Table 15 illustrates the heterogeneity of pain sites, the nature of the original injury and types of

\(^{10}\) In an attempt to boost recruitment, letters were sent to 75 general practices in the ACT and a summary of the research was placed in the newsletter for the ACT Division of General Practice. Despite follow-up calls, 70 practices did not respond at all and only one doctor expressed interest in referring patients to the study. Three practices indicated that they were too busy and one doctor indicated that he was not interested.
medication used. Lifetime exposure to a traumatic event was reported by 97% of participants and the mean number of different types of prior trauma (e.g., natural disaster, accident, assault etc) was 2.24 (SD = 1.46).

**Table 14. Mean and percentage frequency demographic data for entire sample**

<table>
<thead>
<tr>
<th>Demographic (N = 29)</th>
<th>Mean (SD) age in years</th>
<th>41.38 (12.39)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>non-indigenous Australian</td>
<td></td>
<td>72%</td>
</tr>
<tr>
<td>Asia</td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>Europe</td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>Africa</td>
<td></td>
<td>7%</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>married or de facto relationship</td>
<td>45%</td>
<td></td>
</tr>
<tr>
<td>single</td>
<td></td>
<td>24%</td>
</tr>
<tr>
<td>separated or divorced</td>
<td></td>
<td>21%</td>
</tr>
<tr>
<td>other</td>
<td></td>
<td>7%</td>
</tr>
<tr>
<td>Living arrangements</td>
<td></td>
<td></td>
</tr>
<tr>
<td>with partner</td>
<td></td>
<td>14%</td>
</tr>
<tr>
<td>with partner and children</td>
<td></td>
<td>38%</td>
</tr>
<tr>
<td>with children</td>
<td></td>
<td>14%</td>
</tr>
<tr>
<td>with parents or relatives</td>
<td></td>
<td>21%</td>
</tr>
<tr>
<td>alone</td>
<td></td>
<td>14%</td>
</tr>
<tr>
<td>Highest educational qualification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>postgraduate</td>
<td></td>
<td>17%</td>
</tr>
<tr>
<td>undergraduate</td>
<td></td>
<td>14%</td>
</tr>
<tr>
<td>TAFE</td>
<td></td>
<td>31%</td>
</tr>
<tr>
<td>High School Certificate (Year 12)</td>
<td>28%</td>
<td></td>
</tr>
<tr>
<td>Year 11 or less</td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>Employment status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>working full time</td>
<td></td>
<td>24%</td>
</tr>
<tr>
<td>graduated return to work plan</td>
<td>28%</td>
<td></td>
</tr>
<tr>
<td>(restricted hours or duties)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>not working due to pain</td>
<td></td>
<td>35%</td>
</tr>
<tr>
<td>home duties</td>
<td></td>
<td>7%</td>
</tr>
<tr>
<td>voluntary work</td>
<td></td>
<td>3%</td>
</tr>
<tr>
<td>retired</td>
<td></td>
<td>3%</td>
</tr>
<tr>
<td>Compensation status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>receiving compensation/litigation</td>
<td>76%</td>
<td></td>
</tr>
<tr>
<td>prior lump sump settlement</td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>no compensation</td>
<td></td>
<td>14%</td>
</tr>
</tbody>
</table>
Table 15. Classification of pain sites, sources of injury and medication usage for entire sample

<table>
<thead>
<tr>
<th>Pain Site, Source and Medication</th>
<th>Percentage Frequency (N = 29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IASP pain site coding</td>
<td></td>
</tr>
<tr>
<td>more than three sites</td>
<td>45%</td>
</tr>
<tr>
<td>lower back</td>
<td>10%</td>
</tr>
<tr>
<td>shoulders and upper limbs</td>
<td>10%</td>
</tr>
<tr>
<td>neck</td>
<td>31%</td>
</tr>
<tr>
<td>middle back</td>
<td>3%</td>
</tr>
<tr>
<td>Attributed source of injury/pain</td>
<td></td>
</tr>
<tr>
<td>motor vehicle accidents</td>
<td>72%</td>
</tr>
<tr>
<td>work-related accidents, falls or injuries</td>
<td>21%</td>
</tr>
<tr>
<td>work-related assault</td>
<td>3%</td>
</tr>
<tr>
<td>non-work related fall</td>
<td>3%</td>
</tr>
<tr>
<td>Medication used</td>
<td></td>
</tr>
<tr>
<td>simple analgesics</td>
<td>31%</td>
</tr>
<tr>
<td>anticonvulsants</td>
<td>17%</td>
</tr>
<tr>
<td>non-steroidal anti-inflammatory</td>
<td>21%</td>
</tr>
<tr>
<td>narcotic analgesics</td>
<td>59%</td>
</tr>
<tr>
<td>sedatives</td>
<td>3%</td>
</tr>
<tr>
<td>antidepressants</td>
<td>17%</td>
</tr>
</tbody>
</table>

6.2.2 Design

The impact of PTSD on adjustment to and management of chronic pain was examined in a mixed between-group repeated measures design. The independent variable was the presence or absence of PTSD following a sudden injury or onset of pain. The dependent variables were cognitive, affective and physiological measures of pain-related anxiety, fear of movement/reinjury and a range of indicators of adjustment similar to Study 1.

6.2.3 Self-Report Measures

6.2.3.1 Demographic Background, Injury and Pain Characteristics and Treatment History

Participants completed a self-report questionnaire to gather information about demographic background, injury and pain characteristics and treatment history. The demographic data collected consisted of age, sex, nationality, language, marital status, living arrangements, educational level and current work status. Questions regarding pain characteristics and treatment history were the same as in Study 1 and assessed
cause of injury, diagnosis, body parts affected by pain, pain duration, pain frequency, current, average, minimum and maximum pain, expectation of prognosis, number, type and frequency of medications taken, type of professional groups consulted and number of consultations in the past three months.

6.2.3.2 Depression Anxiety Stress Scales

As discussed above, the DASS shows greater accuracy in measuring depression in chronic pain samples compared to more traditional measures, such as the SDS, due to its exclusion of somatic items that artificially inflate scores (Lovibond & Lovibond, 1995). This measure was thus chosen for inclusion in Study 2 in favour of the SDS, which was used in Study 1.

The DASS is a self-report questionnaire, which consists of 42 symptoms of negative affect, rated on a 4-point severity scale, for the past week. The DASS has three subscales, each containing 14 items, measuring the core symptoms of depression, anxiety and stress, such that there is maximum discrimination between the three subscales, whilst retaining high psychometric standards. Thus, in addition to providing greater accuracy for assessing depression, it also provides a broader measure of affective distress than the SDS. Norms are available for use within Australian chronic pain, clinical and normal populations. The original normative study found all three scales had very good to excellent internal consistency (.91, .84, .90, respectively) and demonstrated concurrent validity with the BDI (Lovibond & Lovibond, 1995). Even better internal consistency was achieved within chronic pain and clinical populations (all >.89) (Brown, Chorpita, Korotitsch, & Barlow, 1997; R. Taylor et al., 2005).

6.2.3.3 Tampa Scale of Kinesiophobia

To assess participants’ general fear of reinjury during physical activity, the revised version of the Tampa Scale for Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990) was administered (TSK v2) (Clark, 1996). The TSK v2 contains 13 items rated on a four-point scale: strongly disagree, somewhat disagree, somewhat agree and strongly agree. Under revision, three items from the original TSK were eliminated as they duplicated content from other items and were presented in the negative. Item and factor analyses further indicated that these four reverse-scored items detracted from the scale’s reliability and factor structure (Clark, 2001, personal communication).
6.2.3.4 Photograph Series of Daily Activities

The Photograph Series of Daily Activities ® (PHODA) (Kugler et al., 1999) is a standardised method of assessing pain-related fear of activities of daily living, such as lifting, bending, walking, reaching etc. Individuals are requested to view 100 photographs, placing each along a fear thermometer, ranging vertically from 0 to 100 (with 11 anchor points). The cardboard fear thermometer is located on a table in front of the individual. The standard test instruction is “Please watch each photograph carefully and try to imagine yourself performing the same movement. Place the photograph on the thermometer according to the extent to which you feel that this movement is harmful to your back”. After completion of the test, each photograph is given a rating based on its thermometer position. The PHODA was thus selected as a more engaging task, perhaps better able to assess pain-related fear-avoidance than a questionnaire, by providing visual prompts of the range of activities an individual may face. It also has the further benefit of referencing specific activities rather than activity in general.

Several modifications of the PHODA were made for the purpose of the current study. First, participants' physiological arousal in response to the movements depicted in the photographs was of interest. To standardise presentation, participants were shown a slide-show of the 100 photographs, presented in random order on a PC monitor using Inquisit 1.33 (Millisecond Software, Seattle, WA). The written instruction “imagine yourself performing this activity or movement” was simultaneously presented on the screen above each photograph. All photographs and instructions were presented in the same location on the screen. Activating imaginal exposure in this way has two benefits. Provision of a visual prompt aids access to similar visual imagery. Second, imaginal exposure arguably controls for artefacts in measurement of physiological reactivity attributable to pain and movement during actual performance.

Second, it was of interest whether fear ratings would be influenced by the frequency with which participants were already performing the movement. Thus, after 5 seconds, a rating scale was presented on the screen underneath the photograph and individuals were asked to indicate how often they performed the activity, using a scale ranging from 1 (never) to 6 (more than once daily). Responses were given verbally and entered by the researcher to minimise movement artifact during physiological recording and to control for possible difficulties with using a mouse or keyboard due to injury/pain.
location. The slideshow is referred to below as PHODA Task 1. Entry of the individual’s response prompted presentation of the next slide. The sum of all responses was divided by 100 (number of items) to provide a mean score of PHODA Frequency of activity ratings.

Participants subsequently completed the standard PHODA Fear-rating (PHODA Task 2), with a slight modification to the instruction. Accounting for the heterogenous nature of pain complaints, participants were instructed to rate harmfulness to their “body” rather than their “back”. To obtain a total score, ratings for all 100 photographs were summed and divided by the maximum possible score (10 000).

6.2.3.5 Other Self-report Questionnaires

Participants also completed the Pain Anxiety Symptoms Scale (PASS) (McCracken et al., 1992), the Pain Disability Index (PDI) (Tait et al., 1990) and the Posttraumatic Diagnostic Scale (Foa et al., 1997). They were asked to complete the latter with respect to their current injury, as in Study 1. These questionnaires are considered reliable and valid self-report measures (see Study 1, Section 5.2.2 Measures).

6.2.3.6 Numerical Rating Scales for Current Pain and Affect

Current pain intensity and affective reactivity was assessed by completion of verbal numerical rating scales (NRS) at predetermined points throughout the laboratory session. The NRS for current pain was anchored at 0 no pain and 10 worst pain imaginable. Such individual ratings have sufficient psychometric strength for use in chronic pain research (Jensen et al., 1999). In addition, participants were also asked to rate their current affect (distress, anxiety/fear, tension, irritability/anger and sadness), using a similar NRS anchored at 0 not at all and 10 extremely .... The five individual scales were then summed and divided by 50 to give a mean affective arousal score. Similar NRS have been used in previous, similar research (Spertus, 2000).

6.2.4 Diagnostic Assessment for Current and Lifetime PTSD

A clinical psychologist (BB) administered the SCID to all participants. The SCID has been standardised on clinical populations and is considered a “gold standard” instrument for diagnostic purposes, in both clinical and research domains. Indeed, it is the most widely used semi-structured interview used in PTSD studies (Newman, Kaloupek, & Keane, 1996). It has demonstrated excellent validity and reliability, good
sensitivity and excellent specificity (Newman et al., 1996; R. L. Spitzer et al., 1990). For this study, the SCID-PTSD was administered to determine the presence of PTSD in relation to (1) the initiating event that triggered pain onset, (2) subsequent episodes of extreme or uncontrolled pain and (3) to establish lifetime diagnosis of PTSD.

The inclusion of a structured clinical interview permitted symptoms that were directly attributable to injury or environmental factors to be screened out, thus facilitating greater diagnostic accuracy of PTSD than a self-report questionnaire (Mellman, David, Bustamante, Fins, & Esposito, 2001). To satisfy criteria for intrusive re-experiencing individuals needed to describe relatively unmodified and unelaborated memories intruding upon awareness and not just voluntary rumination about the injurious event and its sequelae. The discomfort of chronic pain often leads to sleep disturbance so to meet this symptom criterion individuals needed to report PTSD-specific disturbance, such as delayed onset due to alertness or waking due to dreams or autonomic dysregulation (e.g., night sweats). Concentration problems also needed to extend beyond disruption due simply to intense pain. In this way, symptoms were included if a psychological and pain-related cause were established but excluded if they were best accounted for by injury or pain.

6.2.5 Psychophysiological Measures

Data was recorded at baseline and during administration of the SCID to investigate differences between participants with and without PTSD in terms of autonomic arousal. Physiological responses to presentation of the PHODA were also taken to determine whether self-reported fear was associated with autonomic activity and whether this differed for participants with and without PTSD. All physiological data were organised and recorded on a PC using Labview 6.1 (National Instruments, Austin, TX). Tags were inserted to correspond with relevant aspects of the stimulus presentation or interview procedure to aid later offline analysis. No visual or verbal cues or feedback were given to participants regarding physiological reactivity during the experiment.

Measures of systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were obtained from a Finipres 2300 Blood Pressure Monitor (Ohmeda, Madison, WI), sampling at a rate of 1Hz. A tape-measure was used to ensure appropriate sizing of the Finipres cuff, which was placed on the middle finger of the non-dominant hand, except for one participant who had a history of lymphectomy, in
which case her dominant hand was used. In all cases, the limb used was not a significant pain site. The cuff was kept at heart height during recording to facilitate accurate blood-pressure readings (Imhol, Wieling, van Montfrans, & Wesseling, 1998), with the participant resting their arm either on a pillow or directly against their body. The Finapres device was switched off in between recording phases. Where readings were unexpected or thought to be unusual during testing, the placement of equipment was adjusted (by tightening or loosening it, or moving it slightly up or down the finger) and or readings confirmed by use of a brachial sphygmomanometry, using a Dinamap Plus Vital Signs Monitor (Critikon Inc., Tampa FL).

Respiration rate (RR) was monitored with a stretch-sensitive belt (Pro-Tech, Mukilteo, WA), worn around the rib cage. Sampling occurred at a rate of 10Hz. Data was converted to breathing frequency traces using Chart 4 (ADI Instruments, Colorado Springs, CO).

Skin conductance (SC) was sampled at 10Hz using a μmhos 2701 (Bioderm, Morro Bay, CA). Silver-silver/chloride electrodes, 8mm in diameter were attached to the palmar surface of the index and third finger of the non-dominant hand. Each was filled with Medsafe TD-246 (Med Associates Inc., St. Albans, VT), an electrode paste specifically designed for skin conductance recordings. To standardise the procedure for skin conductance, all participants washed their hands with soap and water at the commencement of laboratory sessions (Venables & Christie, 1980). The laboratory was also climate-controlled to limit the impact of ambient temperature and humidity on recordings of skin conductance (Venables & Christie, 1980). Average (± SE) conditions were 23.2 (± 0.3) °C and 38.0 (± 0.9)% humidity.

6.2.6 Procedure

Staff from the CIMC, psychologists and physiotherapist advised potential participants of the existence of the study and provided information and consent forms (see Appendix E) to those who expressed interest in participating. It was clearly explained that participation was voluntary and that no treatment would be provided, reinforcing the statements to this effect in the written information. Participants were also made aware that treating practitioners would not be given access to individual results nor feedback unless the participant granted express consent. Signed consent forms were forwarded to the researcher, who then contacted the participant by telephone
and made an appointment for the first session. Participants were also sent a packet of questionnaires (Demographic Information, Pain Characteristics and Treatment History, DASS, PDI and PDS) to complete and bring to the first session (see Appendix F). Participants were asked to refrain from consuming alcohol, caffeine or analgesics within four hours of attending as these are likely to have an impact on physiological measures and current pain levels. Compliance with this request was checked verbally at the start of the session.

On arrival at the laboratory, participants were given a brief overview of the procedure for the session. Participants washed their hands then psychophysiological recording equipment was attached with the participant seated in an upright position. After a 5-minute rest/adaptation period, a 5-minute baseline was recorded for all psychophysiological data, during which the participant was instructed to sit quietly. Participants then completed an NRS for current pain and affect and completed the PASS and TSK. During this time, the questionnaires completed at home were screened for missing data and participants were asked to fill-in any missing items.

Participants then completed the PHODA Task 1, while psychophysiological data were continuously recorded, followed by the PHODA Task 2. The SCID was administered last, to ensure that fear-related measures were not influenced by the forced recall of the circumstances of injury. Participants were not provided with any diagnostic feedback during the experiment. Psychophysiological data were again continuously recorded throughout the interview. The opportunity of a 5-minute break was offered to participants between each task, allowing them to move around and adopt a comfortable posture. At the end of each question a manually operated switch was used to insert a marker within the physiological data for use in later off-line analysis.

As participants returned for further testing on future dates, they were not debriefed until the conclusion of the final session. At that point, participants were paid a $10 per hour honorarium to reimburse them for their travel costs and time.

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11 Patients were advised to continue taking any other prescribed medication (e.g., antidepressants).

12 The results of this testing are reported in Study 3 and 4.
6.2.7 Ethics Approval

The Human Research Ethics Committee of the Australian National University approved the protocol for this study (see Appendix G).

6.2.8 Data Analysis

Prior to data analysis, data was screened for accuracy of data entry, missing values and fit between their distributions and the assumptions of univariate and multivariate analysis. Separate examinations were made within the PTSD and No PTSD groups for univariate outliers, which were deemed where \( z > 3.29 \) (\( p > .001 \), two-tailed test) (Tabachnick & Fidell, 2001). Three univariate outliers were detected within the No PTSD group, one each on pain duration, SCID HR and SCID SC. Fourteen univariate outliers were identified amongst physiological data in the PTSD group: baseline SBP (\( n = 1 \)), baseline HR (\( n = 1 \)), baseline SC (\( n = 2 \)), PHODA SBP (\( n = 1 \)), PHODA DBP (\( n = 2 \)), PHODA HR (\( n = 1 \)), PHODA SC (\( n = 1 \)), SCID DBP (\( n = 4 \)) and SCID SC (\( n = 1 \)). To reduce the impact of these scores whilst conserving data, all outliers were recoded to one unit above the nearest respondent’s score (Tabachnick & Fidell, 2001). No multivariate outliers were identified, using Mahalanobis distance with \( p < .001 \).

Due to equipment failure, one participant from the PTSD group was found to have missing data for the PHODA frequency rating, one participant from the PTSD group was missing all physiological data for the SCID phase and one participant from the No PTSD group was missing all physiological data for the PHODA phase. The breathing trace for three participants, 1 from the PTSD group and 2 from the No PTSD group, was unreadable. Each of these participants was thus excluded from relevant analyses.

Significant negative skew (\( z = 3.59, p < .001 \)) was evident in the distribution of maximum pain ratings. In light of the nature of the scale this is an expected result so raw scores were retained for analysis. To facilitate data reduction and overcome problems with skew and or kurtosis on several distributions, a Composite Affective score was computed from the five individual NRS scales. That is, item ratings for distress, anxiety/fear, tension, irritability/anger and sadness were summed to form the Composite Affective NRS variable.

Independent sample t-tests and chi square analyses were conducted to determine whether PTSD and No PTSD groups differed according to PTSD severity and on the
primary demographic variables of age, gender, educational background, number of prior traumatic exposures and lifetime PTSD diagnoses. Calculation of $t$ was based on the assumption of equal variances unless otherwise specified.

The reliability of each published scale was calculated using Cronbach’s alpha consistency coefficient (the standardised item alpha is reported). Subscales as well as total scores were evaluated for internal consistency in this way.

Multiple analyses of variance (MANOVA) were then conducted to examine differences between PTSD and No PTSD groups on measures of (1) outcome (pain severity, health care utilisation, pain-related disability and distress); (2) pain-related anxiety; and (3) fear of movement/reinjury. Given the survey design, a sequential adjustment was chosen to deal with nonorthogonality in MANOVAs (Tabachnick & Fidell, 2001).

To examine physiological reactivity during presentation of the PHODA Task 1, the mean values obtained for each item were averaged to give a total score. The mean value for physiological data obtained during recall of the traumatic injury during administration of the SCID were also calculated. Physiological measures were then analysed separately using a two factor mixed design ANOVA, with follow-up univariate analyses. Thus, SBP, DBP, HR, SC and RR each served as dependent variables, with the first factor, group (PTSD or No PTSD), as an independent variable and repeated measures on the second factor, time (Baseline, PHODA, SCID). Both Mauchley’s test of sphericity and low Epsilon values ($<.75$) (Hinton, Brownlow, McMurray, & Cozens, 2004) were used to check for violation of the assumption of sphericity. In the face of violation, Greenhouse-Geisser corrections were used to obtain a corrected $F$ value (Hinton et al., 2004; Tabachnick & Fidell, 2001), while in all other cases Wilk’s lambda was used.

Bivariate correlations were also calculated to highlight relationships between variables within each patient group. Separate hierarchical regression analyses were finally performed to determine the unique contribution each block of independent variables made to predicting these.
In all instances, the alpha level was set at .05 and Bonferroni corrections were made to maintain this level of Type I error in the context of multiple comparisons, unless otherwise specified. SPSS for Windows (Version 10) was the software used for all analyses. SPSS calculates alpha to three decimal places, therefore all “.000” output values are reported here as \( p < .001 \).

6.3 Results

6.3.1 Diagnosis, Classification and Severity of PTSD

Based on their responses during the SCID-PTSD module, participants were classified according to whether or not they met full criteria for a current diagnosis of PTSD, in relation to their current injury and pain. On this basis, 14 participants were diagnosed with PTSD (PTSD group) while 15 participants did not satisfy criteria for a current diagnosis of PTSD (No PTSD group).

Given questions about causality, the presence of lifetime PTSD was also assessed, based on retrospective self-report during the SCID-PTSD module, in relation to prior exposure to traumatic events. Table 16 illustrates the prevalence of lifetime PTSD and mean number of prior traumatic exposures. Five participants from the PTSD group met criteria for a lifetime diagnosis of PTSD pre-dating injury onset (i.e. related to a prior exposure to trauma), while nine denied any previous history of PTSD. Seven participants from the No PTSD group met criteria for a diagnosis of PTSD, two of whom reported lifetime PTSD as a direct result of their current injury but which had since fully remitted and five of whom had lifetime PTSD arising from exposure to trauma prior to their current injury. There was no significant difference between the frequency of lifetime PTSD between groups \( \chi^2 (1) = .36, p = .55 \). Similarly, as shown in Table 16, the mean number of prior exposures to traumatic events was equivalent between groups \( t(27) = - 0.41, p = .69, \text{power} = .11 \) and there were no differences between groups regarding the nature of prior traumatic events (all \( p > .05 \)). None of the participants had been exposed to military combat, imprisonment or torture.

Although lifetime PTSD was equivalent across groups, this variable still had the potential to confound subsequent data analysis. Therefore, to determine whether or not it should be used as a covariate in subsequent analyses, a series of univariate and multivariate ANOVAs were conducted using lifetime PTSD (which included current and resolved PTSD) as the independent variable. Due to the number of analyses, the \( p \)
Table 16. Prevalence of Lifetime PTSD and exposure to prior trauma and mean current Posttraumatic Stress Diagnostic Scale (PDS) scores for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD (n = 14)</th>
<th>No PTSD (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifetime PTSD</td>
<td>36%</td>
<td>53%</td>
</tr>
<tr>
<td>Mean no. of prior traumatic exposures⁹</td>
<td>2.36 (1.50)</td>
<td>2.13 (1.46)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>% previously exposed to</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>accident, fire or explosion</td>
<td>50%</td>
<td>40%</td>
</tr>
<tr>
<td>natural disaster</td>
<td>43%</td>
<td>47%</td>
</tr>
<tr>
<td>phys. assault, known perpetrator</td>
<td>29%</td>
<td>27%</td>
</tr>
<tr>
<td>phys. assault, unknown perpetrator</td>
<td>29%</td>
<td>13%</td>
</tr>
<tr>
<td>sex assault, known perpetrator</td>
<td>0%</td>
<td>7%</td>
</tr>
<tr>
<td>sex assault, unknown perpetrator</td>
<td>7%</td>
<td>0%</td>
</tr>
<tr>
<td>sexual contact (&lt;18yrs) with person &gt;5yrs older</td>
<td>0%</td>
<td>13%</td>
</tr>
<tr>
<td>life threatening illness</td>
<td>43%</td>
<td>27%</td>
</tr>
<tr>
<td>other trauma</td>
<td>21%</td>
<td>13%</td>
</tr>
</tbody>
</table>

**PDS Mean scores⁹**

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>No PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>PDS-B (Re-experiencing)</td>
<td>7.11 (4.07)</td>
<td>1.93 (1.58)</td>
</tr>
<tr>
<td>PDS-C (Avoidance/Numbing)</td>
<td>9.71 (4.08)</td>
<td>4.13 (3.38)</td>
</tr>
<tr>
<td>PDS-D (Hyperarousal)</td>
<td>10.43 (3.50)</td>
<td>4.67 (2.82)</td>
</tr>
<tr>
<td>PDS-Total</td>
<td>27.25 (9.74)</td>
<td>10.73 (6.86)</td>
</tr>
</tbody>
</table>

⁹ SD is presented in parentheses.

value was conservatively set at .01. Not surprisingly, those with Lifetime PTSD reported significantly higher PDS symptom severity (M = 21.36, SD = 12.20) than their never-traumatised counterparts (M = 11.75, SD = 7.13), although this large effect only represented a non-significant trend, after correction for Type I error (F (1, 27) = 4.33, p = .05, d = .96). Results otherwise indicated that there was no significant effect of lifetime PTSD on PSI, baseline pain, Composite Affective NRS, PDI total, DASS total, PASS total, TSK, PHODA Fear and Frequency of Activity ratings (even without adjustment for Type I error, as all p > .05), indicating that it was unnecessary to include lifetime PTSD as a covariate in subsequent analyses.

The total and three cluster severity scores derived from the PDS were evaluated for internal consistency. The full scale and re-experiencing subscale demonstrated excellent reliability (Cronbach’s α = .93 and .90, respectively), while the avoidance/numbing and hyperarousal subscales exhibited high reliability (Cronbach’s α = .82 and .80, respectively). Table 16 presents PDS total and subscale scores for the
PTSD and No PTSD group. Supporting the diagnostic classification process, independent t-tests indicated that the PTSD group scored significantly higher than the No PTSD group on re-experiencing symptoms \((t(16) = 4.46, p < .001, d = 1.68)\), avoidance and numbing symptoms \((t(27) = 4.02, p < .001, d = 1.49)\), hyperarousal \((t(27) = 4.90, p < .001, d = 1.81)\) and PDS total \((t(27) = 5.31, p < .001, d = 1.96)\). All of these effect sizes were large.

Although diagnosis of PTSD in this study was based only on the SCID, diagnostic agreement between the SCID and PDS was calculated to aid comparison of the current results with those obtained in Study 1. There was 79.5% agreement between the two measures, with a kappa of .59. The sensitivity of the PDS (the ability to correctly identify individuals diagnosed with PTSD using the SCID) was 85.7% and the specificity (the ability to correctly identify individuals not diagnosed with PTSD using the SCID) was 73.3%.

### 6.3.2 Demographic Analyses

Descriptive statistics for the demographic variables for the PTSD and No PTSD groups are presented in Table 17. There were significantly more females in the PTSD group than the No PTSD group \((\chi^2 (1) = 4.24, p = .04)\). There were no significant differences between the groups in terms of age \((t(27) = .19, p = .85)\) or educational level \((\chi^2 (4) = 1.61, p = .80)\).

<table>
<thead>
<tr>
<th>Demographic</th>
<th>PTSD</th>
<th>No PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>((n = 14))</td>
<td>((n = 15))</td>
</tr>
<tr>
<td>% Females</td>
<td>43%</td>
<td>80%</td>
</tr>
<tr>
<td>Mean age in years (^a)</td>
<td>40.93 (13.50)</td>
<td>41.80 (11.71)</td>
</tr>
<tr>
<td>% reaching each educational level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>less than Year 12</td>
<td>7%</td>
<td>13%</td>
</tr>
<tr>
<td>Year 12</td>
<td>29%</td>
<td>28%</td>
</tr>
<tr>
<td>TAFE/trade qualification</td>
<td>36%</td>
<td>27%</td>
</tr>
<tr>
<td>undergraduate qualification</td>
<td>7%</td>
<td>20%</td>
</tr>
<tr>
<td>postgraduate qualification</td>
<td>21%</td>
<td>13%</td>
</tr>
</tbody>
</table>

\(^a\): Mean age in years was calculated by the mid-point of the range of age at each educational level.
To determine whether or not gender should be used as a covariate in subsequent analyses, a series of univariate and multivariate ANOVAs were conducted using gender as the independent variable and the following measures as dependent variables: PSI, baseline pain, Composite Affective NRS, DASS total, PDS total, PASS total, TSK, PHODA Fear and Frequency of Activity ratings, PDI total and physiological measures at baseline, during the PHODA and the SCID. Due to the number of analyses, the p value was conservatively set at .01. Results indicated that there was no significant effect of gender on any of these variables (even without adjustment for Type I error, as all p > .05), indicating that it was unnecessary to include gender as a covariate in subsequent analyses.

6.3.3 Analyses for Pain Severity, Health Care Utilisation, Disability and Distress

Descriptive statistics for pain severity, health care utilisation, disability and distress variables for the PTSD and No PTSD group are presented in Table 18. The pain severity index was calculated in the same way as for Study 1 and had excellent internal consistency (Cronbach’s α = .92). Compared to the No PTSD group, the PTSD group rated their pain at baseline and in the month preceding assessment as more intense, however, this difference was only a non-significant trend (baseline pain: t (27) = 1.80, p = .08; PSI: t(27) = -1.54, p = .07). As the effect sizes were large (d = .67 and .57, respectively), the lack of significance is likely attributable to insufficient power (.54 and .46, respectively). Similarly, there were no significant differences in the duration of pain (t(27) = .52, p = .61) or the reported number of painful body parts (t(27) = .70, p = .25). However, the effect sizes were small (d = .19 and .26, respectively) and power was low (.13 and .18, respectively). The PTSD group were more likely to report a higher frequency of pain symptoms than the No PTSD group (χ² (5) = 7.22, p < .01). That is, the PTSD group were more likely than the No PTSD group to describe their pain as constant (93% vs 47%, respectively) than to describe having periods that were pain-free (7% vs 53%). Bivariate correlations revealed that baseline pain was significantly moderately correlated with PDS–C severity (r (29) = .43, p = .02) but not with PDS-B or PDS-D severity (p > .05). Pain ratings estimated over a longer duration (i.e. PSI) were significantly and moderately correlated with both PDS-C (r (29) = .46, p = .01) and PDS-D (r (29) = .40, p = .03), but not PDS-B severity (p > .05).

Contrary to findings in Study 1, a MANOVA indicated no significant effect of PTSD with respect to health care utilisation variables (number of different providers
consulted and number of medications taken) (Wilks' lambda = .08, F (2, 26) = 1.17, p = .17), however, power was low (.23). The two groups also reported a similar frequency

Table 18. Mean and percentage frequency of pain severity, health care utilisation, disability and distress variables for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>No PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 14)</td>
<td>(n = 15)</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Pain Severity variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current pain intensity</td>
<td>5.00</td>
<td>2.55</td>
</tr>
<tr>
<td>Average pain intensity</td>
<td>5.68</td>
<td>2.16</td>
</tr>
<tr>
<td>Minimum pain intensity</td>
<td>3.64</td>
<td>1.96</td>
</tr>
<tr>
<td>Maximum pain intensity</td>
<td>8.39</td>
<td>2.24</td>
</tr>
<tr>
<td>Pain Severity Index</td>
<td>5.68</td>
<td>1.86</td>
</tr>
<tr>
<td>Baseline Pain</td>
<td>4.61</td>
<td>2.47</td>
</tr>
<tr>
<td>Duration of pain (months)</td>
<td>32.00</td>
<td>31.10</td>
</tr>
<tr>
<td>Number of painful body parts</td>
<td>6</td>
<td>3.21</td>
</tr>
<tr>
<td><strong>Health care utilisation variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of medications</td>
<td>1.71</td>
<td>1.44</td>
</tr>
<tr>
<td>Number of professions consulted</td>
<td>6.57</td>
<td>3.37</td>
</tr>
<tr>
<td>% frequency of consultations in past 3 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>less than once per week</td>
<td>36%</td>
<td>60%</td>
</tr>
<tr>
<td>once or twice per week</td>
<td>36%</td>
<td>27%</td>
</tr>
<tr>
<td>two or three times per week</td>
<td>21%</td>
<td>13%</td>
</tr>
<tr>
<td>four or five times per week</td>
<td>7%</td>
<td>0%</td>
</tr>
<tr>
<td><strong>Disability variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain Disability Index score</td>
<td>38.32</td>
<td>15.22</td>
</tr>
<tr>
<td>% Currently working</td>
<td>57%</td>
<td>47%</td>
</tr>
<tr>
<td>% Perceived prognosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>get worse</td>
<td>21%</td>
<td>13%</td>
</tr>
<tr>
<td>no change</td>
<td>29%</td>
<td>33%</td>
</tr>
<tr>
<td>improve</td>
<td>43%</td>
<td>27%</td>
</tr>
<tr>
<td>totally recover</td>
<td>7%</td>
<td>27%</td>
</tr>
<tr>
<td>% Received or receiving compensation</td>
<td>100%</td>
<td>73%</td>
</tr>
<tr>
<td><strong>Distress variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASS – Depression score</td>
<td>17.79</td>
<td>10.69</td>
</tr>
<tr>
<td>DASS – Anxiety score</td>
<td>14.07</td>
<td>9.93</td>
</tr>
<tr>
<td>DASS – Stress score</td>
<td>20.07</td>
<td>9.42</td>
</tr>
<tr>
<td>DASS Total score</td>
<td>51.93</td>
<td>27.57</td>
</tr>
<tr>
<td>Composite Affective NRS</td>
<td>12.75</td>
<td>8.94</td>
</tr>
</tbody>
</table>

Note. DASS = Depression Anxiety Stress Scale; NRS = numerical rating scales.
of treatment ($\chi^2 (3) = 2.42, p = .49$) and likelihood of using some medication ($\chi^2 (1) = .42, p = .57$) over the past three months. The PTSD group were significantly more likely than the No PTSD group to be using antidepressant medication (36% vs 0%; ($\chi^2 (1) = 6.47, p = .01$), however, no significant differences were revealed in terms of pain-related medications, such as simple and narcotic analgesics, anticonvulsants, anti-inflammatories or muscle relaxants (all $p > .05$).

Turning to measures of disability, the PDI demonstrated excellent internal consistency (Cronbach’s $\alpha = .94$). The PTSD group perceived a significantly higher level of disability than the No PTSD group, according to PDI scores ($t (27) = 2.19, p = .02, d = .82$). Unlike the findings of Study 1, however, the two groups did not significantly differ in their expectation of a positive or negative prognosis ($\chi^2 (1) = .03, p = .86$) nor their likelihood of being currently at work ($\chi^2 (1) = .32, p = .57$). Both PDS-C and PDS-D severity were moderately correlated with PDI ($r (29) = .62, p < .001$ and $r (29) = .60, p = .001$), whereas there was no significant relationship between PDS-B and PDI ($p > .05$).

The PTSD group were more likely than the No PTSD group to have received, be receiving or seeking compensation ($\chi^2 (1) = 4.33, p = .04$). Given prior evidence associating compensation with a range of factors (Turk & Okifuji, 1996), a series of univariate and multivariate ANOVAs was conducted, using compensation status as the independent variable. Due to the number of analyses, the $p$ value was conservatively set at .01. These analyses indicated no significant effect of compensation status on PSI, baseline pain, Composite Affective NRS, DASS total, PDS total, PASS total, TSK, PHODA Fear or physiological measures at baseline or during administration of the PHODA or the SCID (all $p > .01$). A significant effect of compensation status was found, however, on PDI total ($F (1, 27) = 7.80, p < .01$) and PHODA Frequency of Activity total ($F (1, 26) = 13.52, p = .001$). That is, the four participants who were not receiving or seeking compensation reported less perceived disability (PDI total: $M = 10.25, SD = 8.18$) and more frequent engagement in specific activities (Frequency of Activity: $M = 2.88, SD = .38$) than the participants who were receiving or seeking compensation (PDI total: $n = 25, M = 34.56, SD = 16.90$; Frequency of Activity: $n = 24, M = 2.12, SD = .38$). Although the potential confound is acknowledged, due to the grossly unequal cell sizes and the 100% compensated rate for the PTSD group, compensation status was not further utilised as a covariate.
The DASS total, Depression, Anxiety and Stress scale scores showed excellent internal consistency (Cronbach’s α = .97, .95, .93 and .93, respectively). The PTSD group endorsed higher levels of general distress than the No PTSD group and an independent sample t test indicated that the difference represented a significant and large effect (DASS Total: (t(22)13 = 2.93, p < .01, d = 1.09). A MANOVA conducted on the Depression, Anxiety and Stress scales indicated a significant effect of PTSD (Wilks’ lambda = .71, F (2, 25) = 3.46, p = .03, ηp² = .29). Univariate analyses revealed that the PTSD group reported higher levels of Depression (F (1, 27) = 11.09, p < .01, ηp² = .29), Anxiety (F (1, 27) = 6.12, p = .01, ηp² = .19) and Stress (F (1, 27) = 4.94, p = .02, ηp² = .16) than the No PTSD group. The Composite Affective NRS indicated that the PTSD group also had a significantly higher baseline level of affective distress within the experimental session than the No PTSD group (t (18.25)14 = 3.10, p < .01, d = 1.17). Further, the Composite Affective NRS was moderately correlated with the severity of PTSD (PDS-B: r (29) = .50, p < .01; PDS-C: r (29) = .63, p < .001; and PDS-D: r (29) = .60, p < .001).

Bivariate correlations between the DASS and PDS subscales are presented in Table 19. These highlighted that DASS Depression had a strong significant relationship with PDS-B and PDS-C and a moderate significant relationship with PDS-D. Significant moderate associations also existed between DASS Anxiety and all three PDS subscales and between DASS Stress and all three PDS subscales.

<table>
<thead>
<tr>
<th></th>
<th>PDS-B</th>
<th>PDS-C</th>
<th>PDS-D</th>
</tr>
</thead>
<tbody>
<tr>
<td>DASS Depression</td>
<td>.77***</td>
<td>.79***</td>
<td>.57***</td>
</tr>
<tr>
<td>DASS Anxiety</td>
<td>.59***</td>
<td>.56**</td>
<td>.47**</td>
</tr>
<tr>
<td>DASS Stress</td>
<td>.53**</td>
<td>.63***</td>
<td>.60***</td>
</tr>
</tbody>
</table>

**p < .01, ***p < .001.

13 Degrees of freedom were reduced in this case as calculation of t did not assume equal variance (Levene’s test was significant).

14 As above.
6.3.4 Analyses for Pain-related Anxiety and Fear of Movement/Reinjury

Reliability analyses indicated that the PASS had high to excellent reliability for the total score (Cronbach’s $\alpha = .95$), the Cognitive anxiety symptoms subscale (Cronbach’s $\alpha = .91$), the Fearful Appraisal subscale (Cronbach’s $\alpha = .83$), the Physiological anxiety symptoms subscale (Cronbach’s $\alpha = .90$) and the Escape and avoidance behaviours subscale (Cronbach’s $\alpha = .78$).

Table 20 presents data for the PASS for both groups, illustrating that the PTSD group obtained higher scores on all four subscales and the total scale. An independent sample $t$ test indicated that the difference in PASS total scores was significant ($t(27) = .31, p < .01$). A MANOVA conducted on the four subscales of the PASS was also significant (Wilks’ lambda = .58, $F(4, 24) = 4.31, p < .01, \eta_p^2 = .42$). Univariate analyses revealed that the PTSD group reported significantly higher levels of Cognitive anxiety symptoms ($F(1, 27) = 14.18, p < .001, \eta_p^2 = .12$) and Physiological anxiety symptoms ($F(1, 27) = 9.80, p < .01, \eta_p^2 = .27$) than the No PTSD group. There was a non-significant trend for greater Escape and avoidance behaviours amongst the PTSD group ($F(1, 27) = 3.81, p = .06, \eta_p^2 = .34$), however, this lack of significance was not due to low power (.97). By contrast, the lack of significant difference between groups in terms of the level of Fearful Appraisals ($F(1, 27) = 2.45, p = .13$) could be explained by lack of power (.33).

Table 20. Mean Pain Anxiety Symptom Scale (PASS), Tampa Scale of Kinesiophobia (TSK) and Photograph Series of Daily Activities (PHODA) scores for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD ($n = 14$)</th>
<th>No PTSD ($n = 15$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>PASS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fearful appraisals</td>
<td>18.64</td>
<td>10.65</td>
</tr>
<tr>
<td>Physiological anxiety symptoms</td>
<td>22.86</td>
<td>12.64</td>
</tr>
<tr>
<td>Escape and avoidance behaviours</td>
<td>22.46</td>
<td>8.65</td>
</tr>
<tr>
<td>Total</td>
<td>99.57</td>
<td>35.76</td>
</tr>
<tr>
<td>TSK</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear-rating</td>
<td>.52</td>
<td>.21</td>
</tr>
<tr>
<td>Frequency of activity rating</td>
<td>1.98$^1$</td>
<td>.36</td>
</tr>
</tbody>
</table>

$^1n = 13$, due to missing data for one participant.
Turning to measures for fear of movement/reinjury, the TSK demonstrated good internal consistency (Cronbach’s $\alpha = .72$). As seen in Table 20, the PTSD group demonstrated significantly greater general fear of movement/reinjury than the No PTSD group (TSK: $t(27) = 2.25, p = .02, d = .84$). The PTSD group’s mean rating of harmfulness of specific activities of daily living (PHODA Fear) was also higher than the No PTSD group, a significant and medium difference according to an independent-sample $t$ test ($t(27) = 1.90, p = .04, d = .70$). Finally, the PTSD group reported engaging in the illustrated activities significantly less often than the No PTSD group (PHODA Frequency of activity: $t(26) = -3.09, p < .01, d = 1.17$).

The results of correlational analyses, shown in Table 21, exposed moderate correlations between the PDS-B and measures of pain-related fear and anxiety, the strongest relationship being with the TSK. PDS-C demonstrated weak to moderate correlations with each of these scales. Interestingly, despite the common element of avoidance, the PASS Escape and avoidance subscale had the weakest relationship with the PDS-C. The PDS-D was most strongly related to PASS Physiological and then Cognitive anxiety subscales, not surprising, given both scales’ emphasis on physiological arousal or its cognitive effects (e.g., dizziness, poor concentration). PDS-D also demonstrated moderate correlations with fear of movement/reinjury but was not significantly correlated with Fearful Appraisals of pain or Escape and Avoidance behaviours. All three PDS subscales showed moderate negative correlations with the frequency of specific activities, such that higher PTSD severity was associated with lower engagement in activity.

In addition, it was hypothesised that measures of pain-related anxiety and fear of movement/reinjury should be correlated with each other. Further, it was expected that fear of movement/reinjury would be associated with the frequency with which participants engaged in the movements and with the degree of arousal experienced during exposure to the movements.

As predicted, the TSK, representing a general fear of movement/reinjury, was significantly and positively correlated with pain-related anxiety (all PASS subscales) and fear of movement/reinjury (PHODA Fear rating) (all $p < .05$). Although the TSK was significantly and negatively correlated with self-reported frequency of engaging in
specific movements (PHODA Frequency of activity rating: $r(28) = .53, p < .01$), it was not significantly associated with participants’ overall perceived disability (PDI: $r(29) = .27, p = .16$). By contrast, the PHODA Fear rating showed significant positive correlations with the PDI ($r(29) = .63, p < .001$), in addition to the PASS ($r(29) = .56, p = .001$). On the other hand, unlike the general measure provided by TSK, these ratings of the harmfulness of specific movements were not significantly correlated with the self-reported frequency of performing them ($r(28) = -.21, p = .28$).

### 6.3.5 Additional Analyses of Psychophysiological Data

Descriptive statistics for measures of systolic blood pressure, diastolic blood pressure, heart rate, skin conductance and respiration rate are displayed in Table 22. Bivariate correlations revealed that there were no significant associations between current or longer-term measures of pain (baseline pain NRS or PSI) or distress (Composite Affective NRS or DASS-Total) and any of the physiological measures (all $p > .01$), indicating that it was not necessary to include these variables as covariates in subsequent analyses. Separate mixed design ANOVAs were thus used to evaluate physiological data between groups and over the three time periods (baseline, PHODA Task 1, SCID).

Table 22 illustrates that the No PTSD group curiously recorded a higher mean resting systolic blood pressure than the PTSD group. Although both groups showed
similar elevations during presentation of the PHODA, the PTSD group also appeared to
demonstrate a greater increase in systolic blood pressure than the No PTSD group from
baseline in response to the SCID. While the results of ANOVA indicated a significant
and medium size main effect of time ($F(2, 50) = 4.45, p = .02, \eta_p^2 = .15$), neither the
main effect of group nor the interaction between group and time were significant
(group: $F(1, 25) = .01, p = .93$; group x time: $F(2, 50) = .13, p = .88$). Notably, power
was very low for detecting the latter effects (.05 and .07, respectively). According to
pairwise comparisons, the significant linear trend over time ($F (1, 25) = 10.99, p < .01,$
$\eta_p^2 = .31$) was due to a significant increase in systolic blood pressure from baseline
during the SCID ($D = -8.74, p < .01$). Differences in systolic blood pressure between
baseline and the PHODA and the PHODA and the SCID were not significant ($p > .05$).
Notably, there were no significant correlations between PTSD severity and systolic
blood pressure in any time period (all $p > .05$).

Table 22. Mean psychophysiological data for PTSD and No PTSD groups in each
task

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>PHODA</th>
<th>SCID</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
</tr>
<tr>
<td>PTSD (n = 14)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>114.43</td>
<td>16.40</td>
<td>119.31</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>67.41</td>
<td>8.75</td>
<td>69.20</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>75.26</td>
<td>6.37</td>
<td>74.50</td>
</tr>
<tr>
<td>SC (µmho)</td>
<td>3.95</td>
<td>2.45</td>
<td>4.94</td>
</tr>
<tr>
<td>RR (breaths/min)</td>
<td>18.62</td>
<td>6.58</td>
<td>18.97</td>
</tr>
<tr>
<td>No PTSD (n=15)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>116.52</td>
<td>15.68</td>
<td>119.73</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>69.07</td>
<td>13.24</td>
<td>70.61</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>79.47</td>
<td>7.46</td>
<td>79.86</td>
</tr>
<tr>
<td>SC (µmho)</td>
<td>3.19</td>
<td>1.59</td>
<td>4.43</td>
</tr>
<tr>
<td>RR (breaths/min)</td>
<td>19.87</td>
<td>5.47</td>
<td>19.35</td>
</tr>
</tbody>
</table>

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate;
SC = skin conductance; RR = respiration rate.
Similar to results for systolic blood pressure, the No PTSD group recorded slightly higher resting diastolic blood pressure compared to the PTSD group. For both groups, diastolic blood pressure increased above the baseline during completion of the PHODA and during recall of the traumatic injury as part of the SCID, with the PTSD group appearing to have a greater increase from baseline in response to the SCID than the No PTSD group. Indeed, the main effect of time was significant \( (F(2, 20) = 13.02, p < .001, \eta^2_p = .34), \) reflecting a significant linear trend over the three time periods \( (F(1, 25) = 25.60, p < .001, \eta^2_p = .51) \). There was, however, no significant interaction between time and group \( (F(2, 20) = .79, p = .46) \) nor a significant main effect of group \( (F(1, 25) = .01, p = .94) \). That is, overall differences in diastolic blood pressure between PTSD and No PTSD individuals in the three time periods were not significant. Again, power was very low for detecting the latter two effects (.18 and .15, respectively). Pairwise comparisons indicated that significant increases in diastolic blood pressure occurred from baseline to the SCID \( (D = -8.45, p < .001) \) and diastolic blood pressure was significantly greater for both groups during the SCID than the PHODA \( (D = -6.78, p < .01) \). In addition, no significant relationship was found between diastolic blood pressure and PTSD severity in any time period (all \( p > .05 \)).

Heart rate was greatest during the SCID and the No PTSD group appeared to have a greater heart rate than the PTSD group in all three time periods. Due to violation of the assumption of sphericity, a Greenhouse-Geisser correction was used for the ANOVA. This revealed a significant main effect of time \( (F(1.31, 32.71) = 11.35, p = .001, \eta^2_p = .31) \), however, again there was no significant main effect of group \( (F(1, 25) = 1.76, p = .20) \) nor a significant interaction effect \( (F(1.31, 32.71) = .77, p = .47) \). Power to detect the main effect for group and the interaction was low (.25 and .17, respectively). A significant linear trend was present for the main effect of time \( (F(1, 25) = 11.15, p < .01, \eta^2_p = .31) \), however, there was also evidence of an underlying quadratic trend \( (F(1, 25) = 11.91, p < .01, \eta^2_p = .32) \). Indeed, pairwise comparisons showed that while heart rate was similar at rest and during the PHODA \( (p = 1.00) \), during administration of the SCID, heart rate was significantly elevated above levels recorded during both baseline \( (D = -6.03, p < .01) \) and presentation of the PHODA \( (D = -6.21, p < .01) \). In addition and regardless of time period, heart rate did not significantly correlate with PTSD severity (all \( p > .05 \)).
As seen in Table 22, the PTSD group showed slightly higher skin conductance than the No PTSD group in all three time periods. Further, the No PTSD group showed higher skin conductance during the PHODA than the SCID, whereas the PTSD group exhibited an opposite pattern. Again due to violation of the assumption of sphericity, a Greenhouse-Geisser correction was used to analyse skin conductance data. The mixed design ANOVA revealed that there was no significant main effect of group ($F(1, 25) = .97, p = .34$) nor a significant interaction effect ($F(1.49, 37.18) = .20, p = .75$). Power to detect these effects was again notably low (.16 and .08, respectively). The time main effect was significant ($F(1.49, 37.18) = 4.34, p = .02, \eta_p^2 = .15$), reflecting a significant linear trend ($F(1, 25) = 6.40, p = .02, \eta_p^2 = .20$). Pairwise comparisons indicated that skin conductance was greater than baseline during administration of both the PHODA ($D = -1.11, p < .01$) and the SCID ($D = -1.16, p = .05$), but did not differ significantly between the two active tasks ($p = 1.00$). In contrast to all other physiological measures, skin conductance measured at baseline and again during administration of the SCID was significantly correlated with PDS-D severity (baseline: $r(29) = .42, p = .02$; SCID: $r(29) = .45, p = .02$).

Table 22 also highlights that respiration rate remained similar between groups and across time periods. Indeed, a mixed design ANOVA revealed no significant effects for group ($F(1, 22) = .07, p = .80$), time ($F(2, 44) = .18, p = .84$) or the interaction ($F(2, 44) = .20, p = .82$). The lack of significant findings might again be attributable to lack of power (all power < .08).

If fear of injury and pain are important aspects of the trauma fear-network (see Sharp & Harvey, 2001), it was expected that pain-related anxiety and fear of movement/reinjury, both general and specific, would be correlated with the degree of physiological arousal experienced (1) when describing the traumatic injury during administration of the SCID and (2) during imaginal exposure to movement (PHODA Task 1). With respect to the former prediction, bivariate correlations revealed a general lack of association between the PASS, TSK and PHODA Fear ratings and physiological arousal recorded during the SCID (all $p > .05$, with one exception). The only significant association was between respiration rate and PHODA Fear ratings. These two variables were negatively correlated ($r(26) = -.39, p < .05$). That is, individuals with low fear of movement/reinjury displayed higher respiration rates when describing their traumatic injury than individuals with high fear of movement/reinjury. In addition, none of the
measures were significantly correlated with baseline physiological reactivity (all $p > .05$).

With respect to the second prediction, none of the self-report questionnaires were significantly correlated with systolic and diastolic blood pressure or skin conductance during imaginal exposure (all $p > .05$). By contrast, heart rate during the PHODA Task 1 was significantly and (unexpectedly) negatively correlated with TSK ($r (28) = -.46, p = .01$) and the PASS Cognitive symptoms ($r (28) = -.40, p = .03$), Fearful Appraisal ($r (28) = -.44, p = .02$) and Escape Avoidance subscales ($r (28) = -.50, p < .01$). That is, people with lower fear of movement/reinjury and lower pain-related anxiety responded with higher heart rate reactivity to presented images of potentially harmful movements. Interestingly, the relationship between heart rate and self-reported physiological symptoms showed only a non-significant trend in the same direction (PASS Physiological symptom subscale: $r (28) = -.34, p = .08$), indicating that higher actual heart rate was weakly associated with lower self-reported arousal. In contrast to the pattern seen for heart rate, respiration rates during administration of PHODA Task 1 were significantly and positively correlated with PASS Cognitive symptoms ($r (27) = .40, p = .04$) and Fearful Appraisal subscales ($r (27) = .54, p < .01$) but no other self-report measures of pain-related anxiety or the TSK (all $p > .05$).

6.3.6 Regression Analyses

Three separate hierarchical regression analyses were performed to determine the degree to which each group of independent variables improved prediction of indicators of adjustment (pain severity, pain-related disability and distress) across the entire sample. The focus was narrowed to these three dependent variables as in Study 1. Pain severity was also included as an independent variable in the regression analysis for pain disability.

Entry of independent variables was ordered according to logical and theoretical considerations, similar to Study 1. Pain severity was given primacy in predicting pain-related disability. For the other two analyses, PTSD diagnosis was entered first (dummy coded) as the primary Axis I disorder, followed by PDS-B, C and D severity scores, in order to determine the relative importance of a diagnosis of PTSD compared to the presence of PTSD-like symptoms. Fear of movement/reinjury (specific then general) and pain-related anxiety were entered, given theoretical arguments and prior evidence
that such measures contribute to pain severity, disability and distress. The two physiological measures (heart rate and respiration) correlated with self-report measures of pain-related anxiety and fear and the physiological measure correlated with PTSD severity (skin conductance) were added as a final step to determine whether these variables contributed uniquely to explain variance in adjustment indicators that was not already explained by the self-report variables.

Table 23 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on pain severity. $R$ was not significantly different from zero until after the third step (thereafter all $p \leq .01$).

### Table 23. Sequential Regression of IVs on Pain Severity

<table>
<thead>
<tr>
<th>Variables (n = 26)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj. $R^2$</th>
<th>Chg</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-1.14</td>
<td></td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.27</td>
<td>.07</td>
<td>.03</td>
<td>.07</td>
<td>1.22</td>
<td>.31</td>
<td>1.81</td>
</tr>
<tr>
<td>PDS-B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.39</td>
<td>-.78</td>
</tr>
<tr>
<td>PDS-C</td>
<td>.44</td>
<td>.19</td>
<td>.04</td>
<td>.12</td>
<td>.17</td>
<td>.38</td>
<td>1.06</td>
</tr>
<tr>
<td>PDS-D</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.25</td>
<td>-.48</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td>.77</td>
<td>.59</td>
<td>.48</td>
<td>.39</td>
<td>8.13</td>
<td>.83</td>
<td>19.06***</td>
</tr>
<tr>
<td>TSK</td>
<td>.77</td>
<td>.59</td>
<td>.48</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
<td>.13</td>
<td>.16</td>
</tr>
<tr>
<td>PASS Cognitive</td>
<td></td>
<td></td>
<td></td>
<td>&lt;.01</td>
<td>.56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASS Fearful Appraisal</td>
<td>.85</td>
<td>.73</td>
<td>.55</td>
<td>.14</td>
<td>.15</td>
<td>.75</td>
<td>1.94</td>
</tr>
<tr>
<td>PASS Avoidance</td>
<td></td>
<td></td>
<td></td>
<td>-.17</td>
<td>-.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASS Physiological</td>
<td></td>
<td></td>
<td></td>
<td>&lt;.01</td>
<td>-.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHODA heart rate</td>
<td>.88</td>
<td>.77</td>
<td>.53</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
<td>.17</td>
<td>.79</td>
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<tr>
<td>PHODA respiration rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.13</td>
<td>-.33</td>
</tr>
<tr>
<td>SCID skin conductance</td>
<td></td>
<td></td>
<td></td>
<td>.22</td>
<td>.24</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. PDS = Posttraumatic Stress Diagnostic Scale, PHODA = Photographic Series of Daily Activities, TSK = Tampa Scale of Kinesiophobia, PASS = Pain Anxiety Symptom Scale, SCID = Structured Clinical Interview for DSM-IV.

***$p < .001$. 

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After step 6, with all IVs in the equation, $R = .88$, $F(13, 12) = 3.17$, $p = .03$. Thus, these variables together accounted for a total of 53% (adjusted) of the variability in pain severity. The power for the final model (calculated as if a simple multiple regression) was .56.

As can be seen from Table 23, only PHODA Fear resulted in a significant increment in $R^2$, indicating that this variable uniquely contributed to the prediction of pain severity. Indeed it was the best predictor of pain severity and its addition to the model represented a large effect size ($f^2 = .85$). By contrast, PTSD diagnosis and PDS symptom severity did not contribute to the prediction of pain severity, which could be due to low power (.37 and .44, respectively). Further, general fear of movement/reinjury, pain-related anxiety and physiological arousal (HR, SC and RR) did not reliably improve the prediction of pain severity, after controlling for PTSD and specific fear of movement/reinjury. Notably, inadequate power was not likely to have been the cause to detect a significant effect for general fear of movement/reinjury (as $p = .695$, power = .70). On the other hand, power when adding each of the other (non-significant) steps to the model was low (power = .49 and .57, respectively).

Table 24 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on the PDI. $R$ was significantly different from zero at the end of each step (all $p < .001$). After step 7, with all IVs in the equation, $R = .96$, $F(14, 11) = 8.23$, $p < .001$. Together these variables accounted for a total of 80% (adjusted) of the variability in the PDI. Notably, the power for the final model (calculated as if a simple multiple regression) was .64.

From Table 24, it can be seen that pain severity (PSI) and specific fear of movement/reinjury (PHODA Fear rating) resulted in a significant increment in $R^2$, indicating that each of these variables uniquely contributed to the prediction of PDI. In the final model, pain severity (entered first) was the best predictor of perceived disability, followed by PHODA Fear rating and then PASS Physiological symptoms. The effect size for adding PSI was large ($f^2 = .59$) and medium for PHODA Fear rating ($f^2 = .22$). PTSD diagnosis, PDS severity, TSK, PASS (entered as separate steps) and PHODA heart and respiration rates and SCID skin conductance (entered as one block) did not reliably improve prediction of PDI scores. Power to detect significant effects
for PTSD diagnosis, PDS severity and PASS was low (power = .38, .57 and .53, respectively), however, lack of power did not explain the absence of significant findings for TSK and physiological measures (power = .83 and .76, respectively).

Table 24. Sequential Regression of IVs on Pain Disability Index (PDI)

<table>
<thead>
<tr>
<th>Variables (n = 26)</th>
<th>R</th>
<th>R²</th>
<th>Adj.</th>
<th>Chg</th>
<th>B</th>
<th>β</th>
<th>F chg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>.39</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSI</td>
<td>.80</td>
<td>.65</td>
<td>.63</td>
<td>.67</td>
<td>4.76</td>
<td>.59</td>
<td>43.75***</td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.83</td>
<td>.68</td>
<td>.65</td>
<td>.04</td>
<td>6.94</td>
<td>.22</td>
<td>2.61</td>
</tr>
<tr>
<td>PDS-B</td>
<td></td>
<td></td>
<td></td>
<td>-1.57</td>
<td>-.37</td>
<td>2.63</td>
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<td>PDS-C</td>
<td>.88</td>
<td>.77</td>
<td>.72</td>
<td>.09</td>
<td>1.06</td>
<td>.29</td>
<td></td>
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<tr>
<td>PDS-D</td>
<td></td>
<td>.67</td>
<td></td>
<td></td>
<td></td>
<td>.16</td>
<td></td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td>.91</td>
<td>.83</td>
<td>.77</td>
<td>.06</td>
<td>37.58</td>
<td>.47</td>
<td>6.10*</td>
</tr>
<tr>
<td>TSK</td>
<td>.91</td>
<td>.83</td>
<td>.76</td>
<td>&lt;.01</td>
<td></td>
<td>.01</td>
<td>.01</td>
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<td></td>
<td></td>
<td>-.12</td>
<td>-.08</td>
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</tr>
<tr>
<td>PASS Avoidance</td>
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<td>.90</td>
<td>.83</td>
<td>.08</td>
<td>.31</td>
<td>.18</td>
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<td>PASS Fearful Appraisal</td>
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<td>-.19</td>
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<td></td>
</tr>
<tr>
<td>PASS Physiological</td>
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<td>-.53</td>
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<td>-.40</td>
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<tr>
<td>PHODA heart rate</td>
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<td>.80</td>
<td>.01</td>
<td>-.21</td>
<td>-.10</td>
<td>.42</td>
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<tr>
<td>PHODA respiration rate</td>
<td>.35</td>
<td>.10</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>SCID skin conductance</td>
<td>.30</td>
<td>.04</td>
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</table>

Note. PSI = Pain Severity Index, PDS = Posttraumatic Stress Diagnostic Scale, PHODA = Photographic Series of Daily Activities, TSK = Tampa Scale of Kinesiophobia, PASS = Pain Anxiety Symptom Scale, SCID = Structured Clinical Interview for DSM-IV.

*p < .05, ***p < .001

Table 25 displays the intercept, unstandardised regression coefficients (B) and the standardised regression coefficients (β) from the final model and R, R², adjusted R², R² change and F change after entry of each block of IVs regressed on the DASS. R was significantly different from zero at the end of each step (all p < .01). After step 6, with all IVs in the equation, R = .94, F (13, 12) = 6.80, p = .001. Together these variables accounted for a total of 75% (adjusted) of the variability in the DASS. The power for the final model (calculated as if a simple multiple regression) was .54.
Table 25. Sequential Regression of IVs on Depression Anxiety Stress Scales (DASS)

<table>
<thead>
<tr>
<th>Variables (n = 26)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj. Chg $R^2$</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ chg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>.82</td>
<td>82.35</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.47</td>
<td>.22</td>
<td>.19</td>
<td>.22</td>
<td>.11</td>
<td>6.69**</td>
</tr>
<tr>
<td>PDS-B</td>
<td>.74</td>
<td></td>
<td>.11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PDS-C</td>
<td>.74</td>
<td>.55</td>
<td>.46</td>
<td>.33</td>
<td>.49</td>
<td>5.11**</td>
</tr>
<tr>
<td>PDS-D</td>
<td>.20</td>
<td></td>
<td>.03</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td>.75</td>
<td>.56</td>
<td>.45</td>
<td>.01</td>
<td>-29.52</td>
<td>-.22</td>
</tr>
<tr>
<td>TSK</td>
<td>.84</td>
<td>.71</td>
<td>.62</td>
<td>.15</td>
<td>1.30</td>
<td>.37</td>
</tr>
<tr>
<td>PASS Cognitive</td>
<td>-.49</td>
<td></td>
<td>-.18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASS Avoidance</td>
<td>.93</td>
<td>.86</td>
<td>.76</td>
<td>.15</td>
<td>.83</td>
<td>.30</td>
</tr>
<tr>
<td>PASS Fearful Appraisal</td>
<td>1.31</td>
<td></td>
<td>.51</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASS Physiological</td>
<td>&lt;.01</td>
<td></td>
<td>-.04</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHODA heart rate</td>
<td>.94</td>
<td>.88</td>
<td>.75</td>
<td>.02</td>
<td>.64</td>
<td>-.19</td>
</tr>
<tr>
<td>PHODA respiration rate</td>
<td>-.62</td>
<td></td>
<td>-.12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCID skin conductance</td>
<td>3.45</td>
<td></td>
<td>.30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* $p < .05$, ** $p < .01$, *** $p < .001$

Inspection of Table 25 indicates that PTSD diagnosis, the PDS-B, -C and -D severity scores (entered as a block), the TSK and the PASS subscales (entered as a block) all resulted in a significant increment in $R^2$, indicating that each of these blocks of variables uniquely contributed to the prediction of scores on the DASS. The addition of PTSD diagnosis represented a medium effect ($f^2 = .28$), while the next three steps all represented large effect sizes ($f^2 = .50, .42, .58$, respectively). The best predictor of DASS scores was Pass Fearful Appraisal followed by PDS-C severity (avoidance/numbing). The PHODA Fear ratings and the block of physiological measures did not reliably improve prediction of scores on the DASS, however, power was low (power = .58 and .57, respectively).

### 6.4 Discussion

The primary purpose of this investigation was to replicate and extend the results of Study 1. First, it aimed to replicate findings that chronic pain patients with clinically diagnosed PTSD would report higher levels of pain severity, health-care utilisation,
pain-related disability and distress than patients experiencing only chronic pain. Second, it sought not only to reproduce the findings of higher pain-related anxiety in individuals with PTSD but also to extend these findings by including measures of fear of movement/reinjury. Third, the study intended to determine whether self-reported pain-related anxiety and fear of movement/reinjury would be correlated with measures of actual physiological reactivity. Finally, the study sought to determine whether these factors predicted signs of maladjustment.

6.4.1 Diagnosis and Classification of PTSD

According to the present data and consistent with findings of Study 1, there were no significant differences between individuals with and without PTSD regarding their history of exposure to traumatic events. Only one individual denied prior exposure to trauma, with all other individuals reporting at least one prior event. Multiple exposure was common, with the mean number of prior events greater than 2 for both groups. This finding poses doubts about the cut off of two or more traumatic events used by Spertus et al. (1999) to divide participants into high and no/low trauma groups. It further highlights that prior traumatic history is not a sufficient cause of PTSD, leaving open the possible role(s) of mediating factors in the development of PTSD. As discussed in Chapter 3, these could include, for example, pre-existing factors (e.g., gender, resilience and neuroticism), peri-traumatic factors (e.g., degree of life threat) and post-traumatic factors (e.g., interpretation of the trauma and its sequelae).

One limitation of Study 1 was its reliance on the PDS, a self-report measure, for diagnosis of PTSD. The results of the current study suggest that the PDS had adequate diagnostic validity, however: it demonstrated high agreement with diagnoses based on the SCID, a semi-structured clinical interview, and the figures were highly similar to published figures for the kappa, sensitivity and specificity of the PDS in relation to the SCID (Foa, 1995). The current finding adds validity to the method of diagnosis in Study 1 and suggests that it is reasonable to compare the results from the two studies, despite differences in their method of diagnosis.

Another concern raised in Study 1 was that the method did not control for the possibility of lifetime PTSD nor current PTSD associated with an event other than the presenting injury, which could have been present in either or both groups. Attempts to remedy this in the present study included the use of the SCID, which permitted
detection of lifetime PTSD and concurrent PTSD due either to pre- or post-injury events, whether or not they were related to the index injury. Classification of individuals on the basis of current, injury or pain-related PTSD resulted in an impure sample, that is, both groups had a relatively high but similar proportion of individuals meeting criteria for lifetime PTSD, retrospectively assessed (36% and 27% for the PTSD and No PTSD groups, respectively).

Although a history of lifetime PTSD was not associated with any of the dependent measures, perhaps a more distinct classification could be pursued in future studies, to enable comparison between individuals with and without chronic pain, with and without current PTSD and with and without lifetime PTSD, associated with either the current injury or unrelated events, in a full factorial design. Clearly this would require a much larger sample size than was available for the current study to achieve sufficient power.

The finding of no significant difference between the PTSD group and No PTSD group across all baseline physiological measures is consistent with the null findings of most earlier studies comparing individuals with PTSD and various control groups (Blanchard, Hickling, Taylor et al., 1996; B. T. Litz, Weathers, Monaco, & al., 1996; Malloy et al., 1983; McFall et al., 1990; Orr, Lasko, Shalev, & Pitman, 1995; Orr, Solomon, Peri, Pitman, & Shalev, 1997; Shalev, Orr, & Pitman, 1992b). In addition, chronic pain patients with PTSD exhibited elevated systolic and diastolic blood pressure, heart rate and skin conductance during exposure to trauma-related stimuli. This is consistent with earlier studies that showed that these variables increased in individuals with PTSD upon exposure to trauma-related stimuli (Blanchard et al., 1982; Casada, Amdur, Larsen, & Liberzon, 1998; Orr et al., 1993; Pallmeyer et al., 1986; R.K. Pitman et al., 1990; R. K. Pitman et al., 1987).

The pattern and magnitude of autonomic elevation during the SCID was unexpectedly repeated by chronic pain patients who had not developed PTSD despite exposure to a similar injury event. This equivalence between groups may be problematic as some researchers have argued that “physiological reactivity upon exposure appears to have more diagnostic value” for PTSD than self-report alone (Orr et al., 1993). On the other hand, even though the present study considered reactivity to personal descriptions of trauma, provided during administration of the SCID, the failure to obtain greater elevations in the PTSD group over the No PTSD group could be
attributable to the nature of the task and the task duration. That is, Pitman et al.'s (1987) seminal study of physiological arousal associated with PTSD comprised scripts edited to a standardised 30-second length, whereas the length of narration varied considerably in the present study. It is therefore possible that recovery occurred within the assessed time frame, attenuating the evaluated level of reactivity. Indeed, inspection of previous data (Casada et al., 1998; Orr et al., 1993) confirmed a lower than expected magnitude change in mean heart rate and skin conductance by the PTSD group during the present study, whereas the magnitude change for the No PTSD group was slightly greater than other controls. The contention is further supported by prior research with PTSD individuals that showed HR returns to baseline levels within at least three minutes (and typically only one minute) of a traumatic imagery task (Orr et al., 1993). Alternatively, the lower level of physiological activation may be attributable to other factors, such as the nature of the stimulus itself. In the present study, participants had significant discretion over the description given in the context of an assessment interview, whereas in other studies the research team refined the imagery/narration of the trauma into a script (for example, via discussion with a psychiatric social worker) to ensure that it included explicit traumatic detail and a standard number of visceral and muscular reactions (R. K. Pitman et al., 1987). The duration of the original trauma here (the majority were motor vehicle accidents or falls) may also have differed from that seen in previous studies involving Vietnam veterans.

6.4.2 The Impact of PTSD on General Pain Severity

Replicating findings from Study 1, there was evidence only of a non-significant trend toward greater general pain severity in the PTSD group compared to the No PTSD group. Further, pain duration and the perceived number of body parts afflicted by pain were equivalent across groups, although individuals with PTSD were more likely to describe their pain as constant. This data contrasts with other evidence that PTSD is associated with increased pain severity (Beckham et al., 1997; Geisser et al., 1996; Sherman, 1997; Sherman et al., 2000). In addition, irrespective of PTSD diagnosis, pain severity assessed both on the day of the study and estimated over the previous month was not significantly related to any measures of physiological reactivity taken at resting baseline, during a reminder of the traumatic injury or during exposure to feared movements. Finally, neither the presence of PTSD nor PTSD severity predicted pain severity, even when they were assigned priority over shared variance. As specific fear of movement/reinjury was the only significant predictor of pain severity, it is possible
that PTSD has been previously associated with higher pain severity, indirectly through its connection with increased fear of movement/reinjury.

In conjunction with similar findings from Study 1, these results cast doubt over theoretical arguments that PTSD exerts influence over chronic pain severity, in particular, those that posit an effect via autonomic arousal (Norton & Asmundson, 2003; Sharp & Harvey, 2001). The current results also conflict with evidence of an association between pain severity and self-reported physiological arousal reported by McCracken et al. (1996, 1998) and Evers et al. (2001). Indeed, it is possible that the PASS Physiological symptoms subscale is more accurately a measure of interpretation bias or hypervigilance to somatic symptoms rather than absolute arousal, a claim buttressed by strong correlations found between the PASS Physiological symptoms and the ASI in Study 1 and an absence of significant correlations between the PASS Physiological symptoms and actual physiological arousal measured in the current study.

6.4.3 The Impact of PTSD on Health Care Utilisation, Pain-related Disability and Distress

The only significant difference across the health care utilisation variables was that individuals with PTSD were more likely to use antidepressants. Low dose tricyclic antidepressants are often prescribed to manage pain symptoms, so it is possible that the differential use of antidepressants attenuated effects of PTSD on pain severity. Post hoc analyses do not support this conclusion, however, as there was no significant correlation between pain severity and antidepressant use ($r(29) = .23, p = .23$) and the trend for greater pain severity in the PTSD group disappeared when the five PTSD patients who were using antidepressants were excluded from the analyses ($t(22) = 1.01, p = .32$). Alternatively, use of SSRIs, which are used to treat PTSD (see Chapter 8), may have attenuated differences between groups otherwise expected in the context of PTSD.

Supporting earlier research data, the present study demonstrated higher pain-related disability for patients with PTSD than for patients without PTSD, even though the two groups were equally likely to be at work and had similar expectations of prognosis. A diagnosis of PTSD also increased the likelihood that the pain patient was seeking or receiving compensation, reflecting similar findings in Study 1 and prior research (Turk & Okifuji, 1996). Positive compensation status was only significantly correlated with perceived disability, however, due to the cross-sectional nature of the study, no
conclusions regarding causality can be drawn. It is possible that secondary gains drive higher reports of pain-related disability, yet it is also possible that greater disability motivates (and necessitates) compensation proceedings. Prospective studies would be required to disentangle causal pathways.

Unsurprisingly, data again supported that pain patients with PTSD exhibit greater distress (including more symptoms of depression, anxiety and stress) than patients who did not have PTSD. Notably, the PTSD group demonstrated similar levels of depression, anxiety and stress ($M = 17.79, 14.07$ and $20.07$, respectively) to norms provided for a clinical sample ($M = 17.24, 15.42$ and $21.59$, respectively) (R. Taylor et al., 2005). Similarly, the No PTSD group’s level of anxiety ($M = 6.47$) was within the expected range for a sample of pain patients ($M = 9.70$) but higher than general community norms ($M = 3.41$). By contrast, their level of depression ($M = 7.40$) more closely reflected general population norms ($M = 5.06$) than those of a representative chronic pain sample ($M = 15.15$) and their level of stress fell midway between the general population ($M = 8.18$) and chronic pain ($M = 17.13$) norms (R. Taylor et al., 2005). It is thus possible that the No PTSD group was not representative of patients with chronic pain. On the other hand, it is likely that the pain population upon which the norms were based included individuals with PTSD, inflating the rates of depression and stress. This is supported by the similarity between means for the entire sample in the current study and pain population norms.

Overall, these signs of maladjustment are compatible with the results of prior research. For example, Asmundson et al. (2000) found that PTSD was more common and severe amongst pain patients classified as dysfunctional copers according to the Multiaxial Assessment of Pain (MAP) than amongst interpersonally distressed or adaptive copers. Similarly, but taking a converse approach, Sherman et al. (2000) found that fibromyalgic patients with PTSD — who reported higher levels of pain, life interference, disability and distress — were best characterised as dysfunctional copers, whereas fibromyalgic patients without PTSD were better characterised as adaptive copers. Geisser et al. (1996) also found that a group of pain patients who had experienced an accident and reported high PTSD reported higher levels of depression

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15 The MAP classifies patients as dysfunctional copers on the basis of high pain severity, interference and affective distress and low self-efficacy and activity.
than pain patients who had either experienced an accident but had low PTSD or who had not experienced an accident.

6.4.4 The Impact of PTSD on Pain-related Anxiety and Fear of movement/reinjury

Higher pain-related anxiety was again demonstrated in the PTSD group in contrast to the No PTSD group. Pain-related anxiety was also associated with PTSD severity. These results parallel findings of higher PASS scores amongst pain-free individuals with high self-reported PTSD severity compared to healthy controls (Asmundson & Carleton, 2005) and amongst males with a trauma history compared to males without a history of trauma (Spertus et al., 1999). Follow-up analyses indicated that the overall difference resulted from increased cognitive and physiological symptoms and a trend towards increased escape and avoidance behaviours.

While the PTSD group were no more likely than the No PTSD group to appraise pain in a fearful way, as measured by the PASS Fearful Appraisal subscale, they were more likely to be fearful of causing reinjury or harm through physical activity, an effect that had not been previously investigated. Indeed, their fear of movement/reinjury was consistently higher across both general physical activity and specific movements. Curiously, general (but not specific) fear of movement/reinjury was correlated with a lower frequency of engaging in specific activities, while specific (but not general) fear of movement/reinjury was correlated with overall perceived disability. It is not possible to entertain causal conclusions from the data. While fear could lead to avoidance of activity and thus disability, it is also possible that avoidance is associated with actual pain experienced during the activity. The latter option is certainly a possibility as ratings of how harmful activities were (PHODA Fear-rating) was the only significant predictor of pain severity in this study.

Despite speculation otherwise, self-reported measures of pain-related anxiety and fear of movement/reinjury were not significantly related to baseline autonomic arousal nor with most measures of autonomic arousal during imaginal exposure to either trauma-related stimuli or a range of specific movements. As an exception, individuals with lower pain-related anxiety (PASS Cognitive symptoms, Fearful Appraisal and Escape and Avoidance) and fear of movement/reinjury (TSK) demonstrated higher heart rate reactivity to imaginal exposure to movements. The reason for lower reactivity in the face of higher fear is not clear, however, in line with Casada et al.’s (1998)
reasoning, it is possible that “heart rate can be interpreted as an indicator of cognitive processing” (p.1042). Applying this logic, it would seem that low fearful participants paid less attention to the presentation, whereas the highly anxious and fearful participants were more attentive to the movements, perhaps reflecting hypervigilance. Greater fear of movement/reinjury was also associated with higher respiration during imaginal exposure to movement. This coupling of hypervigilance and hyperventilation may explain the association with self-reported cognitive symptoms (including impaired concentration and dizziness) during imaginal exposure.

Use of imaginal rather than actual activity is important, as the former is less likely to evoke a pain response, which may confound physiological reactivity measures. However, because compliance with the imaginal task cannot be ensured (it is not possible to assess/control for what people actually imagine or think), confidence in this task is lowered. One participant’s comment at the conclusion of the task speaks to both of these issues. The participant, who had PTSD, stated that because imagining the movements initially elicited pain, after a while she deliberately chose to watch the picture presentation without imagining performing the movements herself. This is in line with Vlaeyen et al.’s (1995) argument that avoidance may have defeated physiological arousal. A few other participants also spontaneously commented that the imaginal exposure elicited a pain response, however, they gave no indication of whether this influenced the way in which they performed the imaginal task. Thus, future research may benefit from investigating whether patients with and without PTSD differ in the level of pain evoked by imaginal exposure and whether this is associated with pain-related fear and anxiety constructs, controlling for compliance with the procedure. Alternatively, actual exposure, controlling for pain levels may be utilised, as in Studies 3 and 4.

In addition, the participants for whom imaginal exposure elicited a pain response typically expressed surprised at this result. In a field dominated by physical therapies and goals of physical activation, this raises imaginal exposure as an alternative or preliminary tool to be utilised in treatment to illustrate that psychological factors can evoke pain in the absence of physical stimulation or activity. Without a physical scapegoat to explain pain, this may hasten modification of maladaptive beliefs by highlighting the discontinuity between pain and actual harm.
6.4.5 Summary, Additional Limitations and Subsequent Studies

Using improved methodology, the present study thus broadly replicated the pattern of previous results, showing that pain patients clinically diagnosed with PTSD are more likely to utilise some aspects of health care, perceive greater disability and experience more distress and pain-related anxiety than pain patients without PTSD. It also extended beyond these findings to show that the traumatised group is also more likely to fear reinjury during movement. To this end, there is clear evidence that PTSD negatively impacts upon adjustment to physical injury and management of chronic pain. By contrast, findings regarding a direct impact of PTSD on pain severity were equivocal at best and challenged theoretical links drawn between anxiety and fear and pain severity, via autonomic arousal. That is, there was little evidence that fear of pain or reinjury elicited autonomic arousal, irrespective of the presence or severity of PTSD.

The data, however, are cross-sectional, from a small sample and only consider general pain levels, rated over an extended period of one month or a single time point at resting baseline. Memory for pain is generally regarded as poor and often inaccurate (Haas, Nyiendo, & Aickin, 2002; Peters et al., 2000) and cognitive, affective, behavioural and physiological responses to pain at rest may differ substantially from those under painful provocation. One possible explanation for the attenuated effects compared to other studies could be heterogeneity in terms of the link between pain and the traumatic injury (Spertus, 2000). Traumatic injury may differ in terms of the centrality of pain to the core trauma, with variation expected in the intensity and nature of pain at the time of injury. For some, pain may have been an unconditioned stimulus during the event, that was immediately associated with the fear network. For others, pain may have become more prominent over time and been subsequently associated to the network as a conditioned stimulus. Before discarding these theoretical links entirely, it therefore would be worthwhile to further examine how PTSD might exacerbate pain during actual painful movement (Study 3) and in response to experimentally induced pain stimuli (Study 4).

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16 One case study identified that pain was in some senses a 'safety cue' for a woman with PTSD, who had interpreted pain as an indicator that she had not died in an MVA (J. Parsons, 2002, personal communication).
CHAPTER 7
EXPLORATION OF THE DIRECT IMPACT OF PTSD ON THE EXPERIENCE OF PAIN AND PAIN-RELATED IMPAIRMENT

Cross-sectional studies, including Study 1 and 2, have drawn attention to a potential link between PTSD and the pain severity reported in relation to the general experience of chronic pain (Beckham et al., 1997; R. A. Bryant et al., 1999; Drottning et al., 1995; Geisser et al., 1996; Koren et al., 2001; Sherman, 1997; Sherman et al., 2000). Other studies have been reviewed in Chapter 3. Several possible mechanisms have been proposed to explain this link, including interpretation biases, hypervigilance, psychophysiological reactivity and biological processes (e.g., HPA and endogenous opioid dysregulation), however, little has been done to empirically test such hypotheses beyond survey methods. Indeed, very few studies have investigated the more specific case of evoked arousal associated with PTSD and its effect on actual pain sensitivity (R.K. Pitman, van der Kolk, Orr, & Greenberg, 1990a) and only one has considered acute pain sensitivity in chronic pain patients with PTSD (Spertus, 2000). The aim of this section was thus to explore the possibility that PTSD directly exacerbates the perception of pain in chronic pain patients, by examining ratings of pain and negative affect and behavioural and physiological responses during exposure to trauma-related scripts and under direct pain provocation.

7.1 Specific Effects of PTSD on Response to Induced Pain in Chronic Pain Patients

In the only experimental study in this area to utilise patients with both PTSD and chronic pain, Spertus (2000) set out to evaluate the effect of PTSD on pain experience during a cold-pressor task. The study specifically addressed reactions to pain rather than perceptions of pain intensity. Comparisons were drawn between 27 pain patients with and without PTSD during baseline and two counter-balanced tasks: pain induction (cold-pressor) and a general stressor (mental arithmetic). Drawing on Foa’s fear network model of PTSD (Foa & Kozak, 1986; Foa et al., 1989), Spertus predicted that pain patients with PTSD would respond to the pain induction in a manner consistent with activation of a trauma-fear network. Thus, she expected to find greater affective distress, physiological reactivity and avoidant behaviour in response to the cold-pressor task in this group ($n = 10$) compared to pain patients without PTSD ($n = 17$). Further,
she expected the effect to be trauma-specific, such that the effect would be more intense than any evoked by the stressful mental arithmetic task.

Contrary to prediction, Spertus found no effect of PTSD diagnosis on affective distress, avoidance or physiological reactivity in response to induced pain. That is, although the PTSD group reported higher levels of affective distress across all tasks, both groups showed relatively similar levels of affective reactivity to both the coldpressor task and mental arithmetic. Even with the opportunity to delay the cold-pressor task for up to 60 seconds, both groups commenced immediately. There was also no sign of differential avoidance evident in behavioural intent; the willingness of the PTSD group to repeat the task and their estimation of future pain tolerance was similar to the No PTSD group. In addition, the groups recorded similar heart rate and systolic and diastolic blood pressure during the cold-pressor task. Further investigation of the potential influence of PTSD on pain is warranted, however, in the face of the study’s sample size and problems with PTSD classification (see discussion in Chapter 3).

One other study has considered similar issues, but the delineation of trauma was not well defined and the chronic pain condition was visceral rather than musculo-skeletal. In 2003, a study was undertaken on pain sensitivity in a group of Persian Gulf war (PGW) veterans with chronic gastrointestinal complaints (n = 12) (Durphy et al., 2003). The PGW veterans all suffered from Irritable Bowel Syndrome and complained of chronic abdominal pain. The control group (n = 12) consisted of a combination of seven civilians and five combat veterans all of whom were pain-free. The study found higher ratings of pain intensity and unpleasantness in the PGW veterans group compared to controls, in response to visceral stimulation (rectal distension) and cutaneous stimulation (immersion of the hand/foot in water at 45°C and 47°C). Although psychological factors (somatic awareness and, to a lesser extent, state anxiety) explained a large amount of variance in the pain measures, it is possible that the effects were simply a function of having chronic pain and bore no relationship to trauma/PTSD. The mixture of combat veterans and civilians in the control group clouds interpretation, by blurring the distinction between the veteran and control groups. Further, although the PGW veterans had seen “stressful” active combat and scored higher than controls in terms of intrusions and dissociation, they did not differ from them in terms of avoidance or trauma on the Trauma Symptom Inventory. Thus the references made by the study
authors to the potential role of trauma-related autonomic arousal is at best speculative and demands further investigation.

Although there are benefits to employing a standard pain stimulus, it is also important to explore whether PTSD has any effect during actual painful movement, a task that is likely to have more clinical relevance than tolerance to painful temperature. In addition, it is important to understand whether any differences in reactions to pain that are associated with PTSD are due to differences in perceived intensity of pain.

7.2 Specific Effects of Trauma-cue Exposure on Intensity of Induced Pain

While Spertus attempted the activation of trauma-related arousal via pain, the possibility exists for trauma to become salient via another route, which might then impact on pain perception. In a small study of 16 Vietnam veterans, Pitman et al. (1990a) investigated the effect of PTSD in the context of experimentally induced arousal and pain. The eight veterans with PTSD were matched according to age and combat severity with eight veterans without PTSD. All were medication free, however, the report did not specify whether any of the individuals suffered from chronic pain. The study differed from Spertus (2000) in directly inducing both arousal (via trauma exposure) and pain (via a thermode). Each participant watched a 15-minute dramatisation of combat before application of a standardised heat stimulus under two conditions: injection of naloxone hydrochloride (an opioid receptor antagonist) and a placebo injection. The cross-over order of conditions was randomised and both experimenter and participants were blind to condition. Of particular interest was that the PTSD veterans exhibited a 30% reduction in pain intensity under conditions of placebo compared to naloxone, an effect absent amongst the veterans without PTSD. Pitman et al. (1990a) concluded therefore that the arousal induced by viewing the combat video had resulted in stress-related analgesia in the PTSD group. Because this was reversible by naloxone, they argued that the mechanism was an endogenous opioid-mediated effect. In light of the small sample size and use of the specific group of Vietnam veterans it is important to replicate these results in other samples and in response to clinically relevant painful stimuli (e.g. movement) to determine whether the effects can be generalised. Furthermore, there has been no study conducted to determine the impact of PTSD when it is comorbid with chronic pain.
Pitman et al.'s (1990a) results parallel general research showing an analgesic effect in the face of stressors (Bobey & Davidson, 1970; Mallow, 1981). By contrast, several experimental studies have shown that physiological reactivity induced by a discrete stressor can have a hyperalgesic effect, for example, increasing self-reported severity and reducing threshold and tolerance during cold pressor and tourniquet ischaemia tasks (Bruehl et al., 1992; Caceres & Burns, 1997; France & Stewart, 1995; Peckerman et al., 1991). More generally, negative emotions are also usually associated with increased pain (Rainville, Huynh Bao, & Cretien, 2005).

Some have argued that the disparity relates to two distinct effects: one of anxiety exerted in anticipation of threat (which produces hyperalgesia) and one of fear during actual exposure (which produces analgesia) (Rhudy & Meagher, 2000). Perhaps one way to reconcile Pitman et al.’s (1990a) findings of analgesia with survey-based studies showing heightened pain severity in chronic pain patients with PTSD (Beckham et al., 1997; Geisser et al., 1996), is to consider that the presence of PTSD results in a general level of hyperalgesia, with analgesia occurring in response to active provocation of PTSD symptoms.

7.3 Use of Personally Relevant Stimuli in Trauma-cue Exposure

7.3.1 Individualised trauma scripts

Pitman et al.’s (1990a) study exposed all participants to the same combat video. As discussed in Chapter 6, however, some findings suggest that physiological reactivity is dependent on exposure to individually relevant cues. For example, Pitman and his colleagues (Orr et al., 1993; R.K. Pitman et al., 1989; R.K. Pitman et al., 1990a) found that veterans with PTSD displayed significant changes in heart rate, skin conductance and or EMG, from baseline, in response to individualised combat scripts. In these studies, participants were exposed to 30-second scripts, developed from their own description of their most stressful combat experiences and pre-combat experience. Each script was written in the second person in the present tense and incorporated visceral and muscular reactions. For exposure, individuals were asked to read their script, meanwhile vividly imagining the event. These studies revealed the importance of using personalised scripts as this pattern of results was not repeated for standard scripts of hypothetical combat experiences. That is, cue specificity and thus access to the trauma network is likely to be improved by use of personalised scripts. The potential effects of PTSD on pain therefore may be augmented under conditions in which participants are exposed to individualised trauma-cues.
7.3.2 Pain stimuli

While a range of studies have been conducted into the nature of visual intrusions, comparatively little is known about somatosensory or motoric re-experiencing beyond case studies and anecdotal evidence (Ehlers et al., 2002; Ehlers & Steil, 1995; van der Kolk & Fisler, 1995). This is an important research oversight as somatosensory perceptions are also dominant features of re-experiencing, reported as present in intrusive memories by 20% to 66% of individuals (Ehlers et al., 2002; Ehlers & Steil, 1995; Hellawell & Brewin, 2004; Michael, Ehlers, Halligan, & Clark, 2005). Feelings of repeated performance of an action were also quite common (Ehlers & Steil, 1995). It has been argued that it is the sensory quality that lends a sense of currency rather than remembrance to the experience (Ehlers & Clarke, 2000). This ‘here and now’ quality can be triggered by a wide range of situations and stimuli, internal or external, semantically and or temporally related to the original event and with or without conscious recollection of the event itself (Ehlers & Clarke, 2000; Reynolds & Brewin, 1998). Further, the “here and now” quality of intrusive memories predicts PTSD symptom severity (Michael et al., 2005).

Aspects of acute and chronic pain could thus reflect a powerful form of re-experiencing, involving somatosensory perception and motoric responses, which in turn evoke intense affect and autonomic arousal (Salomons, Osterman, Gagliese, & Katz, 2004). In contrast to the level of interest in pain memory in amputees (i.e. phantom limb pain) (Katz & Melzack, 1990), only four case studies have documented the role of pain as a phenomenon of re-experiencing specific to PTSD. For example, one study presented a case report of an industrial inspector who fell from a scaffold, sustaining lower back and knee pain (Phillips, Bruehl, & Harden, 1997). As part of his work-conditioning exercises, the inspector was asked to traverse a beam. Doing so triggered trauma-related memories, which were associated with anxiety, autonomic arousal and exacerbated low back pain. Undertaking exposure therapy extinguished not only the re-experiencing symptoms but also the pain response when performing the work-related task. Similarly, Salomons et al. (2004) described two case studies in which surgical patients developed PTSD after experiencing awareness under anaesthesia. Both patients subsequently experienced pain symptoms that duplicated the quality and location of the pain they had experienced during surgery. These symptoms were triggered by cues associated with surgery, such as the “partial anaesthesia” effect associated with drifting off to sleep or seeing blue scrubs. Accompanying the pain, the patients also felt a strong
desire to move or flee. Treatment for PTSD resulted in the elimination or significant reduction of the precise, intense and episodic pain (but had no effect on diffuse “background” pain). Uncontrollable pain during hospitalisation after an eye injury also formed the traumatic core of PTSD experienced by a soldier (Schreiber & Galai-Gat, 1993).

By contrast, based on a collection of studies of different groups (childhood sexual abuse survivors, ambulance service staff and individuals involved in road traffic accidents), Ehlers et al. (2002) concluded that “intrusive memories are usually not [their emphasis] about the pain and other physical sensations during the worst part of the trauma” (p.999). Rather, they argued that intrusive memories were more inclined to include stimuli that had “acquired the status of warning signals: stimuli that if encountered again would indicate impending danger” (p.999). Perhaps movements may acquire this status (particularly having predicted pain during the acute stage) resulting in the development of fear-avoidance.

Even if pain is not re-experienced by all or even a majority of individuals, in view of two points it is important to understand how it might be in some individuals. First, a recent study discovered that chronic pain patients who take a “field” rather than “observer” perspective when remembering painful experiences (i.e. a parallel to non-dissociative re-experiencing) report greater current pain severity (McNamara, Benson, McGeeney, Brown, & Albert, 2005). Second, reliving is most likely to form in response to fear, which in turn is most likely to stem from events involving illness or injury (Reynolds & Brewin, 1999). Therefore, another aim of the following studies was to consider the effects of re-experiencing in chronic pain patients with PTSD.

### 7.4 Study 3

Study 3 was designed with three aims in mind. First, the study sought to examine the general effect of PTSD on chronic pain patients’ range of movement and reported pain intensity, affect and physiological reactivity during the task. In line with prior reports of increased pain severity, fear and perceptions of disability associated with PTSD, it was expected that chronic pain patients with PTSD would report more pain, greater negative affect and display greater restriction of movement and more physiological arousal during a standard range of movement assessment than chronic pain patients without PTSD.
Second, chronic pain and trauma have the potential to operate as reciprocal cues. Therefore, in order to be clear that we were examining the effect of PTSD on pain and movement, the range of movement task was repeated, this time after exposure to an individualised trauma narration. If reported pain was greater during and immediately after trauma-cue exposure than at baseline or during standard assessment within the PTSD group, this would suggest the presence of common networks between pain and PTSD. If there was no or less pain reported during and immediately after the trauma-cue exposure than before exposure this would suggest the likelihood of stress-induced analgesia.

Finally, the study sought to examine the relative contributions of PTSD diagnosis, PTSD severity and fear of movement/reinjury to restriction in range of movement across the entire sample, after controlling for the effects of pain intensity.

7.5 Method

7.5.1 Participants

Twenty nine voluntary participants were included in this study, the same group who participated in Study 2. Details of recruitment and demographic data were described in Section 6.2.1 and 6.3.2. Classification according to clinical diagnosis of PTSD was identical to Study 2 (see Sections 6.2.4 and 6.3.1).

7.5.2 Self-Report Measures

Participants had previously completed a range of self-report measures, including questions about Demographic Background, Injury and Pain Characteristics and Treatment History, as well as the PDS, DASS, PDI, PASS, TSK and PHODA (see Sections 6.2).

7.5.3 Psychophysiological Measures

Physiological measures of SBP, DBP, HR and SC were recorded using the same equipment and methods described in Study 2 (see Section 6.2.5). Respiration rate was also recorded, however, due to technical and software difficulties this data was unreadable and therefore was not analysed. Participants were not given any visual or verbal cues or feedback regarding their physiological reactivity during the experiment.
7.5.4 Assessment of Range of Movement

Range of movement (ROM) was measured using a pendulum inclinometer (Plurimeter, Australasian Medical & Therapeutic Instruments Pty Ltd, Albany Creek, Qld) and tape measure. The inclinometer reading is given in degrees, from a neutral-zero starting point. Standard guidelines for documenting joint motion were followed (Gerhardt, 1994) to assess a series of movements, tested in the following order: for the cervical spine - right and left lateral flexion, right and left rotation, flexion and extension; cervical, thoracic and lumbar flexion (forward bend x 3); and right shoulder abduction. Participants were instructed to commence from a neutral position, standing in a comfortable position, looking straight ahead. They were invited to move as far as possible at a comfortable speed. The position was held only as long as necessary to take the reading and participants could then return to the neutral position. No verbal cues or feedback were given to participants regarding their performance during the experiment. Clinically relevant movements (e.g. lifting) have been used in previous studies of chronic pain patients (Vlaeyen et al., 1995a).

7.5.5 Traumatic Injury Narration (Trauma-cue Exposure)

Exposure to cues of the traumatic injury was facilitated by asking participants to vividly recall and narrate the circumstances of their injury. They were instructed to use the present tense and to include as much detail as possible about their thoughts, feelings, behaviour, motoric responses and sensory experiences (in all 5 modalities) during the event. This instruction was intended to facilitate data-driven processing rather than conceptually-driven processing (Ehlers & Clark, 2001; Brewin & Holmes, 2003).

7.5.6 Procedure

The appointment for the current laboratory session was made at the end of Study 2 or later via telephone. Continuation of informed consent was verbally confirmed at the time of making the appointment and again upon arrival. The session was scheduled at a similar time of day to control for effects due to circadian rhythm.

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17 Individuals were interrupted and reminded to use the present tense if they began the narration using past tense. The past-tense statement was thus repeated by the researcher using the correct tense to improve understanding of the instruction.
Participants were given a brief overview of the procedure for the session on arrival. After participants washed their hands, psychophysiological recording equipment was attached with the participant seated in an upright position. After a 5-minute rest/adaptation period, a 5-minute baseline was recorded, during which the participant was instructed to sit quietly. Participants were next asked to stand and a 1-minute standing baseline was recorded, followed by administration of the first ROM assessment. Participants were given a 10-minute break, during which they were able to adopt a comfortable posture (for example, sitting, standing or walking about) and converse about general topics. Following the break, a second 1-minute standing baseline was recorded before participants were instructed to narrate the circumstances of their injury, with their eyes closed, so as to minimise visual distraction and facilitate absorption in the memory. The second ROM assessment was administered in immediate succession to the traumatic injury exposure. NRS of current pain and affective response were obtained immediately after baseline, pre- and post-ROM, and pre- and post-trauma exposure. Psychophysiological data was recorded continuously throughout each task, with the Finipres device turned off in between tasks. At the beginning and end of each task a manually operated switch was used to insert a marker within the physiological data for use in later off-line analysis.

At the conclusion of the final session, participants were debriefed and thanked. At that point, they were paid a $10 per hour honorarium to reimburse them for their travel costs and time.

7.5.7 Ethics Approval

The Human Research Ethics Committee of the Australian National University approved the protocol for this study. The letter of approval is reproduced in G.

7.5.8 Data Analysis

Prior to data analysis, data was screened for accuracy of data entry, missing values and fit between their distributions and the assumptions of univariate and multivariate analysis. Full ROM data was not available for two participants, one from each diagnostic group, as they were unable to attend for a second laboratory session. These participants were excluded from analyses, leaving a total of 27 participants. Physiological data during the trauma narration and second ROM was not available for a third participant, who was excluded from relevant analyses. Separate examinations
were made within the PTSD and No PTSD groups for univariate outliers, which were deemed where $z > 3.29$ ($p > .001$, two-tailed test) (Tabachnick & Fidell, 2001). To reduce the impact of these scores whilst conserving these data, outliers were recoded to one unit above the nearest respondent's score (Tabachnick & Fidell, 2001). No multivariate outliers were identified, using Mahalanobis distance with $p < .001$.

Restriction for each movement performed under the standard condition was analysed using a mixed design ANOVA, with group (PTSD, No PTSD) as the between group factor and ROM as the repeated measure (with 10 levels, 1 for each movement). Only the main effect of group and simple effects of the potential interaction were examined as differences across movements in terms of ROM would be obvious and irrelevant.

With respect to change in range of movement between the two conditions, standardisation was maintained across participants by calculating the percentage change between each individual's standard assessment and post-exposure assessment, relative to his or her standard ROM. This overcomes the potential inaccuracies likely in absolute measures of change. Two primary variables were isolated. First, the mean percentage change for all movements was calculated (total percentage change in ROM). Second, the movement with the largest negative percentage change (i.e. representing the greatest reduction in ROM) was determined on an individual basis. Thus, the second variable isolated for analysis was chosen as clinically relevant for each individual and therefore was not the same for all participants, whereas the first variable was based on an identical series of movements performed by all participants. Group differences in the total percentage change and greatest percentage change were then compared using independent sample $t$ tests.

Group differences in self-reported pain and tension (NRS) over the five time periods (Baseline, standard ROM, Baseline 2, exposure, post-exposure ROM) were analysed by separate linear mixed models fitting PTSD status as a between-groups fixed effect and time as a within-subjects repeated effect. group x time interactions were also included in each model. The advantages of using a mixed design model to examine repeated measures include using all available data, the ability to account for correlations between repeated measures on the same individuals and no assumption of homogeneity of variance across groups and time points (Tabachnick & Fidell, 2001).
To facilitate data reduction, a Composite Affective score was computed from the remaining four NRS scales. That is, item ratings for distress, anxiety/fear, irritability/anger and sadness were summed to form the Composite Affective NRS variable for each of the five time periods. To examine affective reactivity associated with injury events, the Composite Affectivity NRS of responses during baseline was compared with responses during exposure and during standard and post-exposure ROM assessment, using a two-factor, mixed design ANOVA. Thus, affect served as the dependent variable, while group again represented the first, between-group factor and time was the second factor with repeated measures.

To examine physiological reactivity during administration of the standard and post-exposure ROM tasks, the mean values were obtained for the duration of each movement. The mean values obtained for each of the ten movements were then averaged to give a total mean value for the ROM assessment. Differences in physiological reactivity (SBP, DBP, HR and SC) experienced between the two groups (between groups factor) over the five time periods were then analysed separately in the same manner as for other variables (i.e. using a two-factor, mixed design ANOVA).

For all mixed design ANOVAs, both Mauchley’s test of sphericity and low Epsilon values (<.75) (Hinton et al., 2004) were used to check for violation of the assumption of sphericity. In the face of violation, Greenhouse-Geisser corrections were used to obtain a corrected $F$ value (Hinton et al., 2004; Tabachnick & Fidell, 2001); in all other cases Wilk’s lambda was used. Follow-up univariate analyses were used to examine any significant main effects and pairwise comparisons were used to further examine significant main effects of time or simple effects in the face of a significant interaction between group and time.

Correlation analyses between ROM, pain, negative affect, physiological measures and fear- and anxiety-related measures were conducted. Five separate hierarchical regression analyses were performed to determine the degree to which several factors improved prediction of performance (i.e. ROM), pain intensity and negative affect experienced during performance, across the entire sample. Inclusion of pain intensity and negative affect was aimed to address both the sensory and affective components of pain experience, respectively.
In all instances, the alpha level was set at .05 and Bonferroni corrections were made to maintain this level of Type I error in the context of multiple comparisons, unless otherwise specified. SPSS for Windows (Version 10) was the software used for all analyses. SPSS calculates alpha to three decimal places, therefore all “.000” output values are reported here as p < .001.

7.6 Results

7.6.1 Effect of PTSD and Trauma-cue Exposure on ROM

The results of a mixed design ANOVA, with Greenhouse-Geisser corrections for follow-up univariate analyses, indicated a significant main effect for group \((F(1, 24) = 4.57, p = .04)\) but no significant interaction effect between group and ROM \((F(3.89, 93.41) = 1.85, p = .13)\) for standard ROM.\(^{18}\) That is, the PTSD group reported a lower marginal mean ROM \((M = 49.22, SE \pm 3.61)\) than the No PTSD group \((M = 59.73, SE \pm 3.34)\). Post hoc comparisons further revealed that the PTSD group showed lower ROM than the No PTSD group for two specific movements: cervical flexion \((D = 33.37, p = .01)\) and lumbar flexion \((D = 22.86, p = .03)\) in forward bend. There was no significant discrepancy in ROM between groups for any other specific movement \((all \ p > .12)\).

Cervical and lumbar flexion ROM were both negatively associated with pain during ROM \((r = -.62, p < .001; r = -.29, p = .04)\), PTSD severity \((r = -.60, p = .001; r = -.53, p = .001)\), PASS \((r = -.55, p < .01; r = .31, p = .03)\) and PHODA Fear rating \((r = -.46, p = .01; r = .37, p = .02)\). That is, those participants reporting higher pain, PTSD severity, pain-related anxiety and fear of movement/reinjury showed greater deficits in ROM.

Table 26 illustrates the descriptive data for percentage change in ROM between standard assessment and post-exposure assessment. Inspection of this table highlights that there was no significant difference between groups in percentage change in ROM when all movements were considered, however, power for this effect was low (.28). By contrast, when only the movement showing the largest reduction after exposure was analysed, the PTSD group demonstrated a significantly greater percentage change than the No PTSD group.

\(^{18}\) The main effect of movement was also significant \(F (3.89, 93.41) = 85.62, p < .001\) but of no interest, as different movements are inherently associated with different ranges.
Table 26. Mean ROM data for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>ROM</th>
<th>PTSD (n = 13)</th>
<th>No PTSD (n = 14)</th>
<th>t(25)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Mean % change</td>
<td>4.90</td>
<td>18.64</td>
<td>1.28</td>
</tr>
<tr>
<td>Greatest neg. % change</td>
<td>-52.88</td>
<td>17.46</td>
<td>-31.15</td>
</tr>
</tbody>
</table>

** p < .01.

The greatest negative percentage change in ROM showed moderate positive correlations with the severity of all three symptom clusters of PTSD (PDS-B: \( r(27) = .56, p = .001 \); PDS-C: \( r(27) = .63, p < .001 \); and PDS-D: \( r(27) = .59, p < .001 \)), negative affect experienced during exposure (\( r(27) = .55, p < .01 \)), pain-related anxiety (PASS: \( r(27) = .44, p = .01 \)) and ratings of fear of specific movement/reinjury (PHODA Fear rating: \( r(27) = .59, p < .001 \)), however, fear of general movement/reinjury did not survive correction for Type I error (TSK: \( r(27) = .38, p = .05 \)). Thus, individuals with high levels of reported PTSD severity, affective reactivity to exposure, pain-related anxiety and fear of specific movement/reinjury exhibited greater restriction in ROM following exposure to traumatic injury cues. The greatest negative percentage change was also strongly correlated with perceived disability (PDI: \( r(27) = .77, p < .001 \)) and moderately and negatively correlated with estimates of specific activity frequency (PHODA Frequency of Activity: \( r(26) = .54, p < .01 \)). This means that individuals who showed greater restriction in ROM following exposure also perceived higher levels of pain-related disability and reported a lower frequency of engaging in specific activities.

7.6.2 Effect of PTSD and Trauma-cue Exposure on Self-reported Pain, Tension and Affective Response

Table 27 presents descriptive data for self-reported pain, tension and negative affect, for both groups across time. The results of the mixed ANOVA, using the Greenhouse-Geisser correction for follow-up univariate analyses, on self-reported pain intensity indicated significant main effects for both group (\( F(1, 24) = 5.89, p = .02, \eta_p^2 = .20 \)) and time (\( F(2.395, 57.48) = 9.58, p < .001, \eta_p^2 = .29 \)), however, the interaction effect was not significant (group x time: \( F(2.395, 57.48) = .12, p = .97 \)). Notably, power for the interaction was minimal (.07). Thus, while the PTSD group reported
higher pain than the No PTSD group across all time periods, the degree of change in pain over time was parallel for the two groups. Underlying the main effect of time were significant quadratic ($F(1, 24) = 23.75, p < .001, \eta^2_p = .50$) and cubic ($F(1, 24) = 24.79, p < .001, \eta^2_p = .51$) trends. According to pairwise comparisons, the apparent trends were due to a significant increase in pain reported from baseline to standard ROM ($D = -1.26, p < .01$), which significantly decreased again by baseline 2 ($D = 1.83, p < .01$). Indeed, there was no significant difference between the two baselines ($p > .05$). Further, although pain did not significantly increase during exposure ($p > .05$), it increased overall from baseline to post-exposure ROM ($D = -1.77, p < .001$). Finally, there was no significant difference between the level of pain reported during the two ROM assessments ($p > .05$).

Pain intensity during standard ROM was moderately correlated with PTSD diagnosis ($r(27) = .45, p < .01$), PTSD symptom severity (PDS-B: $r(27) = .47, p < .01$; PDS-C: $r(27) = .53, p < .01$; PDS-D: $r(27) = .51, p < .01$) and general fear of movement/reinjury (TSK: $r(27) = .43, p = .01$). Further, strong correlations existed between this variable and pain-related anxiety (PASS: $r(27) = .71, p < .001$), fear of specific movement/reinjury (PHODA Fear rating: $r(27) = .79, p < .001$) and perceived disability (PDI: $r(27) = .78, p < .001$). A similar pattern of correlations was found when the analyses were repeated for pain intensity during post-exposure ROM.

A mixed ANOVA using a Greenhouse-Geisser correction with follow-up univariate analyses on self-reported tension revealed significant main effects for both group ($F(1, 24) = 7.90, p = .01, \eta^2_p = .25$) and time ($F(2.99, 71.80) = 4.12, p < .01, \eta^2_p = .15$), however, there was no significant interaction (group x time: $F(2.99, 71.80) = .31, p = .82$). Again, power for the interaction effect was low (.11). Similar to the findings for pain, the PTSD group reported higher tension than the No PTSD group, across all time periods; however, the degree of change in tension over time was approximately parallel for the two groups. Exploring the main effect of time, revealed significant results for linear and order four trends (linear: $F(1, 24) = 7.61, p = .01, \eta^2_p = .24$; order four: $F(1, 24) = 4.94, p = .04, \eta^2_p = .17$). Pairwise comparisons indicated that the latter was due to an increase in reported tension during the ROM following exposure to the traumatic injury narrative, although this did not survive the Bonferroni correction ($D = -1.04, p = .03$). There was also a non-significant trend for tension to increase from baseline 2 during the exposure period ($D = -1.29, p = .09$). An independent sample $t$ test was used
to further unpack effects at this time period as it was expected that the PTSD group would show a greater increase in tension than the No PTSD group. Results of analysis on the percentage change in tension (i.e. controlling for baseline differences in tension) indicated a difference in the expected direction, but this was again non-significant after Bonferroni correction (t(17.21) = 1.99, p < .03) but note power was low (.63). It is important to note that levels of tension had returned to baseline after the standard ROM assessment before commencement of the exposure task (p > .05).

A moderate positive correlation was found between the amount of tension and pain reported during standard ROM (r(27) = .69, p < .001). In addition, the level of tension was moderately correlated with PTSD symptom severity (PDS-B: r(27) = .54, p < .01; PDS-C: r(27) = .60, p < .001; and PDS-D: r(27) = .47, p < .01), pain-related anxiety (PASS: r(27) = .67, p < .001), perceived disability (PDI: r(27) = .60, p < .001) and frequency of activity (r(27) = -.52, p < .001). The correlation between tension and fear of movement/reinjury during movement was strong (PHODA Fear rating: r(27) = .72, p < .001). Interestingly, the correlations between tension during ROM and PTSD severity were no longer significant when the ROM was performed after trauma-cue exposure (p > .05 for all PDS subscales). The relationship between tension and PHODA Fear was also weakened in this case (r(27) = .53, p < .01), although relationships with other variables remained similar.

Turning to affective responses, the mixed ANOVA, with Greenhouse-Geisser corrections for follow-up univariate analyses, indicated that both main effects were significant (group: F(1, 24) = 6.63, p = .02, ηp² = .22; time: F(2.90, 69.59) = 12.18, p < .001, ηp² = .34), in the absence of any significant interaction effect overall (group x time: F(2.90, 69.59) = 1.15, p = .33). Power for the interaction effect was low (.29). Again, the PTSD group reported higher negative affect than the No PTSD group, with the non-significant interaction suggesting a parallel profile of affective response over time between groups. Significant linear (F(1, 24) = 11.12, p < .01, ηp² = .32), cubic (F(1, 24) = 16.61, p < .001, ηp² = .41) and order four (F(1, 24) = 14.69, p = .001, ηp² = .38) trends were apparent for the main effect of time. Notably, the two baseline affective ratings and ratings of affect related to the standard ROM were equivalent (all p > .05 for pairwise comparisons), indicating that the standard ROM did not generate a significant negative affective response. By contrast, affective ratings taken in relation to both the exposure and post-exposure ROM periods were significantly higher than
Table 27. Mean self-reported pain, tension and affective responses across time for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Standard ROM</th>
<th>Baseline 2</th>
<th>Exposure</th>
<th>Post-exposure ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td><strong>PTSD (n = 13)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>5.54</td>
<td>3.09</td>
<td>6.88</td>
<td>1.93</td>
<td>5.00</td>
</tr>
<tr>
<td>Tension</td>
<td>4.12</td>
<td>2.97</td>
<td>4.07</td>
<td>2.95</td>
<td>3.62</td>
</tr>
<tr>
<td>Affect</td>
<td>2.79</td>
<td>2.09</td>
<td>2.86</td>
<td>2.37</td>
<td>2.47</td>
</tr>
<tr>
<td><strong>No PTSD (n = 14)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>3.64</td>
<td>1.92</td>
<td>4.82</td>
<td>2.32</td>
<td>3.04</td>
</tr>
<tr>
<td>Tension</td>
<td>1.27</td>
<td>1.42</td>
<td>1.69</td>
<td>1.74</td>
<td>1.38</td>
</tr>
<tr>
<td>Affect</td>
<td>1.01</td>
<td>1.14</td>
<td>.73</td>
<td>.81</td>
<td>.97</td>
</tr>
</tbody>
</table>

Note. ROM = Range of Movement.
baseline \( (D = -1.55, p < .001 \) and \( D = -.69, p < .01, \) respectively), although the affective response during exposure was greater than that during post-exposure ROM \( (D = .86, p = .01) \). As the PTSD group was expected to experience a greater change in negative affect during exposure than the No PTSD group, an independent sample \( t \) test was conducted during this time period for the percentage change (i.e. controlling for baseline differences in negative affect). This was not significant \( (t (25) = .13, p = .45) \), however, power was very low (.07), due to the large standard deviations.

Finally, the degree of negative affect experienced during standard ROM was correlated with pain intensity during the ROM \( (r (27) = .64, p < .001) \), PTSD diagnosis \( (r (27) = .54, p < .01) \), PTSD symptom severity \( (\text{PDS-B}: r (27) = .72, p < .001; \text{PDS-C}: r (27) = .53, p < .01; \text{PDS-D}: r (27) = .46, p < .01) \), pain-related anxiety \( (\text{PASS}: r (27) = .71, p < .001) \) and fear of movement/reinjury \( (\text{PHODA Fear rating}: r (27) = .62, p < .001; \text{TSK}: r (27) = .49, p < .01) \). A similar correlational pattern was found when the analyses were repeated for negative affect during post-exposure ROM.

### 7.6.3 Effect of PTSD and Trauma-cue Exposure on Physiological Reactivity

On the whole, physiological reactivity bore no relationship with all other non-physiological measures (all \( p > .05 \)). Descriptive data for all physiological measures are contained in Table 28. The mixed ANOVA revealed only a significant main effect of time on systolic blood pressure \( (F (4, 96) = 4.92, p = .001, \eta_p^2 = .17) \). Neither the main effect of group nor the interaction were significant \( (\text{group: } F (1, 24) = .09, p = .77; \text{group x time: } F (4, 96) = .83, p = .48) \). Notably, power was low (.06 and .22, respectively). A significant order four trend buttressed the main effect of time \( (F (1, 24) = 19.50, p < .001, \eta_p^2 = .45) \). This was due to an increase in systolic blood pressure during exposure compared to each of the baselines \( (\text{baseline 1: } D = -10.81, p < .001; \text{baseline 2: } D = -9.77, p < .001) \).

For diastolic blood pressure, there was a significant main effect of time \( (F (4, 96) = 12.52, p < .001, \eta_p^2 = .34) \), but not group \( (F (1, 24) = .25, p = .62) \), and their interaction was not significant \( (F (4, 96) = 1.18, p = .33) \), according to a mixed design ANOVA. Power was low for the latter two effects (.08 and .36, respectively). The main effect of time reflected significant linear, quadratic and order four trends \( (F (1, 24) = 23.07, p < .001, \eta_p^2 = .49) \);
Table 28. Mean physiological data across time for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Standard ROM</th>
<th>Baseline 2</th>
<th>Exposure</th>
<th>Post-exposure ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
</tr>
<tr>
<td><strong>PTSD (n = 13)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>111.76</td>
<td>15.32</td>
<td>115.25</td>
<td>21.57</td>
<td>113.28</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>65.66</td>
<td>8.98</td>
<td>72.74</td>
<td>13.88</td>
<td>72.20</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>80.56</td>
<td>11.49</td>
<td>82.08</td>
<td>7.15</td>
<td>80.73</td>
</tr>
<tr>
<td>SC (µmhos)</td>
<td>4.44</td>
<td>2.88</td>
<td>4.87</td>
<td>2.72</td>
<td>4.10</td>
</tr>
<tr>
<td><strong>No PTSD (n = 14)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>114.06</td>
<td>12.45</td>
<td>121.34</td>
<td>18.31</td>
<td>114.62</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>65.87</td>
<td>6.52</td>
<td>73.19</td>
<td>10.31</td>
<td>72.49</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>79.60</td>
<td>10.40</td>
<td>82.72</td>
<td>8.29</td>
<td>82.52</td>
</tr>
<tr>
<td>SC (µmhos)</td>
<td>3.45</td>
<td>2.27</td>
<td>4.60</td>
<td>2.54</td>
<td>3.99</td>
</tr>
</tbody>
</table>

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; SC = skin conductance.
In particular, pairwise comparisons revealed that diastolic blood pressure increased above baseline in all other time periods (all \( p \leq .01 \)). In particular, diastolic blood pressure increased in response to exposure (\( D = -5.62, p < .01 \)), but decreased again during post-exposure ROM to a level equivalent to the pre-exposure baseline (\( p > .05 \)).

A significant main effect of time was found for skin conductance, using the mixed design ANOVA, with Greenhouse-Geisser corrections for the follow-up univariate analyses (\( F(2.38, 57.05) = 5.35, p < .01, \eta^2_p = .18 \)). Again, neither the main effect of group (\( F(1, 24) = .49, p = .49 \)) nor the interaction (\( F(2.38, 57.05) = .65, p = .55 \)) were significant, however, power for these analyses was low (.10 and .16, respectively). According to pairwise comparisons, the significant order four trend (\( F(1, 24) = 12.21, p < .01, \eta^2_p = .38 \)) was due to a significant increase in skin conductance associated with exposure (\( D = -.87, p = .01 \)) and post-exposure ROM (\( D = -1.10, p < .01 \)), compared to baseline.

Heart rate exhibited a different pattern of results to other physiological measures. The No PTSD group appeared to experience a drop in heart rate associated with exposure, whereas the PTSD group appeared to experience a rise in heart rate associated with exposure. The mixed design ANOVA, with Greenhouse-Geisser corrections for follow-up univariate analyses, however, revealed no significant effects for group (\( F(1, 24) = .04, p = .85 \)), time (\( F(2.69, 64.64) = 1.65, p = .19 \)) or the interaction (\( F(2.69, 64.64) = 2.17, p = .11 \)). Power was low for these analyses, however (.05, .39 and .50, respectively).

### 7.6.4 Regression Analyses

Five separate hierarchical regression analyses were conducted to determine predictors of self-reported pain and negative affect during ROM and of performance (i.e. cervical and lumbar flexion during standard ROM and greatest negative percentage change in ROM following exposure).

For the first analysis, with pain severity during ROM as the dependent variable, PTSD symptom severity then PTSD diagnosis (dummy coded) were entered to account
for whether the potential effect of PTSD was dimensional or categorical. Pain-related anxiety and fear of movement/reinjury were added in the third step to determine whether these contributed to explanation of variance in performance, beyond that already explained by PTSD-related variables.

Table 29 displays the intercept, unstandardised regression coefficients (\(B\)) and the standardised regression coefficients (\(\beta\)) from the final model and \(R, R^2\), adjusted \(R^2, R^2\) change and \(F\) change after entry of each block of IVs regressed on pain severity during ROM. \(R\) was significantly different from zero at all steps (all \(p < .01\)). After step 3, with all IVs in the equation, \(R = .86, F (4, 22) = 15.26, p < .001\). Thus, these variables together accounted for a total of 69% (adjusted) of the variance in pain severity reported during ROM. The power for the final model (calculated as if a simple multiple regression) was adequate (.99).

Table 29. Sequential Regression of IVs on pain severity during range of movement (ROM)

<table>
<thead>
<tr>
<th>Variables (N = 27)</th>
<th>(R)</th>
<th>(R^2)</th>
<th>Adj. Chg</th>
<th>(B)</th>
<th>(\beta)</th>
<th>(F) chg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td>1.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PDS-Total</td>
<td>.55</td>
<td>.30</td>
<td>.27</td>
<td>.30</td>
<td>-.03</td>
<td>-.17</td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.56</td>
<td>.31</td>
<td>.26</td>
<td>.01</td>
<td>.67</td>
<td>.15</td>
</tr>
<tr>
<td>PASS Total</td>
<td>.86</td>
<td>.74</td>
<td>.69</td>
<td>.42</td>
<td>.02</td>
<td>.38</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td></td>
<td></td>
<td></td>
<td>6.82</td>
<td></td>
<td>.63</td>
</tr>
</tbody>
</table>

Note. PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptom Scale, PHODA = Photographic Series of Daily Activities.

\(**p < .01, ***p < .001\).

As can be seen from Table 29, PASS Total and PHODA Fear rating (entered as a block) and PDS symptom severity resulted in significant increments in \(R^2\), indicating that these three variables significantly contributed to prediction of pain intensity during ROM. These steps represented large effects (\(f^2 = 1.39\) and \(.37\), respectively). PTSD diagnosis did not reliably improve prediction, however, after controlling for PTSD severity. Power for this effect was .61. Inspection of the coefficients indicated that PHODA Fear rating was the best predictor of pain intensity, followed by the PASS Total. It is noteworthy that the coefficient for PDS symptom severity was negative,
suggesting unexpectedly that lower PTSD severity predicted higher pain intensity during ROM.

For the second analysis, with negative affect during ROM as the dependent variable, general affective distress, as measured by the DASS, was entered first. Next, PTSD symptom severity then PTSD diagnosis (dummy coded) were entered to account for whether the potential effect of PTSD was dimensional or categorical. Pain-related anxiety and fear of movement/reinjury were added in the fourth step to determine whether these contributed to explanation of variance in performance, beyond that already explained by the DASS and PTSD-related variables.

Table 30 displays the intercept, unstandardised regression coefficients (B) and the standardised regression coefficients (β) from the final model and R, R², adjusted R², R² change and F change after entry of each block of IVs regressed on negative affect during post-exposure ROM. R was significantly different from zero at all steps (all p ≤ .01). After step 4, with all IVs in the equation, R = .85, F (5, 21) = 10.49, p < .001. Thus, these variables together accounted for a total of 65% (adjusted) of the variance in negative affect reported during post-exposure ROM. Power for the final model (calculated as simple multiple regression) was .95.

<table>
<thead>
<tr>
<th>Variables (N = 27)</th>
<th>R</th>
<th>R²</th>
<th>Adj. R²</th>
<th>Chg B</th>
<th>B</th>
<th>β</th>
<th>F chg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-2.24</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASS Total</td>
<td>.52</td>
<td>.27</td>
<td>.25</td>
<td>.27</td>
<td>-.02</td>
<td>-.19</td>
<td>9.43**</td>
</tr>
<tr>
<td>PDS-Total</td>
<td>.61</td>
<td>.37</td>
<td>.32</td>
<td>.09</td>
<td>.05</td>
<td>.24</td>
<td>3.58*</td>
</tr>
<tr>
<td>PTSD Diagnosis</td>
<td>.61</td>
<td>.37</td>
<td>.29</td>
<td>&lt;.01</td>
<td>-.53</td>
<td>-.11</td>
<td>.03</td>
</tr>
<tr>
<td>PASS Total</td>
<td>.85</td>
<td>.71</td>
<td>.65</td>
<td>.35</td>
<td>-.05</td>
<td>.78</td>
<td>12.67***</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td>1.58</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. DASS = Depression Anxiety Stress Scales, PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptom Scale, PHODA = Photographic Series of Daily Activities.

*p < .10, **p < .01, ***p < .001.
As can be seen from Table 30, PASS Total and PHODA Fear rating (entered as a block) and DASS Total resulted in significant increments in $R^2$, indicating that these blocks of variables uniquely accounted for variance in negative affect during post-exposure ROM. The addition of these variables reflected medium and large effect sizes ($f^2 = .33$ and $1.03$, respectively) in the prediction of negative affect. There was only a non-significant trend for PDS symptom severity to improve prediction of negative affect, while PTSD diagnosis did not significantly improve prediction, after controlling for PTSD symptom severity. While power was adequate for the addition of PTSD diagnosis to the model ($p = .861$, power = .78), it was low for the addition of PDS Total ($p = .07$, power = .42). Inspection of the coefficients from the final model indicated that PASS was the best predictor of pain intensity, followed by PDS symptom severity. Consistent with this, when this analysis was repeated, reversing the order of entry for DASS and PTSD variables such that the latter were entered first (i.e. redistributing shared variance), the non-significant trend for PDS symptom severity became significant ($R^2$ change = .35, $F$ change = 13.50, $p = .001, f^2 = .48$), whereas the DASS no longer significantly contributed to the explanation of variance ($R^2$ change = .02, $F$ change = .66, $p = .43$), though power was low for the latter step (.45).

For the final three analyses on performance variables, pain intensity during the ROM was entered first, given the expectation that restriction of movement is likely to be influenced by pain intensity. PTSD symptom severity then PTSD diagnosis (dummy coded) were entered next to account for whether the potential effect of PTSD was dimensional or categorical. Pain-related anxiety and fear of movement/reinjury were added last to determine whether these contributed to explanation of variance in performance, beyond that already explained by the other variables.

Table 31 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on standard cervical flexion ROM. $R$ was significantly different from zero at all steps (all $p < .01$). After step 4, with all IVs in the equation, $R = .72$, $F (5, 25) = 4.20, p < .01$. Thus, these variables together accounted for a total of 39% (adjusted) of the variability in standard cervical flexion ROM. Power for the final model (calculated as a simple multiple regression) was .54.
Table 31. Sequential Regression of IVs on standard cervical flexion range of movement (ROM)

<table>
<thead>
<tr>
<th>Variables (N = 27)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj. $R^2$</th>
<th>Chg $R^2$</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F_{chg}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>148.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity</td>
<td>.62</td>
<td>.39</td>
<td>.36</td>
<td>.39</td>
<td>-8.41</td>
<td>-.58</td>
<td>15.25***</td>
</tr>
<tr>
<td>PDS-Total</td>
<td>.70</td>
<td>.48</td>
<td>.44</td>
<td>.09</td>
<td>-1.14</td>
<td>-.38</td>
<td>4.20*</td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.70</td>
<td>.49</td>
<td>.42</td>
<td>.01</td>
<td>-3.45</td>
<td>-.05</td>
<td>.30</td>
</tr>
<tr>
<td>PASS Total</td>
<td>.72</td>
<td>.51</td>
<td>.39</td>
<td>.02</td>
<td>-.03</td>
<td>-.03</td>
<td>.46</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>42.89</td>
<td></td>
<td>-.26</td>
</tr>
</tbody>
</table>

Note. PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptoms Scale, PHODA = Photographic Series of Daily Activities.

* $p = .05$, *** $p < .001$.

As can be seen from Table 31, pain intensity reported during ROM and PDS symptom severity resulted in a significant increment in $R^2$, indicating that these two variables provided a unique contribution to the prediction of standard cervical flexion ROM. The size of each effect was large ($f^2 = .54$) and small ($f^2 = .14$), respectively. By contrast, PTSD diagnosis, PASS and PHODA Fear ratings did not reliably improve the accuracy of prediction, after controlling for the other variables. Power for each of these steps was not ideal (power = .64 and .67, respectively), but unlikely to be the cause of an absence of significance (as $p = .588$ and .638, respectively). When the analysis was repeated, reversing order entry such that the two PTSD variables entered after the PASS and PHODA Fear ratings, the unique contribution of PDS symptom severity became a non-significant trend ($R^2$ change = .09, $F$ change = 4.00, $p = .06$), perhaps attributable to the drop in power (power = .50). The unique contribution of the PASS and PHODA Fear ratings increased, but remained non-significant ($R^2$ change = .03, $F$ change = .05, $p = .82$), despite increased power (.75).

Table 32 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on standard lumbar flexion ROM. $R$ was significantly different from zero only at step 2 ($F$ (2, 23) = 4.45, $p = .02$). Indeed, PDS symptom severity was the only factor to result in a significant increment in $R^2$, indicating that this variable provided a unique contribution to the
prediction of standard lumbar flexion ROM, a medium sized effect ($\hat{f}^2 = .23$) explaining 20% of the variance. Conversely, addition of PTSD diagnosis, PASS and PHODA Fear ratings to the model reduced the ability to reliably predict lumbar flexion, such that the final model predicted only 14% (adjusted) of the variance. Notably, the power for the final model (calculated as a simple multiple regression) was low (.49).

Table 32. Sequential Regression of IVs on standard lumbar flexion range of movement (ROM)

<table>
<thead>
<tr>
<th>Variables (N = 27)</th>
<th>R</th>
<th>$R^2$</th>
<th>Adj. R$^2$</th>
<th>Chg</th>
<th>B</th>
<th>$\beta$</th>
<th>F change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>89.43</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity</td>
<td>.29</td>
<td>.08</td>
<td>.04</td>
<td>.08</td>
<td>2.08</td>
<td>.18</td>
<td>2.14</td>
</tr>
<tr>
<td>PDS-Total</td>
<td>.53</td>
<td>.28</td>
<td>.22</td>
<td>.20</td>
<td>-.82</td>
<td>-.35</td>
<td>6.28*</td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.54</td>
<td>.29</td>
<td>.19</td>
<td>.01</td>
<td>-10.95</td>
<td>-.21</td>
<td>.36</td>
</tr>
<tr>
<td>PASS Total</td>
<td>.56</td>
<td>.31</td>
<td>.14</td>
<td>.02</td>
<td>.02</td>
<td>.03</td>
<td>.31</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td>-32.66</td>
<td></td>
<td>-.26</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptoms Scale, PHODA = Photographic Series of Daily Activities.

$p < .05$.

Table 33 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on percentage change in ROM. $R$ was significantly different from zero at all steps (all $p \leq .001$). After step 4, with all IVs in the equation, $R = .77$, $F (5, 21) = 6.30$, $p = .001$. Thus, these variables together accounted for a total of 51% (adjusted) of the variability in percentage change in ROM following exposure. Power for the final model (calculated as a simple multiple regression) was .54.

As can be seen from Table 33, pain intensity reported during post-exposure ROM and PDS symptom severity resulted in a significant increment in $R^2$, indicating that these two variables significantly contributed to prediction of the percentage change in ROM performance following exposure. Pain intensity was the better predictor and demonstrated a large effect size ($\hat{f}^2 = 1.04$), whereas PDS symptom severity demonstrated a medium sized effect ($\hat{f}^2 = .22$). By contrast, PTSD diagnosis, PASS and
PHODA Fear ratings did not reliably improve prediction, after controlling for the other variables. The absence of a significant effect was unlikely to be a function of low power in relation to PTSD diagnosis (power = .78), but likely in the case of PHODA fear and PASS (power = .34). When the analysis was repeated, reversing order entry such that the two PTSD variables entered after the PASS and PHODA Fear ratings, the unique contribution of PDS symptom severity remained significant, though dropped to a medium sized effect ($R^2$ change = .10, $F$ change = 5.28, $p = .03, f^2 = .19$). Similarly, although the unique contribution of the PASS and PHODA Fear ratings increased, it remained non-significant ($R^2$ change = .07, $F$ change = 1.60, $p = .22$). Again, power was low for detecting this effect (.44).

Table 33. Sequential Regression of IVs on percentage change in range of movement (ROM)

<table>
<thead>
<tr>
<th>Variables (N = 27)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj. $R^2$</th>
<th>Chg $R^2$</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity</td>
<td>.65</td>
<td>.43</td>
<td>.40</td>
<td>.43</td>
<td>4.49</td>
<td>.49</td>
<td>18.51***</td>
</tr>
<tr>
<td>PDS-Total</td>
<td>.74</td>
<td>.55</td>
<td>.51</td>
<td>.12</td>
<td>.70</td>
<td>.36</td>
<td>6.47*</td>
</tr>
<tr>
<td>PTSD diagnosis</td>
<td>.74</td>
<td>.55</td>
<td>.49</td>
<td>&lt;.01</td>
<td>5.15</td>
<td>.12</td>
<td>.04</td>
</tr>
<tr>
<td>PASS Total</td>
<td>.77</td>
<td>.60</td>
<td>.51</td>
<td>.05</td>
<td>-.17</td>
<td>-.30</td>
<td>1.35</td>
</tr>
<tr>
<td>PHODA Fear rating</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>22.40</td>
<td>.21</td>
<td></td>
</tr>
</tbody>
</table>

Note. PDS = Posttraumatic Stress Diagnostic Scale, PASS = Pain Anxiety Symptoms Scale, PHODA = Photographic Series of Daily Activities.

$p < .05$, ***$p < .001$.

7.6.5 Selected Qualitative Findings

Comments made by one of the participants are worth noting. After narrating her traumatic injury, Participant 1, spontaneously described an altered proprioceptive effect, a sense of being “off-centre... moved to the left”. This was consistent with her description of the MVA, in which a tram struck the right-hand side of her car, without warning, before dragging it several hundred metres. It is not clear whether her altered proprioception represented the actual movement of her body during the accident (i.e. a somatic memory) or a “protective” reaction to a currently perceived threat evoked by exposure. Her primary site of pain was her cervical spine and two physical effects were observed that were consistent with her altered proprioception. First, her estimation of
the neutral-zero position (i.e. looking straight ahead) showed substantial error and, second, rotation and lateral flexion to the left exhibited a greater change from standard assessment compared to movements to the right. The same participant also reported two reliving experiences during post-exposure ROM assessment. The first she described as the current sense of looking out of the car window during the accident and the second as the current experience of “spinning”. As there was minimal delay between exposure and ROM assessment, it is not possible to say whether these episodes of reliving would have occurred anyway or whether they were cued by the ROM.

7.7 Discussion

The primary purpose of this investigation was to examine the effects of PTSD on chronic pain patients’ pain intensity, affect, physiological reactivity and range of movement, by comparing patients with and without PTSD on these measures. The general effect of PTSD was thus examined using a standard ROM assessment. This had the dual benefits of, first, involving behavioural observation rather than relying on self-reported disability and, second, illuminating the effect of PTSD (as an underlying disorder) under clinically relevant painful provocation rather than simply at rest or averaged across uncontrolled activity levels (cf. Study 2). ROM assessment was then repeated following exposure to an individualised trauma narrative to examine the potential impact on movement and reactions to pain, associated with current activation of PTSD symptoms.

Based on previous research (Study 1 and 2; Beckham et al., 1997; Geisser et al., 1996; Sherman, 1997; Sherman et al., 2000) it was expected that individuals with comorbid PTSD and chronic pain would report greater pain severity, more subjective tension, greater affective distress and higher physiological arousal when asked to complete a range of painful movements, as compared to individuals who suffered from chronic pain alone. The same patients were also expected to exhibit greater restriction of movement than their non-traumatised counterparts, following their reports of higher pain-related disability (Study 2; see also Study 1). The results were partially consistent with hypotheses. At both baseline and during ROM assessment, the PTSD group reported a higher level of pain, tension and negative affect than the No PTSD group. Deficits in cervical and lumbar flexion, assessed in a forward bend, were also present in the PTSD group compared to the No PTSD group. Contrary to prediction and theoretical models (Norton & Asmundson, 2003), these differences in pain-related
responses and movement under standard conditions were not accompanied by marked differences in physiological arousal. This parallels findings of Study 2, which also found that physiological reactivity did not differentiate between chronic pain patients with and without PTSD.

Following on from theoretical propositions advanced in Chapter 4, painful movement could result in the direct activation of a trauma-fear network in individuals with PTSD, resulting in a cascade of negative affect, physiological arousal and avoidant behaviour (Spertus et al., 1999). Alternatively, the PTSD group could be more inclined to perceive painful movement as representing a current threat, with similar consequences to activation of the fear network. If this had been the case, the PTSD group would be expected to have shown comparatively larger increases in negative affect, subjective tension, objective physiological arousal and, perhaps, pain, from baseline to standard ROM.

Importantly, significant interaction effects of group and time were not demonstrated for these measures under standard ROM conditions. Rather, both groups showed parallel increases in pain, tension and negative affect, indicating that performance of movements did not provoke a discernibly stronger reaction in the PTSD group than the No PTSD group. As painful movement did not appear to result in activation of a trauma-network specific to individuals with PTSD, the differences observed in the PTSD group are more likely the result of the chronic impact of PTSD (i.e. changes that occur insidiously over time) rather than effects triggered on a moment-to-moment basis. This fits with findings from prospective studies that the relationship between PTSD and subjective ratings of persistent medical problems or injury severity (both of which are likely to reference pain) are stronger the further from the event (Ehlers et al., 1998; Jeavons, 2000; Mayou et al., 2002).

The second phase of this experiment provides better clarity on this point. To reduce the effect of individual factors and to increase the immediate salience of PTSD symptomatology, exposure to trauma-related cues was experimentally manipulated before repetition of the ROM assessment. Under these circumstances, it was expected that the PTSD group would again demonstrate more restriction, report higher pain, subjective tension and negative affect, and exhibit stronger physiological arousal than the No PTSD group. Further, if the effect of PTSD was due to a current perception of
threat and or specific activation of a trauma-fear network (i.e. common networks between pain and PTSD), then they should have demonstrated a comparatively greater increase from baseline than the No PTSD group. Moreover, their responses under the post-exposure condition should be also greater than their responses under standard conditions. Alternatively, if there is no or less pain and restriction reported during and immediately after the trauma-cue exposure than before exposure this suggests the likelihood of stress-induced analgesia. Overall, the hypotheses regarding the second phase of the experiment (i.e. those related to the acute relationship between PTSD and pain-related variables) were poorly supported by the current results.

Notably, exposure did not facilitate a concurrent increase in pain in either group, although there was a trend towards increased subjective tension, which was stronger for the PTSD group. Further, although exposure generated significant affective distress for both groups, the PTSD group did not report a larger increase in distress than the No PTSD group. Similarly, both groups experienced a similar increase in blood pressure and skin conductance from baseline to exposure. This raises identical concerns to those raised in relation to Study 2. One explanation for such findings is that the diagnostic classification into groups is problematic, however, affective and physiological reactivity to reminder cues represent only two of the five PTSD re-experiencing sub-criteria listed in DSM-IV TR. Alternatively, the demands of verbally narrating the circumstances of the traumatic injury may have attenuated effects, which have tended to be demonstrated in studies involving passive exposure. An example of this is the visual or auditory presentation of traumatic material that is simply read, watched and or heard (R.K. Pitman et al., 1990; R.K. Pitman et al., 1990a). On the other hand, there was an interaction effect (albeit only a non-significant trend) for heart rate, whereby the PTSD group displayed a rise in heart rate and the No PTSD group displayed a drop in heart rate during exposure. Consistent with expectation, their higher heart rate suggests that the PTSD group engaged in more cognitive and emotional processing during exposure than the No PTSD group. This returns to Casada et al.’s (1998) study, as was discussed in Chapter 6.

A potential problem for the study is that the injury narrative task may have prompted one of three quite different reactions within the PTSD group. That is, participants may have dissociated from the memory and associated affective/physiological responses, they may have experienced a flashback/reliving
episode involving intense affect and physiological arousal or they may simply have perceived the recall as an ordinary autobiographical memory. Each of these responses may impact differently on physiological reactivity, with flashback/reliving experiences expected to elicit greater HR reactivity than dissociative or autobiographical recall (Lanius et al., 2005). The current study did not differentiate between the presence of these three responses and their potential to impact differently on dependent variables. Thus, it is possible that differences between the PTSD and No PTSD groups were masked by different recall experiences within the PTSD group. This possible complexity represents an avenue for further inquiry, which would require using a larger sample, with a better indication of participants’ internal responses during attempts to activate symptoms.

According to Brewin and Holmes (2003), attempts to instruct participants to process material in a particular way are often not consistently related to the experience of intrusive memories. They argued that one implication of this is that deliberate attempts to engage in data-driven processing is weakly associated with spontaneous processing, which may involve altered perceptual thresholds. Despite the problems of modelling effects in the laboratory, where experimental control is required to permit influence of causal effects, alternative methods of induction may need to be developed.

Compared to the No PTSD group, the PTSD group did show a significantly greater percentage change in restriction during post-exposure, but only when the most affected movement was considered. This is arguably a better measure, more sensitive to individual pain problems and idiosyncratic fears, than the mean percentage change (i.e. across all movements combined). Although caution must be applied during interpretation as this latter measure did not significantly differ between groups, the opposite valences obtained by the two groups is also noteworthy. Specifically, the PTSD group demonstrated an overall reduction in post-exposure movement (i.e. negative mean change), whereas the No PTSD group demonstrated an overall improvement in post-exposure movement (i.e. positive mean change), relative to the standard ROM. One possible interpretation of this result could be that exposure facilitated a stress-induced analgesia for the No PTSD group, but a hyperalgesic effect for the PTSD group. Such an effect would contravene expectations guided by Rhudy & Meagher’s (2000) hypothesis (wherein fear induces analgesia and anxiety induces hyperalgesia) as, of the two groups, the No PTSD group would be the one least likely to
experience fear due to exposure. However, consideration of the magnitude of change for the No PTSD group (small) indicated that effectively there was no real change for this group, suggesting that an interpretation of stress-induced analgesia would be inappropriate. Further, the occurrence of a hyperalgesic effect during post-exposure ROM loses plausibility in view of the fact that the PTSD group reported equivalent levels of pain, irrespective of ROM condition. Thus, as ratings of pain and restriction were equivalent before and after exposure in the PTSD group, this could represent either no effect or, perhaps, a cortically mediated effect of hypersensitivity, which was then masked by stress-induced analgesia.

In order of their strength of prediction, fear of movement/reinjury, pain-related anxiety and PTSD severity all uniquely contributed to the prediction of reported pain severity during ROM, together explaining almost 70% of variance. Notably, these variables reflect relatively stable beliefs and symptoms, according to reliability data for each measure (see Chapter 5). In combination with the pattern of mostly null findings related to post-exposure ROM, this result therefore supports the earlier conjecture that the relationship between PTSD and pain appears as a function of chronic comorbid disorder rather than a momentary interaction of symptoms. Further, while pain-specific anxiety and fear were stronger predictors of pain severity than PTSD severity, the latter still independently accounted for almost one third of the variance. This suggests a role for features of PTSD other than fear and anxiety. Of course, the relationship may simply be an artefact of shared method variance due to the coincidental overlap of symptoms between PTSD and chronic pain, such as elevated irritability, impaired concentration and sleep disturbance, a possibility highlighted in Chapter 3. Alternatively, the overlap of symptoms may reflect shared underlying psychological or biological processes, such as anxiety sensitivity or alterations in cortical structures and or central processing (see Chapter 4).

Sixty five percent of the variance in affective distress associated with performing movements with pain was accounted for by a model containing PTSD diagnosis and measures of PTSD severity, pain-related anxiety, fear of movement/reinjury and general affective distress (as measured by the DASS). Only pain-related anxiety and PTSD severity uniquely contributed to the model, however; neither diagnosis of PTSD, fear of movement/reinjury or general affective distress (as measured by the DASS) independently explained further variance, after controlling for PTSD severity. Taken
with the findings for pain severity, the present study illustrates that PTSD severity impacts on both sensory and affective components of the pain experience, which is only partially due to its association with elevated pain-specific anxiety and fear. Arguably this conclusion undermines the previous categorical use of PTSD diagnosis. On the other hand, it was necessary to make a categorical distinction to investigate whether prior research findings could be replicated under conditions of accepted diagnostic criteria and standardised, validated measures of PTSD.

In addition, multiple regression analyses indicated that PTSD severity significantly contributed to the prediction of observed restriction in cervical and lumbar flexion, as well as, individual-specific change in ROM following exposure. Conversely, pain-related anxiety and fear of movement/reinjury did not reliably predict these outcomes, irrespective of whether they were awarded priority over potential shared variance with PTSD. This finding suggests that the influence of PTSD on ROM itself occurs independently of pain-related anxiety and fear of movement/reinjury. This is consistent with other researchers’ conclusions in relation to motor function deficits in patients with moderate to severe Whiplash-Associated Disorders (WAD) compared to mild WAD (Sterling, Jull, Vicenzino, & Kenardy, 2003a; Sterling et al., 2005). Indeed, Sterling et al. (2005) found that IES scores significantly increased the accuracy of predicting moderate to severe symptoms of WAD at six months post injury, whereas fear of movement/reinjury did not have significant predictive value.

It is important to acknowledge other limitations of the study, which may have affected the nature of findings. First, the sample size was small, with implications for power to detect significant effects a relevant consideration given the null findings, thus it would be pertinent for future research to attempt replication with a larger sample. Second, participants had sustained a range of musculo-skeletal injuries in a variety of traumatic events. Pain sites and the centrality of pain to the original trauma therefore varied across individuals, potentially attenuating or masking effects. Future research may thus benefit from selecting a more homogenous group of participants for study.

Notably, although the NRS is an ordinal scale, the numbers reflect rank ordering and not magnitude ratio (Price & Harkins, 1992). For example, a 1-point reduction on an 11-point NRS certainly indicates that the pain is less, however, it would be inaccurate to presume that the pain has reduced by 10%. Patients may also remember
previous ratings and apply them again to be consistent, even if the experience is different (Price & Harkins, 1992). These factors may have influenced the results of this study, which are based on differences in NRS ratings.

Finally, replicating the findings of Study 2, there was little evidence of physiological reactivity in the face of pain, movement or trauma-related exposure in the present study. Indeed, physiological measures were generally unrelated to the other measured variables. Again, this challenges theoretical assumptions that autonomic arousal mediates the relationship between PTSD and pain. Theoretical models have also put forward another form of physiological reactivity that may exacerbate pain—increased muscle tension (Flor et al., 1990; Norton & Asmundson, 2003; Sharp & Harvey, 2001). While Orr et al. (1993) found that EMG was a poor determinant of PTSD hyperarousal, raising doubt about the validity of this argument, their null result can be distinguished as the EMG was of the left lateral frontalis facial muscle and not specific to an injury site or painful region (cf. Flor et al., 1990). Future research may thus have greater success identifying acute links between PTSD and pain by including EMG rather than measures of autonomic arousal. Alternatively, indicators of central rather than peripheral neurophysiological processes may instead be worthy of pursuit. Alteration of global pain threshold (as opposed to changes localised at the injury/pain site) may reflect modification to central processing, which may explain the link between PTSD and pain severity. This possibility is explored in the following study.

7.8 Study 4

The design of Study 4 was structurally identical to Study 3, the two diverging only in the choice of pain induction, with a thermal pain threshold task replacing the ROM assessment in the current study. The thermal pain threshold task was identified for inclusion on two bases: it provided an objective, standardised method of pain induction and permitted investigation of the potential for PTSD to affect pain threshold. Changes in thermal pain threshold can be interpreted as reflecting global hypersensitivity or hyposensitivity when assessed in a remote uninjured bodily location (Sterling, Jull, Vicenzino, & Kenardy, 2003b, 2004).
7.8.1 Effect of PTSD and Trauma-cue Exposure on Pain Threshold in Chronic Pain

There is evidence to suggest that chronic pain conditions, such as chronic lower back pain (Bruehl, Chung, Ward, Johnson, & McCubbin, 2002), whiplash (Curatolo, Petersen-Felix, Arendt-Nielsen, Giani, Zbinden & Radanov, 2001), widespread pain and fibromyalgia (Carli, Suman, Biasi, & Marcelongo, 2002; Geisser, Casey et al., 2003; Geisser, Robinson, Miller, & Bade, 2003) are associated with lower pain thresholds and higher pain sensitivity than healthy controls. A detailed review of this is given elsewhere (Bruehl, McCubbin, & Harden, 1999). More specifically, Sterling et al. (2003b, 2004) found generalised hypersensitivity, including thermal hyperalgesia, differentiated patients with moderate/severe symptoms of whiplash from patients with only mild whiplash symptoms and from individuals who had recovered from initial whiplash. As the hypersensitivity was significant even after controlling for general psychological distress (GHQ-28 scores), Sterling et al. (2003b) concluded that the hypersensitivity could instead be due to central pain processing mechanisms (see also Curatolo et al., 2001; Radanov et al., 1996). Notably, in Sterling et al.,’s study, the group with moderate/severe whiplash was also characterised by higher scores on the IES. This may reflect the simple concurrence of two independent variables in this group, however, it raises a question as to whether PTSD and pain hypersensitivity might be more closely linked.

In the absence of explicit research on the issue, however, the question whether comorbid PTSD alters pain threshold in chronic pain patients is an open one. It is possible that peripheral catecholamine release or central sensitisation processes may be invoked by fear conditioning in PTSD, moderating nociceptive thresholds and resulting in global rather than localised pain sensitivity (Salomons et al., 2004). PTSD-related fear could also contribute to cognitive-affective responses to stimuli, regardless of whether they fall above or below pain threshold, particularly where the stimuli appear to be re-experienced pain (Salomons et al., 2004).

7.8.2 The Role of Hypervigilance in Mediating the Effects of PTSD on Chronic Pain

According to theoretical models of chronic pain (Vlaeyen & Linton, 2000) and PTSD (Ehlers & Clarke, 2000) the tendency to interpret pain or posttraumatic experiences as threatening is likely to evoke fear. This fear may in turn influence responses to pain via the promotion of hypervigilance, which is designed to facilitate escape and avoidance from perceived threats (Roelofs, Peters, McCracken, & Vlaeyen,
2003; Roelofs, Peters, Muris, & Vlaeyen, 2002). Attention tends to affect ratings of both pain intensity and unpleasantness, although a stronger effect has been established for the former (Villemure & Bushnell, 2002). In the combined presence of PTSD and chronic pain the likelihood of hypervigilance and its mediating impact on pain is thought to be heightened (Asmundson et al., 2002; Sharp & Harvey, 2001), however, this notion is yet to be empirically established. It is thus important to distinguish whether apparent differences in threshold are simply a function of hypervigilance.

7.8.3 Aims and Hypotheses

There were four specific aims in conducting Study 4. First, the study aimed to explore whether chronic pain patients with PTSD would demonstrate any differences in global thermal pain threshold compared to chronic pain patients without PTSD. If the general presence of PTSD contributed to central sensitisation processes, it was expected that the PTSD group would display lower global pain thresholds than the No PTSD group. As in Study 3, it was anticipated that chronic pain patients with PTSD would report more affective distress and physiological arousal during standard pain induction than patients without PTSD.

A second aim of the study was to examine whether specific trauma-related cues would elicit an immediate change in pain threshold in chronic pain patients with PTSD but not in pain patients without PTSD. Pain threshold was therefore assessed again after exposure to an individualised trauma narrative. If pain threshold was lower immediately after trauma-cue exposure than at baseline or during standard assessment within the PTSD group, this would suggest the presence of common networks between pain and PTSD. If pain threshold was higher immediately after the trauma-cue exposure than before exposure this would suggest the likelihood of stress-induced analgesia.

Third, the study aimed to control for potential confounds, including hypervigilance and perceived change in ‘usual’ pain. Finally, the study sought to examine the relative contributions of usual pain, PTSD diagnosis, PTSD severity, pain-related anxiety, hypervigilance and physiological reactivity to thermal pain threshold across the entire sample.
7.9 Method

7.9.1 Participants

All participants had participated earlier in Studies 2 and 3 and details of recruitment were provided in Section 6.2.1. The sample for the present study comprised only 20 individuals, due to the later inclusion of measures for this particular study. Classification on the basis of PTSD diagnosis was identical to Study 2 and resulted in eleven participants in the PTSD group and nine participants in the No PTSD group. In addition to previously mentioned exclusion criteria, participants would have been excluded if they had experienced any previous injuries to the volar forearm area or had a current or prior history of neurological disorder. None needed to be excluded using these additional criteria, however.

7.9.2 Self-Report Measures

Participants had previously completed a range of self-report measures, including questions about Demographic Background, Injury and Pain Characteristics and Treatment History, as well as the PDS, DASS, PDI, PASS, TSK and PHODA (see Sections 6.2 and 7.3.3.1).

7.9.2.1 Pain Vigilance and Awareness Questionnaire (PVAQ)

Experimental paradigms have been developed to assess hypervigilance by gauging allocation of attention, usually during Stroop or dot-probe tasks. Although this may provide an objective measure of hypervigilance, it is often more convenient to use self-report measures in clinical settings, such as the Pain Vigilance and Awareness Questionnaire (PVAQ) (Roelofs et al., 2003). This tool was used in the present study to investigate potential effects of hypervigilance to pain on pain detection threshold.

The PVAQ (McCracken, 1997) is a 16-item instrument, developed to assess the degree to which an individual pays attention to or is preoccupied with pain (see Appendix I). With respect to the previous 2 weeks, respondents rate how frequently each item was true of their behaviour. Items are rated on a 6-point scale, ranging from 0 (never) to 5 (always). Sound psychometric properties have been reported for the PVAQ, including good internal consistency (Cronbach’s $\alpha = .83$ to .92) (McCracken, 1997; McWilliams & Asmundson, 2001; Norton & Asmundson, 2003; Roelofs et al., 2003; Roelofs et al., 2002) and adequate test-retest reliability of $r = .77$ to .80 (McCracken (McCracken, 1997; Roelofs et al., 2002). Positive correlation with the Body
Consciousness Questionnaire and negative correlation with Ignoring Pain subscale of the CSQ suggest the scale has good construct validity (McCracken, 1997). Further, high scorers on the PVAQ also reported higher reported pain intensity, emotional distress, psychosocial disability and physician visits (McCracken, 1997). Finally, the PVAQ has demonstrated moderate correlations with questionnaires measuring pain-related anxiety (PASS), fear of movement/reinjury (TSK) and catastrophising (Pain Catastrophising Scale) (Roelofs et al., 2003).

7.9.3 Traumatic Injury Narration (Trauma-Cue Exposure)

The method for the traumatic injury narration was identical to Study 3 (Section 7.5.5).

7.9.4 Psychophysiological Measures

Physiological measures of SBP, DBP, HR and SC were recorded using the same equipment and methods used in Study 2 (see Section 6.2.5). Respiration rate was also recorded, however, due to technical and software difficulties this data was unreadable and therefore not analysed. Participants were not given any visual or verbal cues or feedback regarding their physiological reactivity during the experiment.

7.9.5 Thermal Pain Threshold (TPT) Detection Task

Thermal stimuli were provided by a Peltier thermode-system. The thermode consisted of a metallic plate, with a surface area of 4cm x 4cm, which was applied to the volar surface of the forearm. The thermode contained two sensors, the Pt 100 and a Safety Thermistor. The Pt100 was connected to a device that controlled temperature (Proportional Integrated Derivative, PID), which, in turn, was connected to the power supply. Once the researcher selected the desired temperature, an electrical current passed through the thermode, heating the plate. The Pt100 sensed the actual temperature of the plate and fed this information back to the PID, thereby allowing for the desired temperature to be regulated. Insulation was facilitated through an acrylic plate attached to the reverse side of the thermode. The temperature range for this study was 35°C to 51°C, however, the Safety Thermistor also restricted the maximum temperature of the thermode to 56°C to prevent the possibility of tissue damage to participants. The Safety Thermistor operated so that the power supply was automatically disconnected from the thermode if the temperature exceeded 56°C.
To establish the TPT, thermal stimulus intensities ranging from 35 to a possible maximum of 51°C were applied, each for 5 seconds duration, increasing by increments of 1°C, with an accuracy of 0.1°C. Commencement at 35°C corresponds roughly to the average warmth threshold (Defrin & Urca, 1996). Based on the period taken by the Peltier device to stabilise to the desired temperature, the inter-stimulus interval ranged from 30-45 seconds. In order to prevent sensitisation or response suppression, the thermode was applied to four specified locations on the volar forearms in a clockwise direction. Such rotation ensured that there was an interval of approximately 3 minutes before each location was used again. Four trials were undertaken for each participant. Unless otherwise indicated by results, the temperature intensities ranged from 40 to 51°C for the second and third trials. The participant was aware that the stimuli would be given in increasing order, but was blind to the actual temperatures or the magnitude of each increment. No verbal cues or feedback were given to the participant during the TPT tasks.

7.9.6 Procedure

The appointment for the current laboratory session was made at the end of the session for Study 3 or later via telephone. Continuation of informed consent was verbally confirmed at the time of making the appointment and again upon arrival. The session was scheduled at a similar time of day to control for effects due to circadian rhythm.

On arrival at the laboratory, participants were given a brief overview of the procedure for the session. Participants washed their hands then psychophysiological recording equipment was attached with the participant seated in an upright position. After a 5-minute rest/adaptation period, a 5-minute baseline was recorded, during which the participant was instructed to sit quietly.

Participants were seated beside the Peltier apparatus, with the test arm extended and resting on a pillow on the desk. Seating height was adjusted so that the arm rested comfortably at this level, with no shoulder displacement. The temperature display and recording sheet were occluded from the participant and only visible to the experimenter. All participants were advised that application of the thermode may result in thermal inflammation to the skin of the inner forearm (slight pinkness), which would be only temporary, abating soon after cessation of the experiment.
Participants were then administered three trials of the TPT detection task. The inter-trial interval was approximately 3-4 minutes, determined by the cooling rate of the thermode. Upon completion of the third trial, participants were given a 10-minute break, during which they were able to adopt a comfortable posture (sitting, standing or walking about) and converse about general topics. Following the break, a second 1-minute standing baseline was recorded before participants were instructed to narrate the circumstances of their injury, identical to the procedure in Study 3. The fourth trial of the pain threshold detection task was administered in immediate succession of the narration. NRS of current pain and affective response were obtained immediately after baseline, pre-trauma narration and pre- and post-TPT. Psychophysiological data was recorded continuously throughout each task, with the Finipres device turned off in between tasks. At the beginning and end of each task and stimulus presentation a manually operated switch was used to insert a marker within the physiological data for use in later off-line analysis.

Participants were debriefed at the conclusion of the session. At that point, participants were also paid a $10 per hour honorarium to reimburse them for their travel costs and time.

7.9.7 Ethics Approval

The Human Research Ethics Committee of the Australian National University approved the protocol for this study (see Appendix G).

7.9.8 Data Analysis

Prior to data analysis, data was screened for accuracy of data entry, missing values and fit between their distributions and the assumptions of univariate and multivariate analysis. Separate examinations were made within the PTSD and No PTSD groups for univariate outliers, which were deemed where $z > 3.29$ ($p > .001$, two-tailed test) (Tabachnick & Fidell, 2001). Three univariate outliers were found on the affective NRS, another on baseline heart rate and two more on skin conductance during standard TPT and exposure. To reduce the impact of these scores whilst conserving data, all outliers were recoded to one unit above the nearest respondent’s score (Tabachnick & Fidell, 2001). No multivariate outliers were identified, using Mahalanobis distance with $p < .001$. 

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Independent sample t-tests or MANOVAs were conducted to determine whether PTSD and No PTSD groups differed on the primary demographic variables of age and number of prior traumatic exposures, pain characteristics (PSI, baseline pain, pain duration, number of medications and providers) and self-report measures (PDS, PDI, PHODA, TSK, PASS and DASS). Chi-square analyses could not be conducted to compare gender, educational background or work status between the two groups due to insufficient expected cell counts (i.e. \( n < 5 \)).

Standard TPT was determined by averaging the detection threshold over the three trials. All three trials were included to minimise the potential impact of novelty, anticipation and habituation effects. Differences in TPT (dependent variable) were then analysed using a two-factor, mixed design ANOVA, with follow-up univariate analyses. Group (PTSD, No PTSD) was included as a between-groups factor and TPT condition included as a repeated measure with two levels (standard and post-exposure).

To facilitate data reduction, a Composite Affective score was computed from the five individual NRS scales. That is, item ratings for distress, anxiety/fear, tension, irritability/anger and sadness were summed to form the Composite Affective NRS variable for each of the three time periods (Baseline, standard TPT, post-exposure TPT). The value used for the standard TPT represented the mean of the three standard trials. Differences in affect were then analysed using a two-factor, mixed design ANOVA, with follow-up contrasts. Thus, affect served as the dependent variable, while group again represented the first, between-group factor and this time, time was the second factor with repeated measures, with three levels (baseline, standard TPT, post-exposure TPT).

To examine physiological reactivity during standard administration of the TPT, the mean values were obtained for the 10-second period commencing with the 5-second application of the painful heat stimuli and including the 5-second period following cessation of the stimuli. The mean values obtained for each trial were then averaged to give a total mean value. The mean value for physiological data obtained during administration of the TPT following exposure were calculated in an identical manner. Baseline physiological measures were defined as the mean value obtained across the 1-minute resting period. Differences in physiological reactivity (SBP, DBP, HR and SC)
experienced between the two groups (first factor) over time (second factor, repeated measure) were thus analysed separately using a two-factor, mixed design ANOVA, with follow-up univariate analyses. Time had five conditions: baseline, standard TPT, baseline 2, exposure and post-exposure TPT.

For all mixed design ANOVAs, both Mauchley's test of sphericity and low Epsilon values (<.75) (Hinton et al., 2004) were used to check for violation of the assumption of sphericity. In the face of violation, Greenhouse-Geisser corrections were used to obtain a corrected $F$ value (Hinton et al., 2004; Tabachnick & Fidell, 2001), while in all other cases Wilk’s lambda was used.

Correlation analyses between TPT, affect, physiological measures, PVAQ and fear- and anxiety-related measures were conducted. Separate hierarchical regression analyses were finally performed to determine the unique contribution each variable or block of variables made to predicting TPT.

In all instances, the alpha level was set at .05 and Bonferroni corrections were made to maintain this level of Type I error in the context of multiple comparisons, unless otherwise specified. SPSS for Windows (Version 10) was the software used for all analyses. SPSS calculates alpha to three decimal places, therefore all “.000” output values are reported here as $p < .001$.

7.10 Results

7.10.1 Demographic and Self-Report Data

Full details of the demographic background, pain characteristics and results of self-report measures for the sub-set of participants included in Study 4 are presented in Appendix H, Table H.1. Notably, the smaller sample’s characteristics remained similar to those reported in Study 2 for the larger sample. There were more females in the No PTSD group (89%) than the PTSD group (36%), otherwise the two groups did not differ with respect to age, educational background or number of prior traumatic exposures (all $p < .05$). Pain duration, number of medications and number of treatment providers were similar between groups, despite significant differences in pain severity index and baseline NRS pain. Reflecting the results for the larger sample, the PTSD group displayed significantly higher scores on the PDS, PDI, PHODA Frequency of Activity scale, TSK, PASS and DASS. Finally, the PTSD group obtained significantly higher
scores \( (M = 55.14, SD = 15.83) \) than the No PTSD group \( (M = 32.67, SD = 19.70) \) on the PVAQ \( (t(18) = 3.08, p < .01) \), indicating that the presence of PTSD increases the degree to which chronic pain patients are hypervigilant towards their pain.

### 7.10.2 Effect of PTSD and Trauma-cue Exposure on TPT

Table 34 illustrates the descriptive data for TPT conducted under standard and post-exposure conditions. Inspection of this table suggests, first, that the PTSD group had a lower TPT than the No PTSD group, irrespective of TPT condition. Second, the PTSD group’s TPT was lower in the post-exposure condition compared to the standard condition, whereas there was no apparent difference between conditions for the No PTSD group. The results of a mixed design ANOVA, however, indicated that the interaction between group and condition was not significant \( (F(1, 18) = .59, p = .45) \), nor were the main effects of either variable (group: \( F(1, 18) = 2.10, p = .17 \); condition: \( F(1, 18) = .70, p = .42 \)). Notably, the power to detect significant effects was low (.28, .12, and .11, respectively).

**Table 34. Mean TPT for PTSD and No PTSD groups**

<table>
<thead>
<tr>
<th>Condition</th>
<th>PTSD ((n = 11))</th>
<th></th>
<th>No PTSD ((n = 9))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( M )</td>
<td>( SD )</td>
<td>( M )</td>
</tr>
<tr>
<td>Standard TPT</td>
<td>46.42</td>
<td>4.43</td>
<td>48.70</td>
</tr>
<tr>
<td>Post-exposure TPT</td>
<td>45.55</td>
<td>5.70</td>
<td>48.67</td>
</tr>
</tbody>
</table>

Standard TPT demonstrated significant negative associations with negative affect during TPT administration \( (r = -.48, p = .03) \), PDS-C severity \( (r = -.45, p < .05) \), PASS total \( (r = -.53, p = .02) \) and PDI \( (r = -.46, p = .04) \). That is, individuals with higher current negative affect, post-traumatic avoidance and numbing, pain-related anxiety and pain-related disability displayed lower TPT. Higher systolic blood pressure during administration was also associated with higher TPT \( (r = .46, p = .04) \). Relationships between standard TPT and PSI, PDS-B severity and PHODA Fear rating only approached significance (all \( .05 < p < .10 \)). By contrast, there were no significant associations between standard TPT and usual pain during administration, PVAQ, DASS, TSK or other physiological reactivity (all \( p > .05 \)).
7.10.3 Effect of PTSD and Trauma-cue Exposure on Self-rated usual Pain and Affective Response During TPT

Descriptive statistics for self-reported usual pain and negative affect during baseline and TPT tasks are provided in Table 35. With respect to usual pain, mixed design ANOVA indicated that the main effect of time and the interaction of group and time were significant ($F(2, 36) = 10.89, p < .001$, $\eta_p^2 = .38$, and $F(2, 36) = 4.27, p = .02$, $\eta_p^2 = .19$, respectively), however, the group main effect represented only a non-significant trend ($F(1, 18) = 3.97, p = .06, \eta_p^2 = .18$). Finding only a trend rather than a significant effect could be explained by low power (.47). Exploration of these effects, via pairwise comparisons, indicated that within each group similar levels of usual pain were reported between baseline and standard TPT ($p = 1.00$), with only a non-significant trend for the PTSD group to report higher pain intensity than the No PTSD group for each of these periods ($p < .09$). Pain reported during the post-exposure TPT period, however, was higher in the PTSD group compared to the No PTSD group ($D = -2.874, p = .04$) and both groups reported higher pain in this period than in either of the previous periods ($D = -.73$ and -.67, for baseline and standard TPT, respectively).

Table 35. Mean self-reported pain and negative affect experienced across time for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Standard TPT</th>
<th>Post-exposure TPT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
</tr>
<tr>
<td>PTSD ($n = 11$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>5.09</td>
<td>2.06</td>
<td>5.35</td>
</tr>
<tr>
<td>Negative affect</td>
<td>2.51</td>
<td>1.94</td>
<td>3.13</td>
</tr>
<tr>
<td>No PTSD ($n = 9$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>3.22</td>
<td>2.06</td>
<td>3.07</td>
</tr>
<tr>
<td>Negative affect</td>
<td>.56</td>
<td>.75</td>
<td>.33</td>
</tr>
</tbody>
</table>

A mixed design ANOVA, using a Greenhouse-Geisser correction for follow-up univariate analyses, showed significant main effects for both group ($F(1, 18) = 9.43, p$

19 Usual pain within each time period was defined as the individual’s rating of pain associated with his or her experience of chronic pain, in contrast to pain evoked by application of the thermode.
<.01, \eta_p^2 = .34) and time period (F (1.39, 24.99) = 14.30, p < .001, \eta_p^2 = .44) on self-rated affective response during the experiment, however, there was no significant interaction effect (group x time: F (1.39, 24.99) = 2.44, p = .12). Power for detecting a significant interaction effect was low (.38). Irrespective of the time period, the PTSD group reported greater negative affect than the No PTSD group. The main effect of time could be characterised with both linear and quadratic trends (F (1, 18) = 15.22, p = .001, \eta_p^2 = .46; F (1, 18) = 10.73, p < .01, \eta_p^2 = .37, respectively). Indeed, negative affect during the post-exposure TPT period was higher than for both of the other periods, according to pairwise comparisons (baseline: D = -1.52, p < .01; and standard TPT: D = -1.32, p = .001).

### 7.10.4 Effect of PTSD and Trauma-cue Exposure on Physiological Responses to TPT

Table 36 illustrates the descriptive statistics for physiological measures taken across five time periods (Baseline, standard TPT, baseline 2, exposure and post-exposure TPT). A mixed design ANOVA, using a Greenhouse-Geisser correction on follow-up univariate analyses, found a significant main effect of time on systolic blood pressure (F (2.42, 43.54) = 8.51, p < .001, \eta_p^2 = .32), however, the effect of group reflected only a non-significant trend for higher systolic blood pressure in the No PTSD group (F (1, 18) = 3.46, p = .08) and there was no significant interaction between group and time (F (2.42, 43.54) = .67, p = .54). Power for detecting the latter two effects was low (.16 and .17, respectively). Subsequent pairwise comparisons to examine the linear trend across time (F (1, 18) = 14.03, p = .001, \eta_p^2 = .44) highlighted a significant increase in systolic blood pressure from both baselines and the standard TPT to the post-exposure TPT task (cf. baseline: D = -8.73, p = .01; standard TPT: D = -6.76, p = .05; baseline 2: D = -10.34, p = .02).

Inspection of Table 36 suggests that the PTSD group had lower resting diastolic blood pressure than the No PTSD group, that the two groups had similar diastolic blood pressure during standard TPT, baseline 2 and exposure and then diverged again during the post-exposure TPT, with the PTSD group’s diastolic blood pressure increasing and the No PTSD group’s diastolic blood pressure decreasing. According to a mixed design ANOVA, however, only time had a significant main effect (F (4, 72) = 4.19, p < .01, \eta_p^2 = .19); there was no significant effect of group (F (1, 18) = .04, p = .84) nor an interaction between group and time (F (4, 72) = 1.40, p = .24). Power to detect the latter two effects was low (.05 and .41, respectively). A significant order four trend was
Table 36. Mean physiological data across time for PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Standard ROM</th>
<th>Baseline 2</th>
<th>Exposure</th>
<th>Post-exposure ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
</tr>
<tr>
<td>PTSD ($n = 11$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>106.94</td>
<td>11.50</td>
<td>111.18</td>
<td>10.11</td>
<td>104.99</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>64.27</td>
<td>7.19</td>
<td>67.51</td>
<td>7.01</td>
<td>63.52</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>69.32</td>
<td>9.46</td>
<td>70.16</td>
<td>7.36</td>
<td>69.86</td>
</tr>
<tr>
<td>SC (µmhos)</td>
<td>5.99</td>
<td>2.78</td>
<td>6.45</td>
<td>2.75</td>
<td>5.63</td>
</tr>
<tr>
<td>No PTSD ($n = 9$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>115.95</td>
<td>6.72</td>
<td>115.64</td>
<td>4.87</td>
<td>114.69</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>66.75</td>
<td>10.35</td>
<td>67.44</td>
<td>11.36</td>
<td>63.14</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>78.50</td>
<td>4.70</td>
<td>76.81</td>
<td>6.28</td>
<td>77.68</td>
</tr>
<tr>
<td>SC (µmhos)</td>
<td>5.62</td>
<td>2.18</td>
<td>5.91</td>
<td>2.48</td>
<td>5.04</td>
</tr>
</tbody>
</table>

Note: SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; SC = skin conductance.
evident with respect to the main effect of time \((F(1, 18) = 10.35, p < .01, \eta^2_p = .37)\), with exposure associated with a significant increase in diastolic blood pressure irrespective of group \((D = -4.66, p = .03)\).

Turning to heart rate observed in the two groups over time, a mixed design ANOVA revealed a different pattern for this measure compared to other physiological measures. For heart rate, although there was no significant main effect of time \((F(4, 72) = .78, p = .56)\), this is likely due to low power (.23). There was a significant main effect for group \((F(1, 18) = 4.65, p = .05, \eta^2_p = .21)\) and a significant interaction \((F(4, 72) = 2.49, p = .05, \eta^2_p = .12)\). Overall, a significantly lower heart rate was observed in the PTSD group compared to the No PTSD group, irrespective of time. Underlying the interaction, however, was a significant linear trend \((F(1, 18) = 4.24, p = .05, \eta^2_p = .19)\) due to the No PTSD group exhibiting higher heart rate than the PTSD group at both baselines \((D = 3.47, p = .02\) and \(D = 3.61, p = .04\)) during standard TPT \((D = 3.10, p = .05)\) and exposure \((D = 3.20, p = .04)\), but not during post-exposure TPT \((p = .27)\).

Finally, the results in Table 36 suggest skin conductance increased in response to both TPT tasks, irrespective of group. A mixed design ANOVA, with Greenhouse-Geisser corrections for follow-up univariate analyses, revealed a significant main effect for time \((F(1.94, 34.99) = 5.32, p = .01, \eta^2_p = .23)\). The main effect of group and the interaction were not significant \((F(1, 18) = .21, p = .65\) and \(F(1.94, 34.99) = .08, p = .92\), respectively), but power to detect significant effects was low (.07 and .06, respectively). The main effect of time could be characterised by significant linear, cubic and order four trends \((F(1, 18) = 5.13, p = .04, \eta^2_p = .22; F(1, 18) = 6.60, p = .02, \eta^2_p = .27; F(1, 18) = 12.07, p < .01, \eta^2_p = .40\); respectively). Pairwise comparisons revealed, however, that the only significant change in skin conductance over time was a reduction from standard TPT to baseline 2 \((D = .84, p = .02)\).

### 7.10.5 Regression Analysis

A hierarchical regression was conducted to investigate the predictive value of variables for TPT across the entire sample. As chronic pain has been associated with sensitisation processes, pain severity (PSI) was entered first. PDS-Total was entered next to determine whether PTSD severity contributes to TPT. Given that differences in TPT may be associated with psychological processes, the PVAQ and PASS were
entered next. It was also of interest to determine whether TPT was influenced by systolic blood pressure during assessment.

Table 37 displays the intercept, unstandardised regression coefficients ($B$) and the standardised regression coefficients ($\beta$) from the final model and $R$, $R^2$, adjusted $R^2$, $R^2$ change and $F$ change after entry of each block of IVs regressed on standard TPT. $R$ approached significance for the first step ($p = .07$), but did not significantly differ from zero until the fourth step, indicating that pain severity and PTSD severity (on their own) and hypervigilance and pain-related anxiety (as a block), did not reliably improve the prediction of TPT. The power to detect a significant effect was low, however, for those two steps, (power = .41 and .42, respectively). After step 4, with all IVs in the equation, $R = .74$, $F (4, 14) = 3.35$, $p = .03$, reflecting that only systolic blood pressure made a unique contribution to explanation of the variance in TPT. The addition of this variable represented a large effect size ($f^2 = .34$). Nonetheless, these variables altogether accounted for a total of 38% (adjusted) of the variance in standard TPT. Based on the coefficients from the final model, pain-related anxiety, followed by hypervigilance and systolic blood pressure were stronger predictors of TPT.

Table 37. Sequential Regression of IVs on standard Thermal Pain Threshold (TPT)

<table>
<thead>
<tr>
<th>Variables (N = 20)</th>
<th>$R$</th>
<th>$R^2$</th>
<th>Adj.</th>
<th>Chg</th>
<th>$B$</th>
<th>$\beta$</th>
<th>$F$ change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>22.95</td>
</tr>
<tr>
<td>PSI</td>
<td>.41</td>
<td>.17</td>
<td>.12</td>
<td>.17</td>
<td>-.51</td>
<td>-.29</td>
<td>3.63#</td>
</tr>
<tr>
<td>PDS-Total severity</td>
<td>.49</td>
<td>.24</td>
<td>.15</td>
<td>.07</td>
<td>&lt;.01</td>
<td>-.01</td>
<td>1.58</td>
</tr>
<tr>
<td>PVAQ</td>
<td>.59</td>
<td>.34</td>
<td>.17</td>
<td>.11</td>
<td>.11</td>
<td>.53</td>
<td>1.20</td>
</tr>
<tr>
<td>PASS Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.06</td>
<td>-.66</td>
<td></td>
</tr>
<tr>
<td>Standard TPT SBP</td>
<td>.74</td>
<td>.55</td>
<td>.38</td>
<td>.20</td>
<td>.24</td>
<td>.50</td>
<td>6.20*</td>
</tr>
</tbody>
</table>

Note. PSI = Pain Severity Index, PDS = Posttraumatic Stress Diagnostic Scale, PVAQ = Pain Vigilance and Awareness Questionnaire, PASS = Pain Anxiety Symptoms Scale, SBP = systolic blood pressure.

# $p < .10$, *$p < .05$. 

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7.11 Discussion

The primary purpose of this investigation was to examine the effects of PTSD on global pain hypersensitivity in chronic pain patients. Consideration was given as to whether PTSD exerted a general effect and or a specific effect following direct activation of PTSD symptoms. Although the data suggested that the PTSD group had a lower TPT than the No PTSD group, the difference was not statistically significant. This null result is contrary to the expectation that chronic pain patients with PTSD would display a global reduction in pain threshold, due to central sensitisation mechanisms (cf. Sterling et al., 2003b, 2004).

Under standard conditions and across the entire sample, lower TPT in the present study was associated with higher current negative affect, severity of post-traumatic avoidance and numbing, pain-related anxiety and pain-related disability. In contrast, hypervigilance was not related to TPT. TPT was also positively associated with systolic blood pressure. Indeed, systolic blood pressure was the only variable to reliably improve accuracy of predicting TPT. This result is consistent with general findings regarding pain severity and blood pressure (Flor, Miltner et al., 1992), but conflicts with other research which found no correlation between systolic blood pressure and pain threshold, intensity or tolerance (Al’ Absi, Petersen, & Wittmers, 2002; Caceres & Burns, 1997). The latter studies are perhaps distinguishable, however, on the basis that they used a cold pressor task rather than thermal stimuli.

Turning to the second phase of the experiment, visual inspection of the data suggested a pattern consistent with prediction, with the PTSD group recording an additional reduction in TPT following exposure to the trauma narrative but no corresponding effect for the No PTSD group. As with the standard assessment of TPT, this pattern was not statistically significant, however, indicating that direct activation of PTSD symptoms did not affect pain threshold, irrespective of whether PTSD status was taken into account. This is consistent with other research that found widespread hypersensitivity in patients with whiplash injuries was independent of state anxiety (Scott, Jull & Sterling, 2005).

Notably, while ratings of usual pain remained unaffected by standard assessment of TPT, both groups reported an increase in usual pain during post-exposure TPT. In addition, the PTSD group reported a stronger increase in pain than the No PTSD group
at this point, although this did not significantly predict change in TPT. Across the board, the PTSD group gave higher ratings of negative affect than the No PTSD group. They also experienced greater negative affect during post-exposure TPT than during baseline or standard administration of the TPT task, but again this was not significantly associated with change in TPT. Thus while the PTSD group were negatively affected by exposure in terms of their experience of usual pain and affective distress, exposure had no significant effect on global TPT, either directly or indirectly via the effects on usual pain and affect.

Aside from confirmation that PTSD has no effect on global pain sensitivity, several possible reasons exist for the essentially null effects in the present study. First, the small sample size is acknowledged again, although other studies have demonstrated significant effects of pain and physiological responses in PTSD using similarly sized, if not smaller, samples (R.K. Pitman et al., 1990a). It is worth noting that Curatolo et al., (2001) reported that patients with whiplash demonstrated lower pain thresholds for a range of pain threshold tests, except for heat threshold, suggesting that the choice of threshold test may explain the null findings. Another explanation for the failure to demonstrate an effect of exposure on TPT could be that any reactivity to exposure may have dissipated before achievement of pain threshold. Use of a TPT assessment method with a shorter task duration in future research may overcome this potential problem. Fifth, the present study used an ascending order of stimulus presentation for determining TPT. A common complaint is that this procedure may attract response bias due to expectation and that random order stimulus presentation is preferable. On the other hand, while randomisation removes some expectations, it may create others, for example, that all stimuli will be painful if early stimuli are high range. Indeed, in support of this argument others have demonstrated flattened curves and reduced pain thresholds in random series compared to an ascending series presentation (Nielsen, Price, Vassend, Stubhaug, & Harris, 2005). Sixth, coping style may have influenced results. During the present study attempts were made to minimise potentially distracting stimuli by conducting the study in the controlled environment of a laboratory setting. Participants were encouraged not to speak outside required verbal responses and were instructed to attend to the pain stimuli and to concentrate on providing an accurate determination of threshold. Despite these instructions, some participants attempted to converse, either about the heat stimuli, injury-related experiences or unrelated topics, highlighting perhaps a level of distractibility or avoidance, which
might have influenced the accuracy of TPT detection. These factors were not measured, however, the nature of PTSD suggests that individuals with this comorbid disorder might be more prone to such problems. Additional explanations include the perceived relative pleasantness of thermal temperature (especially for those patients who might use heat packs for pain relief or testing conducted during winter months).

Finally, pain intensity is difficult to test realistically in an experimental environment. In this study, participants were aware of the source of pain, when it would occur and how long it would last and (due to ethical requirements) they were informed that the stimuli would not result in tissue damage. Participants also possessed a level of control to request the cessation of the stimuli and ultimately to withdraw from the experiment. It is therefore not clear how these ratings might relate to ratings of a ‘real’ pain experience due to an internal (and in the case of non-specific chronic pain) uncertain source of uncertain duration. That is, when the cause and duration of pain is uncertain, unpredictable and uncontrollable pain is more likely to be perceived as threatening, affecting ratings of intensity and affective distress. Arguably, acute experimentally-induced pain will access the immediate cognitive-affective stage of pain, consisting of an automatic response to the moment-to-moment threat and intrusion of pain, but not the reflective cognitive-affective stage of pain, which elaborates on the meaning of pain in terms of its interruption of daily activities and implications for one’s life and contributes to suffering (Price & Harkins, 1992).

Future studies could thus seek to address these methodological issues and incorporate alternative methods of pain threshold assessment, such as cold pain or pressure. In addition, based on the symptom-specific hypothesis espoused by Flor et al. (1992), a potential avenue for future research to explore is whether PTSD could instead only influence pain threshold at the specific pain site.
CHAPTER 8
IDENTIFICATION OF INJURY, CHRONIC PAIN AND PTSD AND BELIEFS ABOUT THE HELPFULNESS OF TREATMENT OPTIONS: A COMPARISON BETWEEN THE GENERAL PUBLIC AND HEALTH PROFESSIONALS

8.1 Effective Treatment
8.1.1 Chronic Pain

Numerous studies have been conducted regarding the efficacy of different treatment interventions for chronic musculoskeletal pain, therefore it is useful to turn to the results of systematic reviews and meta-analyses for an informed summary of findings. Flor, Fydrich and Turk (1992) reviewed 65 controlled and non-controlled studies and concluded that multidisciplinary treatments were effective and superior to no treatment, waitlist and unidimensional treatments. Cutler and colleagues considered 37 controlled and non-controlled studies of multidisciplinary and non-surgical treatments and concluded that non-surgical treatment of chronic pain was effective in facilitating return to work (Cutler et al., 1994). Guzman and colleagues reviewed 10 randomised controlled studies that compared multidisciplinary biopsychosocial rehabilitation to inpatient or outpatient non-multidisciplinary rehabilitation, usual care or wait-list controls (Guzman et al., 2001). They found strong evidence of improved functional outcomes following intensive multidisciplinary rehabilitation compared to inpatient or outpatient non-multidisciplinary rehabilitation and moderate evidence of reduced pain intensity compared to outpatient non-multidisciplinary rehabilitation and usual care.

In conjunction with multidisciplinary treatment, interventions informed by cognitive behavioural therapy (CBT) are also empirically favoured. One study reported the results of a meta-analysis that showed CBT to produce greater changes for disability, positive cognitive coping and appraisal, psychological state and reduced behavioural expression of pain, when compared to wait list controls (Morley, Eccleston, & Williams, 1999). On the other hand, no significant effects of CBT were shown for mood, negative cognitive coping and appraisal or social role function, when compared to other active treatments. According to a systematic review of randomised controlled trials for chronic low back pain, there is evidence that behavioural treatments have a
moderate positive effect on pain intensity and small positive effects on function and behavioural outcomes compared to wait-list or no treatment controls (van Tulder et al., 2001). By contrast, a systematic analysis of review papers and controlled clinical trials did not find any strong evidence that any specific physical therapy produced long term results better than placebo for chronic musculoskeletal disorders, although any treatment (including placebo) was better than no treatment at all (Feine & Lund, 1997).

Current clinical guidelines for pain management focus predominantly on acute pain rather than chronic pain. Nonetheless, they reflect the above findings. They highlight the lack of evidence for many physical/passive\(^\text{20}\) treatments, recommend against bed rest, emphasise early activity and exercise and suggest a chronological ‘stepped’ treatment plan that includes multidisciplinary or psychosocial assessment (Borkan, Reiss, & Ribak, 1995; Clinical Standards Advisory Group, 1994; Dutch College of General Practitioners, 1996; McNeil et al., 1994; [NHG], ; Roland et al., 1996; Royal College of General Practitioners, 1999; W. O. Spitzer, LeBlanc, Dupuis, & al, 1987; Victorian WorkCover Authority, 1996). Notably, the above collection of guidelines indicates the broad national (e.g., ACT, VIC, NSW) and international (e.g., New Zealand, UK, Netherlands, Israel) support for such recommendations.

### 8.1.2 PTSD

Under the umbrella of the Cochrane Collaboration, a systematic review of randomised controlled trials of psychological treatments for PTSD was conducted (Bisson & Andrew, 2005). They found evidence that both trauma focused cognitive behavioural therapy (TFCBT) and stress management interventions were more effective than other therapies and waitlist or usual care in reducing PTSD symptoms and diagnosis. The TFCBT was also more effective than non-trauma focused therapies and stress management interventions over the longer term, demonstrating a significant effect over the former by the time of follow up and better maintenance of treatment gains than the latter at follow up. Strong evidence for the effectiveness of CBT for PTSD has also been reported in two meta-analyses of controlled studies (Bradley, Greene, Russ, Dutra, & Westen, 2005; Sherman, 1998). The practice guidelines developed for the International Society for Traumatic Stress Studies (Foa, Keane, & Friedman, 2000) are consistent with these reviews in their recommendations for using CBT (particularly

\(^{20}\) Passive treatment refers to treatments that do not require active participation by the patient.
exposure and cognitive therapy) and pharmacotherapy (i.e. antidepressants (SSRIs, MAOIs, TCAs) and anticonvulsants) as first-line treatments for PTSD.

NSW practice guidelines developed specifically for those with MVA-related PTSD also recommend CBT as the first line of treatment within two weeks of the accident and specific adjunctive pharmacotherapy when symptoms persist beyond three months (Motor Accidents Authority, 2003). These recommendations are supported by three studies that demonstrated the specific effectiveness of CBT for treating PTSD following a road traffic accident above waitlist controls, supportive psychotherapy, self-help and repeated assessment (Blanchard et al., 2003; Ehlers, Clark et al., 2003; Fecteau & Nicki, 1999).

8.1.3 Comorbid Chronic Pain and PTSD

Interest in specific treatment for comorbid chronic pain and PTSD is in its infancy. It is not yet possible to draw firm conclusions as to whether treatment for one disorder will lead to spontaneous improvement in the other, whether both conditions must be treated independently nor whether treatment is hindered in the face of comorbid conditions. As far as we are aware, there has been no systematic, controlled evaluation of the relative benefit of single modality treatment. Nor has there been evaluation of the effectiveness of treatment specifically designed to address PTSD and chronic pain concurrently. Some preliminary evidence from non-controlled trials and case studies provides some insight, however.

Two studies found no significant impact of comorbidity on single modality treatment. Bovasso (1993) found patients with chronic low back pain achieved similar outcomes following a functional restoration program, irrespective of whether the individual was also diagnosed with PTSD. In a case study of six individuals with PTSD and chronic pain following MVAs, it was found that a manualised PTSD treatment was effective for reducing PTSD symptoms (Shipherd, Beck, Hamblen, Lackner, & Freeman, 2003). In addition, they noted improvements in pain-related function, although there was no change in pain severity.

Several findings from other studies suggest, on the other hand, that it may be inappropriate to provide treatment as if the two conditions were independent. For example, greater pain severity (as assessed prior to treatment) was one of several factors
that contributed to achieving only partial response to CBT treatment specifically targeting PTSD (Gillespie et al., 2002; S. Taylor et al., 2001). Further, reductions in pain severity were found to predict reductions in PTSD symptom severity following a 12 week CBT program for PTSD (Fedoroff et al., 2000). Likewise, headache patients responded less well to CBT for pain management if they had comorbid PTSD, requiring longer treatment than their non-traumatised counterparts and needing specific treatment of PTSD symptoms before headaches resolved (Hickling et al., 1992; Muse, 1986).

8.2 Knowledge and Beliefs about Diagnoses and Effective Treatments

Health literacy refers to the capacity to access, understand and use information to promote and maintain good health (Nutbeam, Wise, Bauman, & al, 1993). By extension, mental health literacy has been defined as “knowledge and beliefs about mental disorders, which aid their recognition, management and prevention” (Jorm, Korten, Jacomb, Christensen, Rodgers et al., 1997). In both cases, literacy includes a range of factors such as the ability to recognise and distinguish different symptoms and disorders, the ability to obtain relevant information, the level of awareness of available treatment options (including self-management and therapeutic intervention), judgements about the utility of those treatments and views about prognosis.

8.2.1 Pain Management Literacy

Ample evidence highlights that maintenance of activity or early resumption of normal activities and avoiding bed rest are important predictors of positive outcomes in acute musculoskeletal pain conditions (Waddell, Feder, & Lewis, 1997). Similarly, most cases of acute pain do not warrant early investigation or specialist referral (Deyo & Phillips, 1996). Further, chronic pain is best managed by a cognitive-behavioural multidisciplinary intervention (as reviewed above).

 Nonetheless, surveys of general practitioners indicate that many do not follow this evidence in their recommendations to patients or provide very general advice (Cherkin, Deyo, Wheeler, & Ciol, 1995; Little et al., 1996). This divergence in practice has been linked to the general practitioner’s knowledge and beliefs and to the patient’s expectations or preferences (Chew-Graham & C., 1999; Daykin & Richardson, 2004; Little et al., 1998; Little et al., 1996; Schers, Brasperning, Drijver, Wensing, & Grol, 2000).
Research has shown recent improvement in the beliefs held by Norwegian general practitioners and physiotherapists about chronic low back pain (Ihlebaek & Erikson, 2004), by Canadian general practitioners, family physicians and chiropractors in relation to acute whiplash (Ferrari & Russell, 2004) and by Victorian general practitioners about low back pain in response to a state-wide public health campaign (Buchbinder, Jolley, & Wyatt, 2001). For example, Ferrari & Russell (2004) found that only 1% of physicians and no chiropractors believed that acute whiplash patients should be prescribed bed rest or that they should not return to work until pain had completely resolved. Further, they reported that 89% of physicians and 76% of chiropractors believed patients should be encouraged to maintain ordinary activities and 91% of physicians and 84% of chiropractors agreed that exercise therapy was an effective treatment strategy. Physicians were more likely to agree that non-steroidal antiinflammatories and muscle relaxants would be effective, whereas chiropractors were more likely to endorse passive treatments (such as traction, transcutaneous electrical nerve stimulation, manipulation, massage and acupuncture) as effective.

8.2.2 Mental Health Literacy

In Australia, Jorm and colleagues have investigated mental health literacy amongst health professional groups and the public through several large national studies, with depression and schizophrenia as the primary foci (Caldwell & Jorm, 2000; Jorm, 2000; Jorm, Korten, Jacomb, Christensen, & Henderson, 1997; Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997; Jorm, Korten, Jacomb, Rodgers, Polliit et al., 1997). Similar international studies have also been conducted on schizophrenia, depression and mania (Chen, Parker, Kua, Jorm, & Loh, 2000; Kua, Parker, Lee, & Jorm, 2000; Parker, Mahendran, Yeo, Loh, & Jorm, 1999; Yeo et al., 2001). These studies have assessed the ability of a range of health professionals and the public to identify these psychiatric disorders from a vignette. In addition, they have measured attitudes and beliefs regarding risk factors, the utility of interventions and likely prognosis for these disorders. Relatively little is known, however, about individual’s knowledge and beliefs regarding other psychological disorders.

One important finding from Jorm's research is that a significant proportion of the general community fail to recognise the symptoms of psychiatric disorders and have a poor understanding of psychiatric terms (Jorm, 2000). For example, although the majority of the public recognised the presence of a 'mental health problem' in vignette
scenarios, only a minority correctly identified depression and schizophrenia (39% and 27%, respectively) and a significant minority failed to identify a mental health issue and or attributed the problem to a physical cause. When appropriate assessment and treatment is provided this may not be a problem, however, there are indications that psychiatric disorders may be missed by professionals, particularly those involved in primary care, when mental health problems are not presented as such (Jorm, 2000) or when they co-occur with physical problems.

In addition, results of Jorm’s research program indicated that the public and professional groups held widely disparate views regarding the helpfulness of potential interventions for depression and schizophrenia. Standard interventions considered generally helpful by professionals (e.g., antidepressants, psychotherapy) were frequently regarded with more suspicion by the public, who often deemed these treatments as more harmful than helpful and instead favoured a less medicalised approach involving lifestyle changes (Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997). These discrepancies may reflect that the public is simply uninformed about evidence-based interventions and thus dependent on unreliable information sources, core beliefs or personal bias. Regardless, in the absence of change, there is certainly potential for this to impact on the degree to which the public will seek out and comply with treatment recommendations (Jorm, 2000; Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997). Conversely, prevention, early intervention and facilitation of adaptive self-management may be improved by increasing the mental health literacy within the community.

Despite their erroneous beliefs about treatment, the public tend to hold more favourable attitudes towards outcomes than professionals (Jorm, Korten, Jacomb, Christensen, & Henderson, 1997).

Overall, professionals have been found to hold generally similar beliefs to each other about usual interventions for depression and schizophrenia, however, there were some important differences between general practitioners, psychiatrists, clinical psychologists and mental health nurses (Jorm, Korten, Jacomb, Christensen, & Henderson, 1997; Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997). Clinical psychologists and mental health nurses were less supportive of medical interventions than other professional groups and, together with general practitioners, were more likely than psychiatrists to support psychological, psychosocial or lifestyle interventions (Caldwell & Jorm, 2000; Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997). Not
surprisingly, each profession was also more likely to favour their own profession as the 
most helpful (Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997).

Research in primary care settings has also shown that practitioners misdiagnosed or 
failed to recognise underlying psychological problems in almost half of their actual 
patients (Borus, Howes, Devins, Rosenberg, & Livingston, 1988; D. Goldberg, Steele, 
Jonhnson, & Smith, 1982; Higgins, 1994; Ormel, Koeter, van den Brink, & van de 
Willige, 1991). Detection appears to be related to characteristics of both providers and 
patients, including knowledge and attitudes about symptoms as well as the way in 
which the patient presents the symptoms (Badger et al., 1994; Coyne, Schwenk, & 
Fechner-Bates, 1995; DelVecchio-Good, Good, & Cleary, 1987; Doherty, 1997; 
Robbins, Kirmayer, Cathebras, Yaffe, & Dworkind, 1994; Tylee, Freeling, & Kerry, 
1993). For example, detection rates are influenced by a patient’s somatisation 
(interpretation and presentation of psychological distress as physical symptoms) (Cape, 
2001; De Wester, 1996; Kirmayer, Robbins, Dworkind, & Yaffe, 1993; Weich, Lewis, 
Donmall, & Mann, 1995) and symptom attribution style (Bower, West, & Tylee, 2000; 
Greer, Halgin, & Harvey, 2004; D. Kessler, Lloyd, Lewis, & Gray, 1999; Kolk, 
Hanewald, Schagen, & Gijsbers van Wijk, 2002). Thus, the likelihood that a 
practitioner will recognise a psychological disorder is reduced if the patient attributes 
symptoms to environmental events or physical illness, rather than psychological 
distress. Conversely, the rate of detection is increased when the patient explicitly raises 
the problem and presents it as a psychological one. This then raises a potential problem 
in the case of PTSD, which is commonly comorbid with physical injury or somatic 
complaints.

8.2.3 PTSD Literacy

Few studies exist on the recognition of PTSD in primary care settings. In the 
United States, primary care practitioners are reasonably sensitive to the presence of 
anxiety and depression but tend to overlook the specific diagnosis of PTSD (Samson, 
Bensen, Beck, Price, & Nimmer, 1999). In a large study of attendees to primary care 
clinics in Israel, researchers found a very poor detection rate for PTSD (Taubman-Ben- 
Ari, Rabinowitz, Feldman, & Vaturi, 2001). The prevalence of PTSD in the sample was 
9%, however, physicians recognised only 6 from 247 cases of PTSD (a recognition rate 
of 2%). There were few false positive diagnoses, with PTSD suggested for 8 of 373 
individuals (2%) who did not meet criteria. Individuals with PTSD were more likely to
be recognised by physicians as generically “distressed” (49%) rather than experiencing PTSD.

Another study that aimed to assess mental health literacy regarding PTSD in a naturalistic setting was conducted amongst primary medical care providers, social workers and mental health providers servicing Bosnian refugees in Chicago (Weine, Kuc, Dzudza, Razzano, & Pavkovic, 2001). Findings indicated, not surprisingly, that mental health providers demonstrated greatest knowledge about PTSD compared to other providers. Less than half of all providers routinely assessed for PTSD or used standardised testing and, although psychotherapy and psychiatric evaluation were ranked as the first and third interventions of choice, providing no intervention was ranked second.

Munro and colleagues set out to assess recognition of PTSD and selection of appropriate treatment using a postal vignette study (Munro, Freeman, & Law, 2004). Practitioners across two primary care regions in Scotland were provided with three vignettes: PTSD, acute stress reaction and depression. For the PTSD vignette, 67% of the GPs included PTSD in differential diagnosis and 87% referred to secondary care. Only 43% of GPs and 54% of psychiatrists specified use of a selective serotonin reuptake inhibitor (SSRI), the pharmacological intervention of choice for PTSD. The percentage of GPs who appeared to have accurate knowledge to recognise and prescribe appropriately for PTSD (28%) was markedly lower than for depression (90%). Similarly, while description of best practice reached almost 50% for depression, it was only 10% for PTSD. Importantly, this study differs from the three studies mentioned above in its use of vignettes, which control for differences in patient variables (such as the tendency to somatise, poor insight or reluctance to discuss psychological symptoms).

8.3 Study 5

This study aims to contribute to the body of mental health literacy research by assessing knowledge and beliefs about PTSD and chronic pain, as there has been little consideration in the empirical literature of mental health literacy in the context of physical injury. It is possible that individuals could either underestimate or overestimate the degree of psychological comorbidity with physical conditions. For example, there is
some suggestion that the presence of a current physical injury may actually hinder the recognition of psychological problems. Alternatively, one might discredit a pain condition as purely psychosomatic because there are strong psychological symptoms.

In addition, the study aims to compare the knowledge and beliefs about chronic pain and PTSD arising from a traumatic injury held by the public with those held by health professionals. This is important, as there may be limits to delivering evidence-based treatment if the public have beliefs different from health professionals regarding the nature of the problem and the most appropriate treatment. Clearly in a multidisciplinary environment, there is also a need for professionals to work not only collaboratively with their patients but also with each other to deliver quality treatment and service, so an understanding of differences between professional groups is also necessary. Finally, it is important to understand the public’s perceptions of chronic pain and PTSD in order to indicate the level of understanding and support provided by family, friends, employers and co-workers etc, which will ultimately impact on the course of chronic pain and PTSD. Identification of the current beliefs of each group may thus help to foster improved clinical practice and greater communication between professionals and between professionals and their patients.

In summary, this study assesses members of the public and health professionals’ ability to identify the presence of comorbid PTSD and chronic pain in an individual injured in a motor vehicle accident. Second, it assesses beliefs about interventions and outcomes for simple chronic pain and chronic pain comorbid with PTSD, comparing differences between the public and health professionals, and between four different health professions.

8.4 Method

8.4.1 Participants

The regional telephone book (Canberra Region 2004 White Pages) was used to randomly select 925 members of the public. The telephone book was chosen over the electoral roll as a more up-to-date listing of local residents because the study was conducted more than three years after the last electoral roll was compiled. Nearly all households (98%) have a telephone connection (Australian Bureau of Statistics, 2001), suggesting that the telephone book provides a reasonable means of identifying the general population. Profession-based telephone listings (Canberra Region 2004 Yellow
Pages) and professional association listings were used to select participating health professionals. Within the nominated health professions of general practitioners (GPs), psychologists, chiropractors and physiotherapists, all practitioners listed as currently practicing in the ACT were contacted where address details were available. Thus, 243 GPs, 64 psychologists, 60 physiotherapists and 38 chiropractors were invited to participate.

8.4.2 Design

The study was a cross-sectional postal survey incorporating a questionnaire based on a written vignette. There were two primary independent variables as between-subject factors, each with two levels, resulting in a 2x2 factorial design. The first independent variable (vignette) was the type of disorder presented in the vignette (PTSD: PTSD comorbid with chronic pain; and No PTSD: chronic pain in the absence of PTSD). The second independent variable was the participants’ status group (member of the public versus professional). Further, nested within the professional group were four categories of health professionals (general practitioner, psychologist, physiotherapist and chiropractor). Profession thus represented a third independent variable to be considered within a separate 2(vignette) x 4 (profession) factorial design.

8.4.3 Materials

The questionnaires were adapted from those used by Jorm and his colleagues to assess mental health literacy (Jorm, Korten, Jacomb, Christensen, & Henderson, 1997; Jorm, Korten, Jacomb, Christensen, Rodgers et al., 1997; Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997; Jorm, Korten, Jacomb, Rodgers, Pollitt et al., 1997; Parker, Chen, Kua, Loh, & Jorm, 2000; Yeo et al., 2001). Each questionnaire (see Appendix J) commenced with the relevant vignette, which described a male who was injured in a motor vehicle accident. Previous studies reported no effect for the sex of the person described in the vignette on questions of mental health literacy (Jorm, Korten, Jacomb, Christensen, & Henderson, 1997) thus only one sex was chosen to reduce sample size requirements. A decision was made to represent a male rather than a female to otherwise reduce sex-based attributions, based on evidence that both chronic pain problems and PTSD may be more prevalent in females. The type of disorder described by the vignette was randomly allocated to an equal number of prospective participants. Approximately half of the participants therefore read about a male, John, who met DSM-IV TR criteria for PTSD in addition to his experience of chronic pain, whereas the
other half read about a male, David, experiencing symptoms of chronic pain only (No PTSD). The vignettes are reproduced in Appendix K.

In response to the vignette, participants were asked to provide a written response to an open-ended question: “What, if anything, do you think is wrong with John/David?” For the person described, participants were then asked to rate their views about the likely frequency and intensity of the person’s pain, whether he needed professional help (yes/no) and whether nineteen types of practitioners or support providers, ten types of medications and nineteen types of treatments/strategies would be of benefit (likely to be helpful, harmful, or neither helpful nor harmful). Some of the wording differed between the questionnaires given to the public and health professionals to cater for differing knowledge of pharmacological terms.

Return to work was chosen as an indicator of prognosis, as its priority is routinely elevated over the presence of symptoms (such as persistent pain or depression/anxiety) by the workers’ compensation and third party compensation systems for those injured in a motor vehicle accident. Participants were thus asked to indicate their expectation as to how soon after the accident the individual would return to work by selecting one of nine timeframes (ranging from the next day to never).

8.4.4 Procedure
Selected participants were initially sent a letter in September 2004 explaining that they had been selected for the postal survey and inviting them to complete the enclosed questionnaire (see Appendix L). Participants were provided with a postage-paid envelope in which to return the completed questionnaire. A reminder letter was sent approximately one month later, indicating that questionnaires were returnable for a further two weeks and could be re-issued if they had been misplaced.

8.4.5 Ethics Approval
The Human Research Ethics Committee of the Australian National University approved the protocol for this study (see Appendix M).

8.4.6 Data Analysis
Analyses were performed according to the nested division of participants. That is, analyses of vignette effects were first considered in combination with participant status.
(member of the public or professional) and then, considering professionals only, in combination with Profession (GP, psychologist, physiotherapist, chiropractor). With respect to frequency data, where more than 20% of expected frequencies fell below 5, variables were recoded, if appropriate, to guard against reduction of power (Tabachnick & Fidell, 2001).

Descriptive statistics and the chi square test were computed for response rates and demographic data. The open-ended responses were coded into problem categories, such that a category was included where it was identified by at least 5% of all participants. These, nominal ratings of the helpfulness of treatment options and expectations regarding pain frequency and return to work were subjected to chi-square analyses and analysis of odds ratios to investigate whether the independent variables influenced these outcomes. Factorial analysis of variance was used to determine whether the presence of PTSD (vignette) and participant status influenced ratings of pain intensity. The alpha level was set at .05 and Bonferroni corrections were made to maintain this level of Type I error in the context of multiple comparisons. Given the survey design, a sequential adjustment was chosen to deal with nonorthogonality in MANOVAs (Tabachnick & Fidell, 2001).

8.5 Results

8.5.1 Response Rates and Demographic Information

Responses were received from 235 members of the public and 101 health professionals, consisting of 47 GPs, 17 psychologists, 25 physiotherapists and 12 chiropractors. This reflected a response rate of 25% for the public and a response rate of 19%, 26%, 42% and 31% for each profession, respectively. Sending a reminder did not substantially increase the response rate.

Of the returned questionnaires, 170 were based on the PTSD vignette (121 public, 49 professional) and 166 were based on the No PTSD vignette (114 public, 52 professional). There was no significant difference between the percentage of PTSD vignettes compared to No PTSD vignettes that were returned by members of the public and each profession ($\chi^2(4) = 1.255, p = .87$).
Overall, 140 males and 196 females responded, with females comprising 59% of the public and 56% of professional participants, respectively. Participants’ mean age was 48.08 years (SD = 13.32; range 17 to 82 years).

Amongst the public, the distribution of the highest educational qualification was: <Year 9 (4%), Year 10-11 (10%), Year 12 (14%), Trade certificate and TAFE qualifications (13%), Undergraduate (22%) and Postgraduate (35%); indicating that the majority of participants held tertiary qualifications. Of the public, 36% indicated that they had been injured in a motor vehicle accident and 39% indicated that they had experienced chronic pain (defined as pain for more than three months) at some time in their lives. There was no significant difference in the frequency of these prior history variables between vignettes ($\chi^2(1) = .327, p = .57$; $\chi^2(1) = .545, p = .46$). Further, 43% of the public indicated that they knew someone like the person in the vignette and 76% indicated that they knew someone with chronic pain. There was no significant difference in these familiarity factors between vignettes ($\chi^2(1) = 3.313, p = .07$).

The extent to which professionals had contact with individuals with chronic pain varied widely, ranging from 1 to 1100 patients in the past year, representing an estimated 1 to 90% of their usual practice.\(^{21}\)

8.5.2 Recognition of Psychological Problems, Physical Injury and Chronic Pain in the Presence and Absence of PTSD

Coding of participants’ open-ended responses to vignettes began with a general categorisation as to whether participants failed to identify any physical problem and then whether they failed to identify any psychological problem. Next, participants’ responses were coded according to their identification of specific problems. The observed percentage frequency of identifying general and specific problem categories is reported in Table 38. The open-ended question permitted identification of multiple problems and 76% of participants mentioned more than one problem category. The modal response was two, with eight being the highest number of categories mentioned ($n=1$). There was no significant relationship between condition and the number of

\(^{21}\) Several professionals added comments that the figures they provided were rough estimates or “guesstimates”, of which they were uncertain or believed were unreliable.
problems identified ($\chi^2(24) = 27.34, p=.29$). Non-parametric statistics were chosen for subsequent analyses because of the non-independence of cell data.

Table 38. Percentage frequency of participants’ identification of problems as a function of vignette and participant status

<table>
<thead>
<tr>
<th>Identified Problem</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>$\chi^2_{condition}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Public (n=121)</td>
<td>Prof. (n=49)</td>
<td>Public (n=114)</td>
</tr>
<tr>
<td>Psychological</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None identified</td>
<td>2%</td>
<td>6%</td>
<td>23%</td>
</tr>
<tr>
<td>PTSD</td>
<td>46%</td>
<td>88%</td>
<td>10%</td>
</tr>
<tr>
<td>Trauma</td>
<td>17%</td>
<td>2%</td>
<td>13%</td>
</tr>
<tr>
<td>Depression</td>
<td>26%</td>
<td>37%</td>
<td>20%</td>
</tr>
<tr>
<td>Anxiety</td>
<td>27%</td>
<td>37%</td>
<td>14%</td>
</tr>
<tr>
<td>Stress</td>
<td>10%</td>
<td>0%</td>
<td>5%</td>
</tr>
<tr>
<td>Psychosomatic</td>
<td>14%</td>
<td>8%</td>
<td>9%</td>
</tr>
<tr>
<td>Non-specific</td>
<td>9%</td>
<td>0%</td>
<td>13%</td>
</tr>
<tr>
<td>Chronic pain</td>
<td>4%</td>
<td>27%</td>
<td>18%</td>
</tr>
<tr>
<td>Pain</td>
<td>7%</td>
<td>0%</td>
<td>5%</td>
</tr>
<tr>
<td>Physical injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None identified</td>
<td>43%</td>
<td>51%</td>
<td>9%</td>
</tr>
<tr>
<td>Whiplash</td>
<td>19%</td>
<td>20%</td>
<td>28%</td>
</tr>
<tr>
<td>Soft-tissue/muscular</td>
<td>17%</td>
<td>22%</td>
<td>29%</td>
</tr>
<tr>
<td>Spinal/nerve</td>
<td>16%</td>
<td>10%</td>
<td>36%</td>
</tr>
<tr>
<td>Non-specific</td>
<td>19%</td>
<td>2%</td>
<td>13%</td>
</tr>
</tbody>
</table>

Note. df=3 for all chi-squared analyses.
*p<.05; **p<.01; ***p<.001.

Across the combined sample of professionals and the public, references to PTSD, trauma, depression and anxiety were common. Some participants described the problem as psychosomatic (i.e. suggested that the physical symptoms were caused by an emotional, not a physical, problem) and only a small percentage identified the individual as suffering from “stress” or a non-specific “psychological problem”. A
range of physical injuries was suggested, including whiplash, soft-tissue/muscle injury, nerve/spinal damage and non-specific “physical injury”. Sixteen participants (5%) believed the individual might have sustained brain injury. Recognition of pain or chronic pain was common but only nine participants (3%) referred to underlying processes of central sensitisation that maintain chronic pain. Only twelve participants, ten of whom were members of the public, reasoned that the individual’s presentation was motivated by compensation and seven (2%) believed there was nothing at all wrong with him.

As can be seen from Table 38, the two factors (participant status and vignette) significantly affected identification of problem categories, except for stress, psychosomatic problem, whiplash and soft-tissue/muscle injury. Rates of problem identification were dependent on whether the participant was a professional or member of the public. That is, a significant main effect was found for participant status for the following problems: PTSD ($\chi^2 (1) = 29.57, p < .001$), trauma ($\chi^2 (1) = 8.23, p < .01$), depression ($\chi^2 (1) = 14.49, p < .001$), stress ($\chi^2 (1) = 4.07, p = .04$), non-specific psychological problem ($\chi^2 (1) = 7.63, p < .01$), chronic pain ($\chi^2 (1) = 61.54, p < .001$), pain ($\chi^2 (1) = 8.66, p < .01$), spinal/nerve damage ($\chi^2 (1) = 4.65, p = .03$) and non-specific physical injury ($\chi^2 (1) = 13.56, p < .001$).

The presence of PTSD also influenced rates of problem identification. Significant main effects were found for vignette for no physical problem identified at all ($\chi^2 (1) = 23.37, p < .001$) and no psychological problem identified at all ($\chi^2 (1) = 22.87, p < .001$), as well as the following specific problems: PTSD ($\chi^2 (1) = 59.59, p < .001$), anxiety ($\chi^2 (1) = 15.02, p < .001$), chronic pain ($\chi^2 (1) = 4.41, p = .04$), soft-tissue/muscular injury ($\chi^2 (1) = 5.33, p = .02$) and spinal/nerve damage ($\chi^2 (1) = 13.36, p < .001$).

Because significant interactions occurred between participant status and vignette, analysis of simple effects is more informative than the main effects. The following sections thus separately consider professionals’ and the public’s responses to each vignette.
Differences in responses to vignettes by professionals

In terms of broad recognition, 94% of professionals recognised some form of psychological disorder in the PTSD vignette, compared to a recognition rate of 89% in the No PTSD vignette. While this suggested that professionals might be more likely to not identify a psychological disorder at all when the vignette described simple chronic pain than when it described PTSD comorbid with chronic pain, this difference was not significant ($\chi^2 (1) = .91, p = .34; \text{OR} = .50, 95\% \text{ CI [.12, 2.12]}$).

With respect to recognition of specific disorders, 88% of professionals correctly identified the individual as having PTSD in the PTSD vignette, with a further 2% identifying “trauma”, but not the specific diagnosis of PTSD. By contrast, in the No PTSD vignette, although there were no diagnostically specific markers of PTSD, 33% identified PTSD and a further 6% referred to the presence of trauma. So, while professionals were fourteen times more likely to identify the diagnosis of PTSD when it was actually present than when it was not ($\chi^2 (1) = 31.72, p < .01; \text{OR} = 14.76, 95\% \text{ CI [5.26, 41.42]}$), these results indicate a substantial false positive rate in the No PTSD condition.

References to anxiety (non-specific) and depression were also common in both vignettes, although different effects for the two disorders were seen across conditions. Not surprisingly, anxiety was five times more likely to be mentioned in the PTSD vignette than the No PTSD vignette (anxiety: $\chi^2 (1) = 10.55, p < .01; \text{OR} = 5.46, 95\% \text{ CI [1.84, 16.23]}$). On the other hand, depression was identified by just over one third of professionals in the PTSD vignette and by half the professionals in the No PTSD vignette. That is, depression was almost half as likely to be mentioned in the presence of PTSD than in its absence, although this difference was not significant (depression: $\chi^2 (1) = 1.81, p = .18; \text{OR} = .58; 95\% \text{ CI [.26, 1.29]}$). As with PTSD, depression was frequently presumed, despite the absence of diagnostically specific markers.

Differences between vignettes were also evident in the level of identification of chronic pain problems and physical injuries. Professionals were 2.5 times less likely to identify chronic pain in the PTSD vignette compared to the No PTSD vignette ($\chi^2 (1) = 4.99, p = .03; \text{OR} = .39, 95\% \text{ CI [.17, .90]}$). Similarly, over half (51%) did not discuss...
any physical injury or pain problem when PTSD was presented in conjunction with a chronic pain problem, whereas less than a third did not mention physical injury or pain when presented with only a chronic pain problem. That is, professionals were nearly three times more likely to not identify any form of physical injury in the PTSD vignette compared to the No PTSD vignette ($\chi^2 (1) = 6.18, p = .02; \text{OR} = 2.83; 95\% \text{CI} [1.23, 6.49]$). Interestingly, the type of vignette had no significant effect on the specific type of physical injuries identified by professionals (whiplash: $\chi^2 (1) = .59, p = .44; \text{OR} = .70; 95\% \text{CI} [.28, 1.76]$; soft-tissue injury/damage: $\chi^2 (1) = .54, p = .46; \text{OR} = .71; 95\% \text{CI} [.29, 1.76]$; spinal/nerve damage: $\chi^2 (1) = 1.63, p = .20; \text{OR} = .48; 95\% \text{CI} [.15, 1.51]$).

8.5.2.2 Differences in responses between GPs, psychologists, physiotherapists and chiropractors

It was not possible to conduct a valid or meaningful statistical analysis of a possible interaction effect between profession and vignette, due to the large number of cells with low expected frequency counts (i.e. <5). Thus, only the main effects of profession (collapsing across vignettes) were subjected to chi-square analysis. Results are only reported for problems where the expected frequencies for cells approached or were greater than 5 (for additional details see Table N.1 in Appendix N).

The likelihood of identifying PTSD was significantly different across professions (minimum E = 4.87, $\chi^2 (3) = 19.71, p < .01$). As would be expected due to each profession’s clinical focus, psychologists were most likely to identify PTSD (88%), closely followed by GPs (70%), with physiotherapists and chiropractors much less likely to identify PTSD (36% and 25%, respectively).

Inspection of the differences in response between vignettes revealed some interesting patterns to qualify this result. In the PTSD vignette, GPs and psychologists demonstrated perfect accuracy in identifying PTSD (100%). Physiotherapists and chiropractors exhibited only moderate recognition of PTSD (67% and 60%, respectively). Although GPs and psychologists were more likely to recognise PTSD than physiotherapists ($\text{OR}_{\text{GP/physio}} = 12.78, 95\% \text{CI} [1.31, 125.07]$; $\text{OR}_{\text{psych/physio}} = 6.11, 95\% \text{CI} [.60, 62.23]$) and chiropractors ($\text{OR}_{\text{GP/chiro}} = 17.25, 95\% \text{CI} [1.42, 210.12]$; $\text{OR}_{\text{psych/chiro}} = 8.25, 95\% \text{CI} [.65, 104.20]$), the sample size limits confidence in these
For the No PTSD vignette, psychologists demonstrated a high level of false positive identification of PTSD (77%). They were more likely to identify PTSD than GPs, who demonstrated a moderate level of false positive identification (44%; OR = 3.18, 95% CI [.52, 19.64]). In turn, GPs were more likely to falsely identify PTSD than physiotherapists and chiropractors, who were relatively unlikely to falsely identify PTSD (8% and 0%, respectively) (OR \(_{\text{GP/physio}} = 9.43\), 95% CI [1.06, 84.04]; OR \(_{\text{GP/chiro}} = 6.40\), 95% CI [.70, 58.52]). The small sample sizes again restrict confidence in these differences.

Disregarding the vignette manipulation, the professions significantly differed in their identification of depression: GPs (57%), psychologists (41%), chiropractors (33%) and physiotherapists (24%) \((\chi^2 (3) = 8.13, p = .04)\). Inspection of the differences in each vignette, suggested that this effect was most prominent in the No PTSD vignette, where physiotherapists raised depression less often (15%) than the other professions (GPs = 68%, psychologists = 57%, chiropractors = 43%). Wide confidence intervals indicate low confidence in these results representing real differences (OR \(_{\text{GP/physio}} = 11.69\), 95% CI [2.08, 65.61]; OR \(_{\text{psych/physio}} = 7.33\), 95% CI [.88, 61.33]; OR \(_{\text{chiro/physio}} = 4.13\), 95% CI [.49, 34.50]).

For the vignettes combined, differences in the frequency of not mentioning a physical problem were significant (minimum \(E = 4.63\), \(\chi^2 (3) = 14.53, p < .01\)). GPs (53%) and psychologists (47%) were more likely than physiotherapists (24%) and chiropractors (0%) to not identify a physical problem. In addition, inspection of responses for each vignette suggested that the presence or absence of PTSD further influenced this result. In the PTSD vignette, more GPs did not identify a physical problem (69%) than psychologists (50%), followed by physiotherapists (42%) and chiropractors (0%). In the No PTSD vignette, psychologists and GPs exhibited similar rates of not identifying a physical problem (43% and 40%, respectively) compared to physiotherapists and chiropractors, who were also similar (8% and 0%, respectively). Finally, the results appear to suggest that GPs, psychologists and physiotherapists were

\[22\text{ According to Smithson (2003, p.30), when calculating confidence intervals for odds ratios, } \]
more likely to not identify a physical problem in the context of PTSD, although the small sample sizes mean again that these differences cannot be stated with confidence.

8.5.2.3 Differences in responses to vignettes by the public

In terms of broad recognition, nearly all of the public recognised some form of psychological disorder in the PTSD vignette (98%), compared to a recognition rate of only 77% in the No PTSD vignette. This reflected that the public were seventeen times less likely to identify the presence of a psychological disorder when the vignette described chronic pain alone compared to when PTSD was also described ($\chi^2 (1) = 25.03, p < .01; OR = 17.58; 95\% CI [4.07, 76.03]$).

With respect to recognition of specific disorders, 46% of the public correctly identified the individual as having PTSD in the PTSD vignette, with a further 17% identifying “trauma”, but not the specific diagnosis of PTSD. This moderate public recognition of trauma following a traumatic injury was much lower than professionals (see above). By contrast, in the No PTSD vignette, 10% falsely identified PTSD and a further 13% referred to the presence of trauma. Thus, the public were seven times more likely to recognise the diagnosis of PTSD when it was actually present than when it was not ($\chi^2 (1) = 37.26, p < .01; OR = 7.80, 95\% CI [3.81, 15.99]$). Mention of trauma did not significantly differ across vignettes ($\chi^2 (1) = .53, p = .47; OR = 1.31, 95\% CI [.63, 2.70]$). Anxiety was twice as likely to be mentioned in the PTSD vignette than the No PTSD vignette ($\chi^2 (1) = 6.23, p = .01; OR = 2.30, 95\% CI [1.18, 4.46]$). On the other hand, depression was equally as likely to be mentioned in both vignettes ($\chi^2 (1) = .98, p = .32; OR = 1.36, 95\% CI [.74, 2.52]$).

Unlike professionals, the public were equally likely to mention pain or chronic pain in both vignettes (pain: ($\chi^2 (1) = .14, p = .71; OR = .84; 95\% CI [.33, 2.14]$; chronic pain: ($\chi^2 (1) = .17, p = .68; OR = .78; 95\% CI [.23, 2.62]$). More importantly, however, their identification of chronic pain disorder was very low (<5%). Forty three percent of the public did not identify a physical injury or pain in the PTSD vignette compared to 18% in the No PTSD vignette, a similar pattern to that shown by professionals. That is, in the presence of PTSD, the public were 3.5 times more likely to not identify a physical injury ($\chi^2 (1) = 17.86, p < .01; OR = 3.54; 95\% CI [1.94, 6.47]$). In particular, the public were significantly less likely to suggest a spinal/nerve damage ($\chi^2 (1) = 12.68, p < .01; OR = .33; 95\% CI [.18, .62]$) or soft-tissue / muscular injury ($\chi^2 (1) = 5.18, p = \ldots$)
The type of vignette had no significant impact on suggestions of whiplash ($\chi^2 (1) = 2.69, p = .10; \text{OR} = .60; 95\% \text{CI} [.33, 1.11]$).

### 8.5.3 Ratings of Pain Characteristics

#### 8.5.3.1 Pain Intensity

Across all participants, collapsed across vignette, status and profession, ratings of the intensity of pain were moderate ($M = 5.424, SD = 1.768$). Mean ratings by professionals and the public for each vignette are presented in Table 39. A two-way analysis of variance indicated a significant interaction between vignette and participant status ($F(1,318) = 4.96, p = .03$). Although the main effect for participant status was significant ($F(1,318) = 6.89, p = .01$), the main effect for vignette was not ($F(1,318) = 3.61, p = .06$). Notably, the power to detect a significant effect for vignette was only moderate (power = .47). The public estimated similar levels of pain intensity, whether PTSD was present or not, whereas professionals estimated higher levels of pain in the context of PTSD than in its absence.

**Table 39. Mean expected pain intensity as a function of vignette and participant status**

<table>
<thead>
<tr>
<th>Participant group</th>
<th>Vignette</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PTSD</td>
<td>No PTSD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$n$</td>
<td>$M$</td>
</tr>
<tr>
<td><strong>Public</strong></td>
<td>5.29</td>
<td>1.76</td>
<td>121</td>
<td>5.22</td>
</tr>
<tr>
<td><strong>Professionals</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GPs</td>
<td>6.25</td>
<td>1.58</td>
<td>49</td>
<td>5.38</td>
</tr>
<tr>
<td>Psychologists</td>
<td>6.05</td>
<td>1.36</td>
<td>22</td>
<td>5.00</td>
</tr>
<tr>
<td>Physiotherapists</td>
<td>5.50</td>
<td>1.65</td>
<td>10</td>
<td>6.00</td>
</tr>
<tr>
<td>Chiropractors</td>
<td>7.25</td>
<td>1.50</td>
<td>12</td>
<td>5.62</td>
</tr>
<tr>
<td></td>
<td>6.20</td>
<td>1.82</td>
<td>5</td>
<td>5.58</td>
</tr>
</tbody>
</table>

Amongst professionals, psychologists estimated greater pain intensity in the No PTSD condition compared to the PTSD condition, whereas the other professions rated greater pain intensity in the PTSD condition compared to the No PTSD condition. Physiotherapists also estimated greater pain intensity than other professions in the PTSD condition. A separate two-way ANOVA revealed, however, that there was no
significant main or interaction effects of vignette and profession (vignette: $F(1,89) = 3.08, p = .08$; profession: $F(1,89) = 1.50, p = .22$; interaction: $F(1,89) = 1.34, p = .27$).

### 8.5.3.2 Pain Frequency

For the pain frequency variable, 58% of cells had an expected frequency count of less than 5 so the variable was recoded such as to combine the two highest and three lowest categories to form two new categories, constant and intermittent or infrequent, respectively. This meant there was a total of four categories of frequency instead of the seven original categories. Five percent of participants did not select any category.

Irrespective of condition, the modal category selected for frequency of pain was constant (50% of all participants), followed by usually present, but with short periods without pain (22%), often present, but with pain-free periods lasting up to several hours (15%) then intermittent or infrequent (9%). Professionals were more likely than the public to believe the pain would be constant for both the PTSD and No PTSD vignettes (63% vs 44% and 67% and 43%, respectively). More members of the public compared to professionals believed the pain would be “often present with pain free periods lasting up to several hours” for both the PTSD and No PTSD vignettes (19% vs 6% and 18% vs 6%, respectively), however, these apparent differences were not statistically significant ($\chi^2(12) = 17.79, p = .12$).

### 8.5.4 Beliefs about Available Providers, Medications and Interventions

Using a three-point scale, participants rated a list of 19 available providers of treatment or support, 8 medications and 19 interventions as either helpful, harmful or neither helpful nor harmful for managing the problems faced by the individual in each vignette. Some respondents wrote “don’t know” or other comments on the side of the questionnaire or left the item blank. Such responses were coded as a separate category, other, during data entry. Table 39 presents the percentage of the public and professionals rating the various interventions as helpful or harmful for the person in each vignette.

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23 The three lowest categories were: Often present, but pain free for much of the day, Occasionally present for brief periods, but not every day, and Rarely present, pain every now and then, with days or weeks in between.
Following the criteria for consensus used by Jorm, Korten, Jacomb, Rodgers and Pollitt (1997; see also Parker et al., 1999), an intervention was classed as *helpful* when at least 66% of a group rated the intervention as helpful and an intervention was classed as *harmful* when at least 66% of a group rated it as harmful. A consensus rating that the intervention was *not helpful* was deemed where less than 25% rated it as helpful.

Table 40. Percentage frequency of the public and all professionals' ratings of providers as helpful and harmful for each vignette

<table>
<thead>
<tr>
<th>Providers</th>
<th>PTSD Public (n = 121)</th>
<th>PTSD Professional (n = 49)</th>
<th>No PTSD Public (n = 114)</th>
<th>No PTSD Professional (n = 52)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>help</td>
<td>harm</td>
<td>help</td>
<td>harm</td>
</tr>
<tr>
<td>General Practitioner</td>
<td>80</td>
<td>0</td>
<td>96</td>
<td>0</td>
</tr>
<tr>
<td>Pharmacist</td>
<td>22</td>
<td>4</td>
<td>31</td>
<td>10</td>
</tr>
<tr>
<td>Massage Therapist</td>
<td>74</td>
<td>3</td>
<td>59</td>
<td>6</td>
</tr>
<tr>
<td>Physiotherapist</td>
<td>69</td>
<td>2</td>
<td>67</td>
<td>4</td>
</tr>
<tr>
<td>Chiropractor</td>
<td>46</td>
<td>17</td>
<td>22</td>
<td>37</td>
</tr>
<tr>
<td>Surgeon</td>
<td>15</td>
<td>26</td>
<td>0</td>
<td>65</td>
</tr>
<tr>
<td>Specialist</td>
<td>55</td>
<td>7</td>
<td>47</td>
<td>10</td>
</tr>
<tr>
<td>Osteopath</td>
<td>42</td>
<td>6</td>
<td>37</td>
<td>16</td>
</tr>
<tr>
<td>Pain Clinic</td>
<td>79</td>
<td>1</td>
<td>92</td>
<td>0</td>
</tr>
<tr>
<td>Counsellor</td>
<td>83</td>
<td>1</td>
<td>90</td>
<td>2</td>
</tr>
<tr>
<td>Social Worker</td>
<td>32</td>
<td>3</td>
<td>45</td>
<td>4</td>
</tr>
<tr>
<td>Telephone Counsellor</td>
<td>39</td>
<td>7</td>
<td>45</td>
<td>4</td>
</tr>
<tr>
<td>Psychiatrist</td>
<td>59</td>
<td>7</td>
<td>74</td>
<td>6</td>
</tr>
<tr>
<td>Psychologist</td>
<td>76</td>
<td>3</td>
<td>98</td>
<td>0</td>
</tr>
<tr>
<td>Mental Health Nurse</td>
<td>44</td>
<td>7</td>
<td>63</td>
<td>0</td>
</tr>
<tr>
<td>Family and Friends</td>
<td>68</td>
<td>3</td>
<td>78</td>
<td>4</td>
</tr>
<tr>
<td>Naturopath</td>
<td>35</td>
<td>6</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>Clergy</td>
<td>32</td>
<td>5</td>
<td>47</td>
<td>2</td>
</tr>
<tr>
<td>Self</td>
<td>13</td>
<td>65</td>
<td>10</td>
<td>65</td>
</tr>
</tbody>
</table>

Note.
1. Colour coding key: achieved at least 66% consensus rating: *helpful*, *harmful*, *neither helpful nor harmful*.  
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8.5.4.1 Providers

8.5.4.1.1 Ratings of providers as helpful, not helpful and harmful. Combined across all conditions, the mean number of providers rated as helpful by participants was 9.16 (SD= 3.44). Overall, pain clinics were most likely to be considered helpful (total = 82%), followed by general practitioners (total = 81%), counsellors (total = 80%), physiotherapists (76%), psychologists (total = 74%) and massage therapists (total = 68%). Trying to deal with the problem on one’s own was most likely to be seen as harmful (total = 60%).

Applying the consensus criteria adopted by Jorm (see above), Table 40 shows that the public agreed that the person in the PTSD vignette would be helped by a counsellor, GP, pain clinic, clinical psychologist, massage therapist, physiotherapist and family or friends. Professionals agreed that a clinical psychologist, GP, pain clinic, counsellor, family or friends, psychiatrist and physiotherapist would help the person with PTSD. None of the providers reached consensus as harmful, as rated by either the public or professionals, although, for both groups, trying to deal with it on one’s own almost reached the consensus criterion. For professionals, a surgeon almost reached the consensus criterion. Both the public and professional groups agreed that consulting a surgeon or trying to deal with it on one’s own would not be helpful. In addition, the public agreed that a pharmacist/chemist would not be helpful and professionals agreed that a chiropractor would not be helpful.

For the No PTSD vignette, the public agreed that a physiotherapist, pain clinic, GP, specialist and counsellor would be helpful to the person, whereas professionals agreed that a pain clinic, physiotherapist, clinical psychologist, GP, counsellor and massage therapist would be helpful. Again, none of the providers were agreed to be harmful by either professionals or the public. The public agreed that trying to deal with the problem on one’s own or seeking help from a person of the clergy/priest/minister, a pharmacist/chemist, a surgeon, a social worker or a telephone counsellor would not be helpful. The professionals agreed that a surgeon, trying to deal with the problem on one’s own, a naturopath and a telephone counsellor would not be helpful.

8.5.4.1.2 Differences in ratings of providers. Subsequent analyses of differences in ratings were limited to the providers that achieved a consensus rating of helpful (see above). The frequency with which a helper was rated as helpful was then compared to
the frequency of all other ratings (i.e. harmful, neither, don’t know or other responses combined). Significant main effects were found for vignette for the following providers: physiotherapists ($\chi^2 (1) = 10.29, p < .01$), clinical psychologists ($\chi^2 (1) = 12.99, p < .01$), psychiatrists ($\chi^2 (1) = 15.38, p < .01$), counsellors ($\chi^2 (1) = 5.79, p = .02$) and family/friends ($\chi^2 (1) = 7.68, p < .01$). Further, significant main effects were found for status for the following providers: GPs ($\chi^2 (1) = 9.62, p < .01$), specialists ($\chi^2 (1) = 7.93, p < .01$); pain clinic ($\chi^2 (1) = 12.24, p < .001$), psychiatrists ($\chi^2 (1) = 6.99, p < .01$) and counsellors ($\chi^2 (1) = 6.63, p = .01$).

General practitioners, pain clinics and massage therapists were just as likely to be considered helpful by participants for the PTSD vignette as in the No PTSD vignette (all $p > .05$). Physiotherapists and chiropractors (76% and 83%) were less likely than GPs and psychologists (100% and 90%) to believe a general practitioner would be helpful. All professions were similar in their likelihood of rating pain clinics as helpful (range 90-100%). GPs and physiotherapists were more likely to believe a massage therapist would be helpful in the No PTSD vignette (84% and 62%) than the PTSD vignette (59% and 33%). Inversely, psychologists and chiropractors were more likely to be believe a masseur would be helpful in the PTSD vignette (43% and 71%) than the No PTSD vignette (70% and 100%).

Both the public and professionals were less likely to rate a physiotherapist as helpful in the PTSD vignette than in the No PTSD vignette (public: OR = .54, 95% CI [.30, .99]; professionals: OR = .22, 95% CI [.07, .66]). Psychologists (59%) were the least likely of the four professions to rate physiotherapists as helpful (the other professions ranged from 83-84%).

Specialists were less likely to be rated as helpful for the PTSD vignette than the No PTSD vignette by members of the public ($\chi^2 (1) = 4.84, p = .03$; OR = .58, 95% CI [.32, .94]), but there was no such effect amongst professionals ($\chi^2 (1) = .08, p = .79$; OR = 1.12, 95% CI [.51, 2.44]). While the percentage frequencies were below consensus cut off (i.e. 66%), each profession was similarly likely to view specialists as helpful (range 40%-58%).
Mental health practitioners were considered more helpful in relation to the PTSD vignette than the No PTSD vignette. The public were 2.5 times more likely to rate psychologists and psychiatrists as helpful in the PTSD vignette (psychologists: $\chi^2 (1) = 11.28, p < .01; \text{OR} = 2.57, 95\% \text{CI} [1.47, 4.48]$; psychiatrists: $\chi^2 (1) = 12.14, p < .01; \text{OR} = 2.53, 95\% \text{CI} [1.49, 4.28]$). Similarly, professionals were twice as likely to rate psychiatrists as helpful in the PTSD vignette than in the No PTSD vignette ($\chi^2 (1) = 4.19, p = .04; \text{OR} = 2.37, 95\% \text{CI} [1.03, 5.48]$). Each profession exhibited similar frequencies of helpful ratings for psychiatrists (58-66%, respectively).

The difference between vignettes was even more prominent in relation to clinical psychologists. That is, professionals were 7.5 times more likely to regard a clinical psychologist as helpful in relation to the PTSD vignette compared to the No PTSD vignette ($\chi^2 (1) = 4.51, p = .03; \text{OR} = 7.48, 95\% \text{CI} [.88, 63.11]$). Although the effect was significant, the confidence interval was large. Professions were similar in their likelihood of rating psychologists as helpful (range 83-100%).

Further, the public were twice as likely to believe a counsellor would be helpful in the PTSD vignette than in the No PTSD vignette ($\chi^2 (1) = 6.46, p = .01; \text{OR} = 2.20, 95\% \text{CI} [1.19, 4.06]$). By contrast, there was no significant difference in professionals’ ratings of counsellors’ helpfulness between vignettes ($\chi^2 (1) = .26, p = .61; \text{OR} = 1.37, 95\% \text{CI} [.40, 4.64]$). Psychologists (71%) were least likely to rate a counsellor as helpful compared to other professions (range 83-94%).

The public were almost twice as likely to believe a family member or friend would be helpful in supporting the person in the PTSD vignette over the No PTSD vignette ($\chi^2 (1) = 6.27, p = .01; \text{OR} = 1.96, 95\% \text{CI} [1.15, 3.33]$), however, professionals rated this group of providers as similarly helpful for both vignettes ($\chi^2 (1) = 1.82, p = .18; \text{OR} = 1.83; 95\% \text{CI} [.76, 4.42]$).

8.5.4.2 Medications

8.5.4.2.1 Ratings of medications as helpful, not helpful and harmful. Collapsing across all conditions, the mean number of medications rated as helpful by individual participants was 3.191 (SD = 1.736). Overall, analgesics were most often considered helpful (69%) and antipsychotics were most often considered harmful (62%).
As shown in Table 41, professionals agreed that antidepressants would be helpful for the person in both vignettes and that analgesics would also be helpful for the person in the No PTSD vignette. By contrast, the public agreed that analgesics would be helpful for the person in both vignettes and that antiinflammatories would also be helpful to the person in the No PTSD vignette. None of the medications reached consensus as harmful in any condition. Consistently for both vignettes, the public agreed that antibiotics, anticonvulsants and antipsychotics would not be helpful to the person, whereas professionals agreed that a greater number of pharmacological interventions would not be helpful (antibiotics, antipsychotics, herbal remedies, sedatives, vitamins and minerals). In addition, the public agreed that anxiolytics would not be helpful for the person in the PTSD vignette and herbal remedies would not be helpful for the person in the No PTSD vignette. Professionals agreed that anticonvulsants would not be helpful for the person in the PTSD vignette.

Table 41. Percentage frequency of the public and professionals’ ratings of medications as helpful and harmful for each vignette

<table>
<thead>
<tr>
<th>Medication</th>
<th>PTSD Public (n = 121)</th>
<th>PTSD Professional (n = 49)</th>
<th>No PTSD Public (n = 114)</th>
<th>No PTSD Professional (n = 52)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>help</td>
<td>harm</td>
<td>help</td>
<td>harm</td>
</tr>
<tr>
<td>Vitamins &amp; minerals</td>
<td>27</td>
<td>0</td>
<td>22</td>
<td>2</td>
</tr>
<tr>
<td>Herbal remedies</td>
<td>31</td>
<td>2</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>Analgesics</td>
<td>69</td>
<td>17</td>
<td>63</td>
<td>12</td>
</tr>
<tr>
<td>Anti-inflammatories</td>
<td>61</td>
<td>14</td>
<td>47</td>
<td>16</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>4</td>
<td>57</td>
<td>25</td>
<td>39</td>
</tr>
<tr>
<td>Antidepressants</td>
<td>48</td>
<td>38</td>
<td>84</td>
<td>4</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>2</td>
<td>45</td>
<td>0</td>
<td>57</td>
</tr>
<tr>
<td>Sedatives</td>
<td>36</td>
<td>43</td>
<td>20</td>
<td>53</td>
</tr>
<tr>
<td>Antipsychotics</td>
<td>7</td>
<td>61</td>
<td>8</td>
<td>57</td>
</tr>
<tr>
<td>Anxiolytics</td>
<td>44</td>
<td>34</td>
<td>65</td>
<td>20</td>
</tr>
</tbody>
</table>

Note. 1. Colour coding key: achieved at least 66% consensus rating: helpful, harmful, neither helpful nor harmful.
8.5.4.2.2 Differences in ratings of medications. Subsequent analyses of differences in ratings were limited to the medications that achieved a consensus rating of helpful. The frequency with which medications were rated as helpful was compared to the combined frequency of all other ratings (as above). The main effect of vignette was significant for anti-inflammatories ($\chi^2 (1) = 4.35, p = .04$) but not for analgesics ($\chi^2 (1) = .85, p = .36$) or antidepressants ($\chi^2 (1) = 1.97, p = .16$). The main effect of status was significant for anti-inflammatories ($\chi^2 (1) = 5.03, p = .03$) and antidepressants ($\chi^2 (1) = 44.72, p < .001$) but not for analgesics ($\chi^2 (1) = 1.09, p = .30$). Thus, anti-inflammatories were 1.5 times less likely to be recommended as helpful in the PTSD vignette than the No PTSD vignette, irrespective of status (OR = .623, 95% CI [.40, .97]). Further, the public were 1.7 times more likely to consider anti-inflammatories helpful than were professionals, irrespective of vignette (OR = .58, 95% CI [.36, .94]). Professionals were six times more likely than the public to regard antidepressants as helpful, irrespective of vignette (OR = 6.23, 95% CI [3.52, 11.02]). The public and professionals considered analgesics similarly helpful between vignettes.

With respect to differences between professions in their beliefs about medications, GPs (68%), chiropractors (58%) and psychologists (53%) were all more likely than physiotherapists (24%) to view anti-inflammatories as helpful. By contrast, beliefs about analgesics were similar across professions (GPs: 75%; physiotherapists: 60%; chiropractors: 58%; psychologists: 53%). The helpfulness of antidepressants was most likely to be acknowledged by GPs (92%), followed by physiotherapists (84%) and psychologists (77%) and least likely to be acknowledged by chiropractors (50%).

8.5.4.3 Non-pharmacological interventions

8.5.4.3.1 Ratings of interventions as helpful, not helpful and harmful. Collapsing across all conditions, the mean number of treatments and strategies rated as helpful by individual participants was 8.74 (SD= 3.11). Overall, relaxation and exercise were most often considered helpful (both total = 87%), followed by physical therapy (total = 80%), counselling (total = 78%) and doing more physical activity (total = 71%). Avoidance of activity was most often seen as harmful (total = 75%).

As shown in Table 42, for the person in the PTSD vignette, relaxation training, regular exercise, physical therapy and counselling were agreed to be helpful by the public. Professionals agreed on more interventions being helpful, nominating regular
Table 42. Percentage frequency of the public and all professionals’ ratings of treatments as helpful and harmful for each vignette

<table>
<thead>
<tr>
<th>Treatment</th>
<th>PTSD Public (n = 121)</th>
<th>PTSD Professional (n = 49)</th>
<th>No PTSD Public (n = 114)</th>
<th>No PTSD Professional (n = 52)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>help</td>
<td>harm</td>
<td>help</td>
<td>harm</td>
</tr>
<tr>
<td>Surgery</td>
<td>8</td>
<td>47</td>
<td>0</td>
<td>94</td>
</tr>
<tr>
<td>Bed Rest</td>
<td>21</td>
<td>35</td>
<td>6</td>
<td>84</td>
</tr>
<tr>
<td>Spinal manipulation</td>
<td>45</td>
<td>24</td>
<td>25</td>
<td>49</td>
</tr>
<tr>
<td>Physical Therapy</td>
<td>82</td>
<td>1</td>
<td>65</td>
<td>6</td>
</tr>
<tr>
<td>Regular ex / gym</td>
<td>84</td>
<td>4</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>Injections</td>
<td>9</td>
<td>36</td>
<td>10</td>
<td>49</td>
</tr>
<tr>
<td>Acupuncture</td>
<td>55</td>
<td>5</td>
<td>57</td>
<td>2</td>
</tr>
<tr>
<td>More phys. activity</td>
<td>64</td>
<td>10</td>
<td>92</td>
<td>0</td>
</tr>
<tr>
<td>Avoid phys. activity</td>
<td>1</td>
<td>71</td>
<td>0</td>
<td>94</td>
</tr>
<tr>
<td>Self-help books</td>
<td>54</td>
<td>3</td>
<td>76</td>
<td>2</td>
</tr>
<tr>
<td>Time off work</td>
<td>31</td>
<td>20</td>
<td>20</td>
<td>55</td>
</tr>
<tr>
<td>Go out more</td>
<td>57</td>
<td>3</td>
<td>82</td>
<td>0</td>
</tr>
<tr>
<td>Courses on relaxing</td>
<td>87</td>
<td>1</td>
<td>98</td>
<td>0</td>
</tr>
<tr>
<td>Cut out alcohol</td>
<td>25</td>
<td>3</td>
<td>35</td>
<td>8</td>
</tr>
<tr>
<td>Occasional alcohol</td>
<td>41</td>
<td>5</td>
<td>33</td>
<td>6</td>
</tr>
<tr>
<td>Counselling</td>
<td>80</td>
<td>2</td>
<td>96</td>
<td>0</td>
</tr>
<tr>
<td>CBT</td>
<td>49</td>
<td>3</td>
<td>86</td>
<td>0</td>
</tr>
<tr>
<td>Psychotherapy</td>
<td>22</td>
<td>10</td>
<td>45</td>
<td>2</td>
</tr>
<tr>
<td>Hypnosis</td>
<td>42</td>
<td>9</td>
<td>49</td>
<td>6</td>
</tr>
</tbody>
</table>

Note:
1. Colour coding key: achieved at least 66% consensus rating: *helpful*, *harmful*, *neither helpful nor harmful*.

exercise, relaxation training, counselling, doing more physical activity, cognitive behavioural therapy, getting out more and reading self-help books as helpful. For the person in the No PTSD vignette, the public agreed that the same interventions, except for counselling, would be helpful as for the PTSD vignette. Professionals also agreed that most of the same interventions would be helpful as for the PTSD vignette, except
that physical therapy was agreed to be helpful and reading self-help books no longer reached the consensus criterion.

Both the public and professionals agreed that avoiding physical activity would be harmful for the person in both vignettes. In addition, professionals agreed that surgery and bed rest would be harmful for the person in both vignettes. The public agreed that surgery, injections, cutting out alcohol altogether and psychodynamic psychotherapy would not be helpful for the person in both vignettes and that bed rest would not be helpful for the person in the PTSD vignette. Professionals agreed that the person in both vignettes would not be helped by injections or taking time off work and that the person in the PTSD vignette would not be helped by spinal manipulation.

8.5.4.3.2 Differences in ratings of interventions. Subsequent analyses of differences in ratings were again limited to only those interventions that achieved consensus ratings of helpful and harmful. The frequency with which an intervention was rated as helpful was then compared to the combined frequency of all other ratings. Similarly, the frequency with which an intervention was rated as harmful was compared to the combined frequency of all other ratings (i.e. helpful, neither, don’t know or other responses).

Of the interventions that reached consensus ratings, the main effect of vignette was only significant for reading self-help books ($\chi^2 (1) = 8.01, p < .01$), counselling ($\chi^2 (1) = 8.37, p < .01$) and CBT ($\chi^2 (1) = 5.21, p = .02$). The main effect of status was significant for the following helpful interventions: regular exercise therapy ($\chi^2 (1) = 19.15, p < .001$), doing more physical activity ($\chi^2 (1) = 36.97, p < .001$), getting out more ($\chi^2 (1) = 23.69, p < .001$), reading self-help books ($\chi^2 (1) = 16.59, p < .001$), relaxation training ($\chi^2 (1) = 6.08, p = .01$), counselling ($\chi^2 (1) = 13.95, p < .001$) and CBT ($\chi^2 (1) = 70.44, p < .001$). The main effect of status was also significant for the harmful interventions of surgery ($\chi^2 (1) = 47.74, p < .001$), bed rest ($\chi^2 (1) = 75.34, p < .001$) and avoidance ($\chi^2 (1) = 17.56, p < .001$).

Examination of the simple effects revealed that professionals were half as likely as the public to view physical therapy as helpful in the PTSD vignette (OR = .42, 95% CI [.20, .88]) but held similar views to the public about the helpfulness of physical therapy.
in the No PTSD vignette (OR = 2.38, 95% CI [.85, 6.65]). Further, professionals were five times more likely to view physical therapy as helpful in the No PTSD vignette compared to the PTSD vignette (OR = .20, 95% CI [.07, .60]). Interestingly, those professions that perform physical therapy (i.e. physiotherapists and chiropractors) were moderately likely to believe in the helpfulness of physical therapy (76% and 75%, respectively), compared to GPs who were more likely to view it as helpful (85%) and psychologists who were less likely to view it as helpful (65%).

The pattern of results was similar for three informal methods of activation (regular exercise, doing more physical activity and going out more), with professionals more likely to favour the method than the public. That is, professionals were nine and fourteen times more likely than the public to recommend exercise as helpful in the PTSD and No PTSD vignettes, respectively (PTSD: OR = 9.71, 95% CI [1.27, 74.41]; No PTSD: OR = 14.33, 95% CI [1.89, 108.88]). This reflected that all GPs, psychologists and chiropractors and 96% of physiotherapists believed exercise was helpful. Professionals were six and seventeen times more likely than the public to believe doing more physical activity would be helpful in the PTSD and No PTSD vignettes, respectively (PTSD: OR = 6.43, 95% CI [2.17, 19.08]; No PTSD: OR = 17.54, 95% CI [4.07, 75.65]). This strategy was strongly supported by each type of profession (all >88%), particularly GPs (96%) and chiropractors (100%). Going out more was at least three times more likely to be rated favourably by professionals compared to the public (PTSD: OR = 3.35, 95% CI [1.49, 7.51]; No PTSD: OR = 4.78, 95% CI [2.07, 11.05]). The simple effects of vignette were not significant for this variable (all $p > .05$). All four professions also had a similar likelihood of rating going out as a helpful strategy (range 75%-85%).

Important differences are also evident with respect to psychologically based interventions. Counselling was five times more likely to be viewed as helpful by professionals than the public for the PTSD vignette (OR = 5.81, 95% CI [1.32, 25.64]). A similar pattern occurred for the No PTSD vignette (OR = 3.48, 95% CI [1.44, 8.41]). Interestingly, professionals view of counselling was similar irrespective of vignette (OR = 3.66, 95% CI [.72, 18.54]), whereas the public were twice as likely to view counselling as helpful when PTSD was present (OR = 2.19, 95% CI [1.21, 3.94]). More GPs viewed counselling as helpful (98%) than other professions (range 82%-88%).
Approximately 30% of physiotherapists and chiropractors did not view counselling as helpful in the No PTSD vignette, yet all viewed it as helpful in the PTSD vignette.

The pattern of results shown for counselling was repeated for CBT. CBT was six times more likely to be supported as helpful by professionals than the public in the PTSD vignette (OR = 6.31, 95% CI [2.63, 15.14]). This difference increased to twenty five times in the No PTSD vignettes (OR = 25.17, 95% CI [9.19, 69.11]). The relative increase difference between vignettes was due to an increased likelihood of CBT being considered helpful by the public in the PTSD vignette compared to the No PTSD vignette (OR = 2.55, 95% CI [1.48, 4.40]) as professionals’ view of CBT was equivalent across vignettes (OR = .64, 95% CI [.19, 2.16]). GPs were most likely to endorse CBT as helpful (98%), compared to psychologists (88%) and physiotherapists (80%), with chiropractors least likely to endorse CBT as helpful (67%).

Professionals were more likely than the public to rate self-help books as helpful within both the PTSD vignette (OR = 2.66, 95% CI [1.26, 5.58]) and the No PTSD vignette (OR = 3.09, 95% CI [1.56, 6.12]). While professionals’ ratings of helpfulness were similar across vignettes (OR = 1.76, 95% CI [.75, 4.20]), the public were twice as likely to view self-help books as helpful in the PTSD vignette compared to the No PTSD vignette (OR = 2.07, 95% CI [1.22, 3.49]). Psychologists (53%) and chiropractors (58%) were less likely to view self-help books as helpful than physiotherapists (68%) and GPs (79%). This difference was caused by fewer psychologists and chiropractors believing self-help would be helpful in the No PTSD vignette (29% and 43%, respectively) compared to the PTSD vignette (70% and 80%, respectively). The only significant difference in beliefs about relaxation training was seen within the PTSD vignette, where professionals were seven times more likely than the public to view relaxation training as helpful (OR = 7.31, 95% CI [.94, 56.76]). Chiropractors (83%) were less likely than the other professions (range 92-98%) to view relaxation as helpful.

Within the PTSD vignette, professionals were 17 times more likely than the public to view surgery as harmful (OR = 17.22, 95% CI [5.08, 58.39]), a difference that dropped, yet remained significant, in the No PTSD vignette (OR = 4.77, 95% CI [2.23, 10.22]). This reflected that professionals were four times more likely to view surgery as harmful in the PTSD vignette compared to the No PTSD vignette (OR = 4.11, 95% CI [267]
whereas the public’s view of surgery was similar, irrespective of vignette (OR = 1.14, 95% CI [.68, 1.91]). All four professions held similar beliefs about the harmfulness of surgery (range 83-92% rated surgery as harmful).

For both vignettes, bed rest was more likely to be viewed as harmful by professionals than the public (PTSD: OR = 78.14, 95% CI [10.39, 587.57]; No PTSD: OR = 11.45, 95% CI [.19, 25.24]). While the public’s view did not change across vignettes, professionals were twelve times more likely to view bed rest as harmful in the PTSD vignette compared to the No PTSD vignette (OR = 12.00, 95% CI [1.49, 96.47]). Psychologists were less likely to rate bed rest as harmful (71%) than the other three professions (range 79%-92%).

Professionals were six times more likely than the public to view avoidance as harmful in the PTSD vignette (OR = 6.24 95% CI [1.82, 21.40]) and three times more likely than the public to view avoidance as harmful in the No PTSD vignette (OR = 3.34, 95% CI [1.38, 8.10]). Professionals’ view of avoidance was similar across vignettes, however (OR = 2.39, 95% CI [.58, 9.80]). Within the professional group, chiropractors (75%) were less likely than the other professions to believe avoidance was harmful (Physiotherapists: 96%; GPs: 92%; Psychologists: 88%).

8.5.5 Ratings of Prognosis

Participants were asked to select the most likely timeframe for return to work. Due to low expected frequencies for several categories of the variable return to work, the category <1 week was recoded so as to include the response next day and a new category (>3 months) was formed by combining the categories <6 months, <12 months, >12 months and never.

A clear majority of respondents expected the individual with chronic pain and PTSD would return to work within the first month of injury. Expectations were differently distributed between professionals and the public, however. One third of professionals expected a return to work within the first week after the accident, 47% within 2 weeks and 59% within 1 month, cumulatively. A further 18% expected return to work to occur within 3 months and 20% expected return to work to take more than 3 months. This represented a bimodal distribution of professional opinion, suggesting that professionals expect either that patients return to work almost immediately or there
will be a delay of approximately 3-6 months. Only 2% of professionals were unsure or did not specify a time for return to work. By contrast, the public expected a more rapid return to work and their responses represented a unimodal distribution. Twenty six percent expected a return to work within the first week after the accident, 54% within 2 weeks, 83% within 1 month and 91% within 3 months, cumulatively. Only 6% believed his return to work would take more than 3 months post injury and 3% were unable to estimate a time frame.

A clear majority of respondents expected the individual with chronic pain only to return to work within the first month of injury. Both groups were more likely to expect a more rapid return here than for the PTSD vignette. Thirty five percent of professionals expected return to work within the first week, 67% within 2 weeks, 85% within 1 month and 90% within 3 months. Six percent believed the individual would take more than 3 months to return to work and 4% did not specify a time frame. Therefore, by contrast to the PTSD vignette, the cumulative frequency distribution of return to work estimates was more even and reflected that professionals were more likely to expect a faster return than for the PTSD vignette. Curiously, the public’s expected time frame for return to work was typically longer than professionals for the No PTSD vignette, by contrast to the shorter time frame relative to professionals for the PTSD vignette. Thirty two percent of the public expected return to work within the first week, 55% within 2 weeks, 74% within 1 month and 88% within 3 months. Only 6% expected return to work would occur after more than 3 months and 6% were unsure of a time frame.

8.6 Discussion

The present study examined knowledge and beliefs about PTSD and chronic pain in the context of a physical injury sustained in a motor vehicle accident. The first aim was to determine whether there would be any difference in professionals’ and the public’s responses to an individual when his presentation involved chronic pain as opposed to psychological trauma and chronic pain. In this regard, it was of interest whether there would be differences in participants’ ability to identify the presenting problem and, further, whether there would be differences in their beliefs about treatment and prognosis for these presentations. Second, the study sought to determine whether the public, professionals and specific professions differ from each other in their knowledge and beliefs.
Despite standard healing rates and the breadth of evidence illustrating low correlations between physical injury, chronic pain and disability, professionals were still likely to adopt a structural/medical approach when interpreting the presenting problem in the vignette. That is, almost half of all professionals responding to the PTSD vignette and nearly three quarters of all professionals responding to the No PTSD vignette referred to the presence or possible presence of a physical injury to explain pain that had persisted for more than six months after a motor vehicle accident. True to their professions, chiropractors always diagnosed a physical injury and physiotherapists were inclined to do the same, especially in the absence of PTSD. Despite their broader professional orientation, a majority of GPs and psychologists (approximately 60%) also suspected physical injury in the absence of PTSD. While the likelihood of physical injury dropped in the presence of PTSD, injury was still considered likely by a significant minority of these two professional groups.

By contrast, only one quarter of all professionals responding to the PTSD vignette and half of all professionals responding to the No PTSD vignette referred to the presence of a chronic pain problem or disorder. Professionals were therefore generally poor at recognising chronic pain disorder, with 52% and 73% missing this problem altogether, whether in the respective absence or presence of PTSD. When PTSD was not present, physiotherapists followed by psychologists were most adept at recognising chronic pain, with GPs poorest at identifying this problem. Psychologists’ view of chronic pain was not influenced by comorbid PTSD, whereas GPs and both physical-based professions’ demonstrated a low rate of detection of chronic pain under conditions of comorbidity. This approach indicates a significant divergence in clinical understanding from the empirical literature regarding chronic pain problems and raises the need for further education of clinicians.

Prior research has also indicated that primary care practitioners fail to diagnose mental disorders in up to half of their actual patients (Borus et al., 1988; D. Goldberg et al., 1982; Higgins, 1994; Ormel et al., 1991). Other research has revealed that practitioners may be reasonably sensitive to psychological distress, anxiety and depression, but less sensitive to specific diagnostic categories, such as PTSD (Samson et al., 1999). Indeed, the detection of PTSD has been shown to range from an extremely
poor 2.4% in a natural sample (Taubman-Ben-Ari et al., 2001) to a moderate 67% in a vignette study (Munro et al., 2004).

In the current study, professionals demonstrated a high sensitivity for detecting PTSD, with 88% accurately identifying the disorder as presented in the vignette. GPs and psychologists demonstrated an impressive 100% accuracy, with physiotherapists demonstrating less awareness and chiropractors demonstrating poor awareness of the disorder. This is not surprising given their likely educational training and professional approach. Further, there was good specificity when PTSD was actually present to the extent that PTSD was more frequently identified than the more generic categories of anxiety and depression. Specificity was less sound, however, when one considers that 77% of psychologists and 44% of GPs incorrectly suggested the disorder in the absence of diagnostic symptoms. Despite insufficient diagnostic symptoms, depression was also twice as likely to be identified in the simple presentation of chronic pain than when there was comorbid PTSD. This is interesting in the face of empirical literature, which shows similarly high levels of comorbidity between both chronic pain and depression, and PTSD and depression.

Arguably, false positive diagnosis of PTSD is less of a concern during screening as this reduces the risk of not providing psychological treatment when it is actually appropriate. On the other hand, the effectiveness of referral and treatment might be compromised when one considers the impact of the presence of PTSD on the identification of chronic pain and physical injury in this study. Only a quarter of professionals discussed chronic pain in the presence of comorbid PTSD, compared to half of all professionals identifying chronic pain in its simple presentation. Similarly, half of all professionals did not identify a physical injury when PTSD was present, compared to only a quarter in the face of simple chronic pain. That is, professionals were at least twice as likely to not identify chronic pain and three times more likely to not identify a physical problem in the presence of PTSD than in its absence.

These findings are striking for two reasons. First, this lack of correspondence between vignettes occurred even though the vignettes provided equivalent descriptions of pain, physical symptoms and physical disability. Why, then, were they viewed differently? Second, the lower rate of identifying chronic pain disorder is in apparent contradiction with the finding that professionals rated the individual’s pain as higher
under circumstances of comorbid PTSD (and previous research findings of higher pain severity in this group). That is, when PTSD was present, professionals seemed to recognise chronic pain disorder less often, grant it lower diagnostic priority and or consider it secondary to psychological distress. If true, this could influence decisions as to when and whether chronic pain is treated and may mean less consideration is given to addressing the most common processes that maintain chronic pain, such as catastrophising, inactivity, avoidance and neurophysiological processes such as central sensitisation, for example. This single diagnosis focus may be an artefact of a health system that encourages health practitioners to focus on only one disorder at a time.

Unfortunately, the present results do not provide insight into professionals’ reasoning so this poses an avenue for future exploration. Nonetheless, these findings do not bode well for prognosis, particularly if the relationship between chronic pain and PTSD is one of mutual maintenance or of shared vulnerability.

A different pattern of results emerged from analysis of public opinion. Although almost all the public (98%) identified some form of psychological disorder when symptoms of PTSD were comorbid with chronic pain, only a minority specifically referred to PTSD (46%). This is only slightly higher than the rate of public detection of depression (39%) and schizophrenia (27%) shown in prior studies (Jorm, 2000). A significant minority used non-specific terms, such as trauma, anxiety, stress and “psychological problem”, suggesting that the public’s knowledge of specific psychiatric terms is poorly developed. As Jorm et al. (1997) discussed, it is not immediately clear whether lack of knowledge about technical terms poses a real problem, however, it is possible that it might where adequate assessment and treatment is not readily accessible.

The presence of PTSD symptoms influenced the public’s identification of both psychological and physical problems. The public were seventeen times more likely to not identify a psychological problem in the absence of PTSD. That is, one quarter of the public did not recognise psychological problems when the individual was described as having only simple chronic pain. This suggests the public have a poor awareness of the contribution of psychological factors to chronic pain, which may impede understanding, satisfaction and compliance with appropriate, evidence-based treatment recommendations.
In terms of treatment options and recommendations, according to Section 8.1.1, evidence-based practice for chronic pain would require endorsement of a multidisciplinary pain clinic, emphasising cognitive behavioural therapy, exercise and physical activation rather than passive treatments, avoidance and bed rest. Analgesia, low dose tricyclic antidepressants and anti-convulsants may also be appropriate. From Section 8.1.2, evidence-based practice for addressing PTSD should promote cognitive-behavioural therapy, possibly with adjunctive pharmacotherapy involving antidepressants and anticonvulsants. In the absence of best-practice treatment for comorbid PTSD and chronic pain, it would be reasonable to expect adherence to, at least, guidelines for the independent disorders. The present study suggests that, in practice, this is not currently the case.

Applying Jorm et al.’s (1997) criteria for consensus to the current data, it was possible to consider the public and professionals’ views of treatment options and differences between these views in the present study. While some beliefs matched empirical evidence and clinical guidelines, there were several noteworthy discrepancies. This was not unexpected in the case of the public, who may have very little access to specific knowledge about the nature and efficacy of medications and therapies. Indeed, strong selection of the ‘neither’ category for some medications and treatments quite possibly reflects the public’s lack of knowledge rather than a true decision regarding the effectiveness of the intervention.

Helpful providers (based on consensus criteria applied to all participants) included pain clinics, GPs, physiotherapists, massage therapists, counsellors and clinical psychologists. Endorsement of physiotherapists and massage therapists by both the public and professionals was contrary to clinical guidelines, given the individual had experienced pain for more than six months. It is also interesting to note that physiotherapists and specialists were more likely to be considered helpful in the simple presentation of chronic pain, even though physical features were equivalent across vignettes.

Despite strong empirical evidence of the contribution of psychological factors in chronic pain, psychologists were less likely to be viewed as helpful in managing the simple presentation of chronic pain than chronic pain with comorbid PTSD, by both the
public and professionals. This indicates that greater education of the clinical and general community is required with respect to the management of chronic pain.

Professionals were generally more likely than the public to believe counsellors and psychologists would be helpful. By contrast, the public were more likely to endorse the helpfulness of counsellors rather than clinical psychologists. These findings may reflect a lack of awareness about the professional distinction between clinical psychologists and counsellors. In combination with the generally low regard for all psychologically-based providers, however, it appears that the public may be poorly informed about these providers or could be sceptical or suspicious of their care.

The public’s suspicion of antidepressant medication, as highlighted in prior research (Jorm et al., 1997), was again borne out in the present study. That is, while antidepressant medication is considered a standard and effective intervention for both PTSD and management of chronic pain, the public did not reach consensus about the helpfulness of such medication, whether or not PTSD was present. Indeed, professionals were six times more likely than the public to recommend use of antidepressants. Again it is not clear from the results whether the public’s opinion was based here on a simple lack of knowledge about medications or on erroneous beliefs about them. In either case, this difference may have implications for compliance with recommended treatment.

Anticonvulsant medication was also not recognised as helpful despite evidence of its utility in the empirical literature and clinical guidelines, for both chronic pain and PTSD. The public’s consensus was that anticonvulsants would not be helpful, irrespective of the presence of PTSD, and professionals’ consensus was that it would not be helpful in managing chronic pain comorbid with PTSD. Again, this result is likely to reflect the public’s limited access to knowledge about or experience with anticonvulsants. Professionals’ views about anticonvulsants were also contrary to expectation, as, based on the literature, such medication is (arguably) doubly indicated when PTSD is comorbid with chronic pain. Moreover, anticonvulsants are now recommended as a primary treatment for chronic pain, yet one quarter to two fifths of professionals rated them as harmful. It is therefore likely that not only the public but professionals require education about effective medications. As pointed out by an anonymous reviewer, it is possible that different results might have been achieved by
use of the term "mood stabiliser", by which anticonvulsant medication is also known. On the other hand, use of that term might confound one of the purposes of the study; that is, to assess knowledge of effective treatments for chronic pain, not just associated psychological sequelae or comorbid psychological disorder.

Helpful interventions (based on consensus criteria applied to all participants) included relaxation training, regular exercise, physical therapy, counselling and doing more physical activity. There was professional consensus that avoidance, surgery and bed rest would be harmful and public consensus that avoidance would be harmful and surgery would not be helpful. The majority of public and professional opinion thus generally reflected clinical guidelines, except for the inclusion of physical therapy and counselling. The public was significantly less likely than professionals to believe methods of physical activation would be helpful and the majority only viewed bed rest as not helpful when the presentation included PTSD. This reflects that a significant minority of the public have a poor understanding of treatment for chronic pain, which may again interfere with the nature of treatment sought and adherence with treatment.

Physical therapy, as a passive, provider-based intervention, is contra-indicated according to clinical guidelines. Despite this, the majority of professional and public opinion showed similar error in believing physical therapy would be helpful to treat simple chronic pain. The presence of PTSD also decreased the likelihood of professionals rating physical therapy as helpful. While this difference could be attributed to greater knowledge of best-practice for this condition, it is more likely to reflect that practitioners view (and thus treat) pain differently (arguably, less seriously) when it is accompanied by PTSD. This conclusion is supported by the increased likelihood of professionals viewing surgery and bedrest as harmful when there is comorbid PTSD, despite there being no differences in the physical presentation.

Several important points arise with respect to beliefs about psychologically-based treatment. First, professionals were between 5 and 25 times more likely than the public to endorse such intervention, whether in the form of counselling or cognitive-behavioural therapy and irrespective of presentation. Indeed, professionals as a whole demonstrated excellent knowledge of the effectiveness of CBT in treating simple chronic pain and chronic pain comorbid with PTSD, with at least 85% rating CBT as helpful in these circumstances. By contrast, less than half of the public viewed CBT as
helpful to treat chronic pain and PTSD and only one quarter viewed CBT as helpful to treat chronic pain. The public thus demonstrated a moderate to low regard for the utility of CBT. This could be due to a lack of knowledge of the technical term, CBT. More seriously, it could reflect poor understanding or devaluing of psychological therapy generally, particularly with respect to the simple presentation of chronic pain. That is, the public are more likely to view chronic pain in physical terms, requiring physically-based treatment. This conclusion is supported by the public’s reduced likelihood of rating counselling and self-help books as helpful for treatment of simple chronic pain relative to chronic pain with comorbid PTSD.

Professionals were equally likely to regard counselling and CBT as helpful in treating chronic pain and were more likely to regard it as helpful than CBT in treating PTSD. This highlights that professionals demonstrate a high level of awareness of the potential benefit of psychologically-based treatments but suggests they have poor knowledge about the specific effectiveness of different treatments. Not surprisingly, public opinion mirrored professional opinion on this point. Counselling was more likely than CBT to be endorsed by the public as helpful, although the public were again less likely to endorse counselling for simple chronic pain. Clearly, these findings have implications for appropriate referral by professionals and treatment-seeking by the public.

Given that several disciplines are often involved in the treatment of individuals with chronic pain with or without PTSD, it is important to understand if and where professions differ in their beliefs about treatment as conflicts may arise where variation occurs. In many cases, the four professions displayed similar beliefs about treatment, however, there were some important differences in opinion. Unlike previous studies that have shown that professionals are most likely to endorse their own profession and treatments as helpful, this was not always the case in this study. GPs were most likely of the four professions to endorse GPs and antidepressants as helpful. Interestingly, they were also most likely of all the professions to endorse physical therapy, CBT and counselling. That is, those who deliver physical therapy (physiotherapists and chiropractors) were only moderately likely to believe their own intervention would be helpful. Psychologists were also surprisingly less likely than GPs to identify CBT as helpful (but more likely than physiotherapists and chiropractors). Psychologists’ lower likelihood of rating counselling as helpful as compared to other professions may arise
from professional bias, however, is arguably consistent with empirical evidence supporting CBT over supportive counselling. Psychologists were the least likely of the professions to rate physiotherapists or physical therapy as helpful and least likely to believe bedrest would be harmful. Chiropractors were the least likely of the professions to believe in the utility of psychologically based treatments, including antidepressants, CBT, counselling, self-help books and relaxation training and were most likely to endorse passive treatments, such as acupuncture and spinal manipulation (cf. Ferrari & Russell, 2004). Massage was viewed differently, dependent on both profession and the presenting problem. While more GPs and physiotherapists believed massage would be helpful for simple chronic pain than for chronic pain with comorbid PTSD, psychologists and chiropractors were more likely to believe the reverse. Whilst these inconsistencies may warrant further education of professions, these findings may not generalise beyond the current study.

8.6.1 Limitations

The limitations of this study must be acknowledged before drawing conclusions from the above results. The response rate was reasonable to the extent that it was comparable to the typical return rate of 20-30% for mailed survey studies (Christensen, 1994). Nonetheless, the rates are still low and there may be problems in generalising the results to the general population and professional community.

One concern is that participation rates and responses were influenced by whether the prospective participant had experienced or were familiar with the circumstances represented in the vignette. In this study, the reported rates of chronic pain and of personal injury due to a motor vehicle accident were 39% and 36%, respectively. These figures are higher than rates suggested by prevalence data. That is, the prevalence of chronic pain in Australia was estimated four years ago at around 20% (Blyth et al., 2001). Although the rate of incidence of injury due to motor vehicle accidents is available for the ACT, the overall prevalence is not. Data released in the ACT Road Safety Plan 2005-2006 (Department of Transport, 2005) indicated that 387 people from 8288 ‘on-road’ recorded crashes in 2003 required medical treatment, with 138 of those admitted to hospital (from a population of approximately 300,000 in the ACT). Preliminary figures from 2004 suggest a further decline with 350 people requiring medical treatment, 125 of which were admitted to hospital. These figures had approximately halved since the introduction of the ACT Road Safety Strategy in 1999.
The report also described data from a study commissioned to examine the number of ACT residents involved in road crashes in the neighbouring state of NSW (which surrounds the territory). This revealed that an additional 543 ACT residents sustained injury in NSW road crashes during the period 1999-2003. Due to conditions of anonymity and financial constraints, it was not possible to follow-up non-responders. Therefore, it is not possible to clearly determine whether or how such personal experience (or other demographic factors) influenced participation.

Educational background may indeed have influenced participation and thus results. Members of the public with postgraduate qualifications were disproportionately represented amongst those who responded to the survey. The reason for this is not clear. Possible explanations are that those with lower levels of education found the questionnaire too difficult or perhaps people with postgraduate qualifications were more likely to value and thus participate in the research. Regardless, the great potential for educational background to influence beliefs suggests further caution in interpreting the results.

The response rates for the current study were also much lower than those reported by Jorm, Korten, Jacomb, Christensen & Henderson (1999) (ie. 61-75% for professionals and 85% for the public)²⁴, raising questions about drawing direct comparisons between their results and those presented above. Reminder letters were sent, however, these did not appear to have a significant impact on increasing the response rate and it may have been better to include a copy of the questionnaire with the reminder (Caldwell & Jorm, 2000).

The number of respondents from each profession was also small. Partly this was a problem of low response rates, however, in fairness, it also reflected actual population sizes (e.g., only 38 chiropractors practice in the ACT). Regardless, the small numbers elevated the risk of making Type II errors in formal statistical analyses of differences between professional groups. It is possible that those professionals who responded were a biased group, perhaps with a specific interest in the area of chronic pain and or PTSD.

²⁴ Although the content of the survey in the present study was very similar to that used by Jorm et al., (1997), public response rates are not immediately comparable as Jorm’s study involved face-to-face
Using a vignette-based survey has the benefit of controlling patient variables (such as style of reporting) and reducing biases that may arise from the existing relationship a real patient has with a participant. Nonetheless, it also has its limitations (Jorm, Korten, Jacomb, Rodgers, & Pollitt, 1997; Parker et al., 2000). The questionnaire used here was designed with the public in mind and was thus less suited to the requirements of clinical decision-making. Responses were briddled by the simplified case information and quite a few professional respondents commented that there was too little information to inform a clinical opinion. Participants were also required to make forced-choice decisions about interventions for a prototypical case at one point in time, without reference to the specific nuances that occur in the normal evolution of a case, which might affect one’s judgment. Because treatment components were not described in detail, it is possible that participants implied different interpretations of what was meant. In addition, the present study did not allow for sequential or hierarchical ordering of treatment interventions (Parker et al., 2000). Where respondents found the rating format unsuitable, they had few options other than writing a comment on the questionnaire. Ratings were sometimes qualified thus by a written comment indicating that any judgment about intervention would depend on the particular skills or approach adopted by the specific helper, the level of expertise in delivering the intervention and the subjective indications or contraindications for the individual to be treated. It would be useful to address these issues in a questionnaire specifically designed for health professionals and based on more detailed clinical information.

In the present study, the choices available for judging interventions were helpful and harmful, with a residual choice of neither helpful nor harmful. An additional residual category of don’t know/other was added during coding due to the high number of comments and blank responses, however, it could not be considered independently during analysis as it was not an explicit option for all participants. The frequency for rating interventions as helpful was reported in contrast to all three alternatives as this method permitted conclusions about consensus. As raised by Parker et al., (1999), it would be prudent to consider the degree to which residual categories are endorsed, however. The reliability of considering only the helpful: harmful ratio is reduced, of course, if residual categories are strongly endorsed.

__interviews conducted by ABS staff rather than a mailed-out survey.__
Finally, it is not clear how closely people’s responses to a hypothetical scenario match their actual behaviour in clinical practice and daily life. As discussed above, general practitioners’ detection of mental disorders is influenced by the way in which their patients present; that is, they are more likely to detect a mental disorder if the individual explicitly raises relevant symptoms and discusses them as a psychological problem. Thus, while it is commendable that most professionals accurately detected PTSD in the current study, because the vignette provided an explicit and coherent report of criterion symptoms it is questionable whether such high detection sensitivity would extend beyond the “model” patient used here. Priming also may have occurred as participants were aware that the survey originated from the ANU School of Psychology.

8.6.2 Conclusion and Future Directions

The above findings indicate that the public and professionals have at times incongruous beliefs regarding the nature of pain and associated psychological problems and the utility of various treatments, which vary further according to the presence of a comorbid psychological disorder. These disparate understandings may have consequences similar to the effects of miscommunication and competing explanatory models of pain on the doctor-patient relationship (Kouyanou, Pither, & Wessely, 2001). Where patients’ knowledge and beliefs differ from their health providers, they may feel disbelieved, judged and frustrated, leading to suspicion and dissatisfaction with appropriate advice, non-compliance with appropriate treatment and or inappropriate treatment-seeking. Furthermore, a lack of congruence between beliefs and clinical guidelines was demonstrated not only for the public but also for professionals. These iatrogenic factors may increase the risk of poor outcomes when treatment failures compound presenting problems, further reinforcing patients’ sense of hopelessness.

As much as this survey data provides insight into ‘what’ beliefs the professional and community hold regarding treatment of PTSD and chronic pain, it merely draws attention to the need for closer investigation as to ‘why’ these beliefs are held. Future research needs to be directed to the reasoning behind the beliefs expounded in the present study to enable greater understanding of the clinical implications of such beliefs. If not informed by an understanding of the rationale behind them, attempts to address problematic beliefs would lack feasibility. Quite distinct approaches would be required depending on whether differences result, for example, from a lack of knowledge, from personal or professional experience, or from personal or professional prejudices.
CHAPTER 9

GENERAL DISCUSSION

An implied relationship between PTSD and chronic pain has attracted growing interest due to the complex presentation and poor outcomes seen in individuals who experience these disorders comorbidly. Despite this, very little is know about the mechanisms that underlie the apparent links, not least because research has been relatively atheoretical, ad hoc and subject to methodological limitations. The current program of research, guided by recent theoretical models (i.e., Sharp & Harvey, 2001; Asmundson et al., 2002), explored the relationship over five studies.

9.1 Summary of findings

9.1.1 Study 1

Using a sample of 120 individuals, the aim of Study 1 was to determine whether patients with heterogenous chronic pain problems would differ in terms of anxiety-related variables, pain-specific coping strategies and indicators of adjustment, on the basis of PTSD classification. This classification was based on the PDS, a self-report questionnaire, and the two groups were found to be similar in age, gender and prior number of traumatic events. Results indicated a non-significant trend toward higher pain severity in the PTSD group compared to the No PTSD group. By contrast to individuals without PTSD, those with PTSD demonstrated higher anxiety sensitivity and pain-related anxiety and more frequently engaged maladaptive cognitive (catastrophizing, praying/hoping, fewer coping self-statements) and behavioural (resting and guarding) coping strategies. Further, they demonstrated poorer adjustment to pain, including more severe depression and perceived pain-related disability, as well as exhibiting higher rates of utilising health care providers and medication. They were also more likely to be pessimistic about their prognosis, less likely to be working and more likely to be seeking compensation than their non-traumatised counterparts. A significant but small proportion of variance in pain severity (20%) and greater proportions of pain-related disability (52%) and depression (65%) could be explained by the self-report measures using multiple regression analyses. Keeping in mind that this study was cross-sectional, it provides some support for the role of anxiety sensitivity as espoused by Asmundson et al. (2002). Nonetheless, the effects of PTSD
were significant, even after controlling for anxiety sensitivity, suggesting that anxiety sensitivity is not sufficient to explain the apparent relationship between pain and PTSD.

### 9.1.2 Study 2

Improving the methodology of Study 1, Study 2 more clearly differentiated PTSD diagnosis, using both the PDS and PTSD-module of the SCID to assess for current injury-related PTSD amongst 29 chronic pain patients. The formal diagnostic interview permitted detection of lifetime PTSD, which was significantly present in both groups, but did independently have an effect on dependent measures. Affective and psychophysiological reactivity during interview was also assessed. Baseline SC was moderately correlated with PTSD severity, providing support that PTSD results in general baseline hyperarousal. Both groups recorded a similar elevation in physiological reactivity during the SCID. This was not considered fatal to diagnosis as the description was given during diagnostic interview rather than under strictly controlled experimental condition (cf. Orr et al., 1993).

The aim of Study 2 was three-fold: (1) to examine the effect of PTSD on fear of movement/reinjury; (2); to replicate the findings of Study 1 regarding the effect of PTSD, self-reported variables and physiological reactivity on indicators of adjustment to chronic pain; and (3) to explore factors related to physiological reactivity during imaginal exposure to activity (using the PHODA). Results broadly replicated Study 1 regarding the effects of PTSD on pain severity, distress (DASS), disability, compensation status and pain-related anxiety. Consistent with prediction, the PTSD group also demonstrated higher fear of movement/reinjury, regarding general and specific movements. These measures were not significantly correlated with physiological reactivity experienced whilst describing the traumatic injury during the SCID, contrary to theoretical prediction that fear of movement/reinjury would be associated with the trauma network (Sharp & Harvey, 2001). Also contrary to this prediction was the finding that PTSD severity was not associated with physiological reactivity experienced during imaginal exposure to feared movements. By contrast, fear of movement/reinjury and pain-related anxiety showed a significant negative correlation with heart rate and a positive correlation with respiration during imaginal exposure.

Baseline and one-month pain ratings were not significantly correlated with any physiological measures. Further, multiple hierarchical regression analysis indicated that
53% of the variance in pain severity could be accounted for by a model including PASS, TSK, PHODA Fear, physiological correlates, PTSD diagnosis and PTSD severity, although only the PHODA Fear rating made a unique contribution to explaining the variance, even though PTSD variables were assigned priority. This is contrary to theoretical predictions that PTSD impacts on pain via hyperarousal (cf. Sharp & Harvey, 2001). The same model, with the addition of pain severity also explained 80% of the variance in pain-disability, but again only pain severity and PHODA Fear rating made a unique contribution to the variance, again after controlling for PTSD. A different pattern of results emerged when the original model was used to predict distress. This time PTSD diagnosis and severity, TSK and PASS, in order, all resulted in a significant increment in the accuracy of prediction (which accounted for 80% of variance). Interestingly, the PTSD group were more inclined to be taking antidepressants, although the reason for prescription was not clear. If SSRIs, which are recommended in the treatment of PTSD, this may have attenuated differences between groups that would otherwise have been expected in the context of PTSD. Alternatively, given the neurolytic effects of tricyclics and their synergistic effect on analgesia, this finding might explain the lack of difference on pain severity.

Here, the differential accuracy of prediction across the multiple regression analyses (see also Study 1), the lack of physiological reactivity to imaginal stimuli, and the lack of association between physiological arousal and anxiety/fear measures (irrespective of PTSD) suggests that the relationship between PTSD and chronic pain operates most strongly through the third causal pathway espoused in Chapter 4 – relating to bidirectional affective and behavioural responses – rather than either of the unidirectional modes specific to pain (i.e., PTSD hyperarousal elevates pain) or fear (i.e. activation of fear network by pain). Of course, the effect of a third variable (i.e. the fourth causal pathway; see also Asmundson et al., 2002) cannot be ruled out on the basis of this study.

9.1.3 Studies 3 and 4

The next two studies involved a systematic experimental manipulation to further test the prediction that PTSD directly exacerbates pain perception in chronic pain patients. More specifically, the studies employed the same essential design, to examine whether PTSD influences pain experiences generally and or specifically (via changes in pain, negative affect and physiological responses during exposure to individualised
trauma-related scripts) under controlled conditions of painful movement (Study 3) or with respect to global pain threshold (Study 4). In both cases, the task was thus performed under standard conditions and repeated post-exposure.

Painful movement was operationalised in Study 3 using an assessment of ROM. Under standard conditions, the PTSD group demonstrated greater restriction of movement than the No PTSD group for two of ten specific movements: cervical and lumbar flexion during forward bend. The magnitude of ROM was significantly correlated with pain severity, PTSD severity, pain-related anxiety and fear of movement/reinjury, such that more restricted ROM was seen in participants with higher scores on these measures. Further, when ROM was re-assessed post-exposure, the PTSD group demonstrated a significantly greater percentage change in ROM from the standard condition than the No PTSD group. Bivariate correlations indicated that greater deficits following exposure were significantly correlated with PTSD severity, negative affective response to exposure, pain-related anxiety and fear of movement/reinjury. The consistency between experimentally induced disability and global ratings of perceived disability, increased confidence in the validity of the task.

Individuals with PTSD reported consistently higher levels of pain, tension and negative affect than without PTSD, irrespective of the task. Nonetheless, assessment of ROM evoked parallel increases in pain across groups, and exposure had no significant effect on pain levels. The converse pattern occurred for negative affect, with exposure but not ROM leading to higher levels, irrespective of group. There was also a non-significant trend for tension to be increased, irrespective of group, by both ROM and exposure, suggesting the perception of tension may include sensory and affective components. Thus, the results again did not support the two unidirectional pathways proposed in Chapter 4, according to theoretical models. First, there was no significant interaction effect of PTSD status and exposure on physiological reactivity, undermining claims that hyperarousal underpins the apparent relationship between chronic pain and PTSD. Second, there was no clear evidence supporting activation of a fear network by pain. Instead the available evidence pointed again to the third causal pathway involving the impact of bi-directional interactions between PTSD and pain occurring over a chronic course.
Global pain threshold was assessed via the application of a thermode to an inner forearm unaffected by pain in Study 4. Contrary to hypotheses, the two groups (PTSD: \( n = 11 \); no PTSD: \( n = 9 \)) demonstrated equivalent thermal pain thresholds (TPT), under standard conditions and post-exposure. This result suggested that PTSD did not affect pain threshold either in its capacity as a general disorder or under circumstances of direct symptom activation. On the other hand, ratings of usual pain and negative affect increased during post-exposure TPT, more so in the group with PTSD, however, these changes did not predict changes in TPT. More generally, lower pain threshold was associated, across the entire sample, with lower systolic blood pressure, currently high affect, and higher scores for PTSD avoidance and numbing, pain-related anxiety and pain-related disability. Hypervigilance, albeit self-reported, was not correlated with pain threshold.

9.1.4 Study 5

This study was a cross-sectional postal survey incorporating a vignette-style questionnaire. Similar in style to questionnaires used in other mental health literacy research, it was designed to assess members of the public and health professionals’ ability to identify the presence of comorbid PTSD and chronic pain in an individual injured in a motor vehicle accident. Second, it assessed beliefs about interventions and outcomes for simple chronic pain and comorbid pain and PTSD, comparing differences within and between the public and health professionals, and between four different health professions (GPs, psychologists, physiotherapists and chiropractors).

The study found that, in apparent disregard of evidence-based healing rates and understanding about the nature of chronic pain, professionals were still likely to adopt a structural/medical approach when interpreting the presenting problem in the vignette. This appeared to be particularly the case in the context of the singular presentation of chronic pain and for physical therapists (consistent with their training and professional orientation). By contrast, chronic pain disorder was relatively neglected as a diagnostic category. Moreover, although psychologists’ views of chronic pain were stable (independent of PTSD), chronic pain was less well recognised by other professions in its presence. Professionals were relatively accurate in detecting PTSD when it was present but also prone to false positive recognition. Further, depression was more likely to be detected in the singular presentation of chronic pain rather than under comorbid conditions. The public was quite accurate in their ability to detect at least some form of
psychological problem in the face of PTSD but demonstrated poor awareness of the contribution of psychological factors to chronic pain.

With respect to treatment, several differences emerged, not only between the public and professionals (e.g., in endorsement of psychological treatment and recommended medications), but also between professionals and standard clinical guidelines (e.g., in endorsement of passive physical treatments, counselling versus CBT). In addition, both professionals and the public were less likely to endorse psychologists as helpful if PTSD was absent. Choices about treatment options more relevant for chronic pain were conversely less likely to be endorsed in the presence of PTSD (despite no differences in the vignette description of pain severity).

9.2 Other Implications and Future Directions

It is important to seek to replicate the findings from Studies 1-4 given the relatively small sample sizes. Larger numbers would perhaps permit improved definition of groups, as would the inclusion of a more homogenous sample with respect to pain problems or nature of the traumatic event. It may also be useful to explore some of the issues raised here under prospective investigation. Assessment of EMG reactivity may be worth including in addition to or instead of the autonomic measures tested here.

It is important to recognise that the findings of Studies 3 & 4 were obtained within the context of experimental manipulations. In contrast to studying individuals in their natural environment, as they react to spontaneous post-traumatic or painful stimuli, the current manipulations may not accurately reflect the circumstances in which individuals find themselves nor their true responses to these circumstances (e.g., Brewin & Holmes, 2003). While laboratory-based experimental manipulations of clinical issues carry constraints related to ecological validity, it is only through the type of research designs employed in this thesis that the psychological and autonomic processes underlying the differential responses of chronic pain patients with and without PTSD can be identified. Moreover, the confounds inherent in naturalistic pain experiences would have made inferences about general psychological process an impossibility. Future research may benefit from improving the means of directly activating the trauma-network, using alternative measures of pain threshold (e.g., site specific; cold pressor, pressure pain), or selecting specifically feared and functionally relevant movements.
Likewise, it is not clear how well responses to a vignette reflect actual clinical decisions and behaviour, which are subject to the nuances of personal interaction with a patient. Future research could extend study 5, whilst retaining relative control over certain factors, by tailoring the questionnaire to professionals, perhaps including a more detailed case history and questions designed to elicit information about reasoning and judgments that are applied. Alternatively, studies could proceed within a naturalistic setting.

Greater understanding of the impact of PTSD on adjustment to and management of chronic pain has important clinical implications, particularly for the assessment and treatment of existing patients with chronic pain and the planning of early intervention strategies for patients with acute or sub-acute conditions to avert a chronic course. This may help to minimise individual suffering and disability, improve the allocation of health resources and reduce associated health care costs.

Indeed, the level of maladjustment and poor outcomes found here in patients with comorbid chronic pain and PTSD (Studies 1 – 4) suggest that this group is in high need of appropriate management and treatment. Given prior research findings that this group performs poorly in standard treatment, future research needs to be directed to the development of evidence-based treatment specific to the needs of this group. In the meantime, the results of Study 5 suggest that education aimed at increasing professional awareness and implementation of current clinical guidelines (relating to the independent disorders) is likely to improve the outcomes presently achieved in clinical practice. Public dissemination of this information may also improve adherence to and satisfaction with treatment recommendations.

Overall, Studies 1-4 support prior research findings of poor adjustment to chronic pain in patients with comorbid PTSD. This poor adjustment is broadly associated with and at times predicted by various fear-avoidance constructs. Taken together the studies provided little to no support for a direct link between PTSD – operating on a moment-to-moment basis via hypervigilance, hyperarousal or activation of a trauma fear-network – and pain severity. On the other hand, PTSD appears to impact on the more chronic aspects of adjustment to and management of chronic pain. The fifth study highlights that comorbidity significantly affects individuals’ detection of presenting
problems and their choices about treatments, with discrepancies occurring between the public and professionals, between different professions, and between opinion and clinical guidelines.

It may be that until such factors are better understood, individuals who experience PTSD simultaneously with chronic pain really are caught in a jam.
REFERENCES


Clark, M. E. (1996). *Tampa Scale (v.2)*. Unpublished manuscript, James Haley Veterans Hospital, Tampa, Florida.


arthritis: II. Sexual, physical and emotional abuse and neglect. Psychosomatic Medicine, 59, 572-577.


Part 1: Background Information

Date completed: ____________________

1. Name: __________________________

2. Gender: _________________________  
   female / male (please circle)

3. Date of Birth: ____________________  
   4. a. Country of Birth: ________________________
   b. If not Australia, how long have you lived here? ________________________

5. Language(s) spoken: ________________________

6. Marital status: ________________________  
   (please circle)  
   1 single  2 married  3 divorced/separated  4 de facto  5 widowed  6 other relationship (e.g., girlfriend/boyfriend)

7. Do you live... ________________________  
   (please circle)  
   1 alone  2 with a partner or spouse  3 with a partner or spouse and child(ren)  
   4 with children only  5 with parents  6 with other relatives  7 with friends or flatmates

8. Highest level of education... ________________________  
   (please circle)  
   1 university postgraduate  2 university undergraduate  3 T.A.F.E.  4 HSC (Year 12)  
   5 Year 11  6 School Certificate (Year 10)  7 Year 9  8 Less than Year 9

Part 2: Pain History

1. When did your pain first start? _____ / _____ / _____  
   (Be as specific as possible)

2. How did your pain begin? ________________________  
   (Please circle only one. If more than one option applies, circle the best one.)  
   1 work-related accident  2 at work, but not due to an accident (please describe)  
   3 accident at home  4 car, truck or motor-bike accident  
   5 sporting injury  6 after surgery  7 after an illness  8 pain just began, no clear reason  
   9 other reasons (please describe) ________________________

3. Please describe your current pain problem. What have you been told is wrong with you? ________________________
   ________________________
   ________________________

4. Please list all the places in your body where you have felt pain in the past month, starting with the place that bothers you most: ________________________
   ________________________
   ________________________
   ________________________
5. In the past month, how often have you had pain?
(Please circle one).
1 constant - always present, always the same intensity
2 always present but the intensity varies
3 usually present, but I have short periods without pain
4 often present, but I have pain-free periods lasting up to several hours
5 often present, but I am pain-free for much of the day
6 occasionally present for brief periods, but not every day
7 rarely present - I have pain every now and then, with days or weeks in between.

6. Please indicate the intensity of your pain by circling a number on the line below:

a. How intense is your pain is right now?
n0 pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

b. In the past month, what was your average pain level?
n0 pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

7. What do you realistically expect will happen to your pain in future?
[please circle]
1 get worse
2 no change
3 improve
4 totally recover

8. Do you receive or are you seeking compensation (e.g., through litigation) for your pain? (please circle)
   Yes No

Part 3: Treatment History

1. Please list each medication you took for pain during the past week, and circle the number of days you took each medication during the past week. Please tick here [ ] if you do not take any medication.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>7</td>
</tr>
</tbody>
</table>

   0 1 2 3 4 5 6 7 days
   0 1 2 3 4 5 6 7 days
   0 1 2 3 4 5 6 7 days
   0 1 2 3 4 5 6 7 days

   0 1 2 3 4 5 6 7 days

2. Please circle the people you have seen since your pain began.

<table>
<thead>
<tr>
<th>Type</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>acupuncturist</td>
<td>10</td>
</tr>
<tr>
<td>anaesthetist</td>
<td>11</td>
</tr>
<tr>
<td>chiropractor</td>
<td>12</td>
</tr>
<tr>
<td>homeopath</td>
<td>13</td>
</tr>
<tr>
<td>hypnotherapist</td>
<td>14</td>
</tr>
<tr>
<td>massage therapist</td>
<td>15</td>
</tr>
<tr>
<td>naturopath</td>
<td>16</td>
</tr>
<tr>
<td>neurologist</td>
<td>17</td>
</tr>
<tr>
<td>neurosurgeon</td>
<td>18</td>
</tr>
<tr>
<td>occupational therapist</td>
<td>19</td>
</tr>
</tbody>
</table>

3. How many times have you seen a professional about your pain in the last 3 months? (please circle)
   1 0-3 consultations
   2 4-6 consultations
   3 7-12 consultations
   4 13-24 consultations

Please continue to the next page
### Part 4: Thoughts and Reactions to Pain

This part is to help us better understand the way you view your pain problem. There are no right or wrong answers – answer according to what you truly think or feel (not according to what other people think). Please use the rating scale below to indicate how often you do each of the following thoughts or activities. **Circle any number from 0 (never) to 5 (always) for each item.**

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I think that if my pain gets too severe, it will never decrease</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>My mind is calm when I am in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>When I feel pain, I try to stay as still as possible</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>I become sweaty when in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>When I feel pain, I am afraid something terrible will happen</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>My thoughts are agitated and keyed up as pain approaches</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>I go immediately to bed when I feel severe pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>Even though it hurts, I know that I'm going to be OK</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>My body gets shaky when I hurt</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>I feel disoriented and confused when I hurt</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>When pain gets severe, I call my doctor or go to the emergency room</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>I begin trembling when engaged in an activity that increases pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13</td>
<td>When I feel pain, I become afraid of dying</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>I can't think straight when in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15</td>
<td>I will stop any activity as soon as I sense pain coming on</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16</td>
<td>Even if I do an activity that causes pain, I know it will decrease later</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17</td>
<td>Pain seems to cause my heart to pound or race</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18</td>
<td>I think I have a serious medical problem that my physician has failed to uncover</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19</td>
<td>As soon as pain comes on, I take medication to reduce it</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20</td>
<td>I have pressure or tightness in my chest when in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21</td>
<td>When I feel pain, I think I might be seriously ill</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22</td>
<td>During painful episodes it is difficult for me to think of anything besides pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>23</td>
<td>I avoid important activities when I hurt</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>24</td>
<td>When I sense pain, I feel dizzy or faint</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>25</td>
<td>Pain sensations are terrifying</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>26</td>
<td>When I hurt, I think about pain constantly</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>27</td>
<td>I take medication if I know I need to do something that usually increases pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>28</td>
<td>I have trouble catching my breath when I have pain sensations</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>29</td>
<td>I dread feeling pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>30</td>
<td>I am bothered by unwanted thoughts when I'm in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>31</td>
<td>If a chance comes to do something I enjoy, I do it even if it causes pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>32</td>
<td>Pain makes me nauseous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>33</td>
<td>When pain comes on strong, I think I might become paralyzed or more disabled</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>34</td>
<td>I find it hard to concentrate when I hurt</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>35</td>
<td>I seek reassurance that I am O.K. during times of more severe pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>36</td>
<td>I find it difficult to calm my body down after periods of pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>37</td>
<td>I worry when I am in pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>38</td>
<td>My stomach bothers me when I experience pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>39</td>
<td>I try to avoid activities that cause pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>40</td>
<td>I can think pretty clearly even while experiencing severe pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Part 5: Coping with Pain

For each statement below, please indicate how often you think or behave like that when you feel pain, by circling a number between 0 (never) and 6 (always):

<table>
<thead>
<tr>
<th>When I feel pain ...</th>
<th>Never</th>
<th>Sometimes</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I try to feel distant from the pain, almost as if the pain was in somebody else's body</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>2. I leave the house and do something, such as going to the movies or shopping</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>3. I try to think of something pleasant</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4. I don't think of it as pain but rather as a dull or warm feeling</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>5. It is terrible and I feel it's never going to get any better</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6. I tell myself to be brave and carry on despite the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7. I read</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8. I tell myself that I can overcome the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9. I take my medication</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10. I count numbers in my head or run a song through my mind</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>11. I just think of it as some other sensation, such as numbness</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>12. It is awful and I feel that it overwhelms me</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>13. I play mental games with myself to keep my mind off the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>14. I feel my life isn't worth living</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>15. I know someday someone will be here to help me and it will go away for a while</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>16. I walk a lot</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>17. I pray to God it won't last long</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>18. I try not to think of it as my body, but rather as something separate from me</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>19. I relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>20. I don't think about the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>21. I try to think years ahead, what everything will be like after I've gotten rid of the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>22. I tell myself it doesn't hurt</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>23. I tell myself I can't let the pain stand in the way of what I have to do</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>24. I don't pay any attention to the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>25. I have faith in doctors that someday there will be a cure for my pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>26. No matter how bad it gets, I know I can handle it</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>27. I pretend it's not there</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>28. I worry all the time about whether it will end</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>29. I lie down</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>30. I replay in my mind pleasant experiences in the past</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>31. I think of people I enjoy doing things with</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>32. I pray for the pain to stop</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>33. I take a shower or bath</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>34. I imagine that the pain is outside of my body</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>35. I just go on as if nothing happened</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>36. I see it as a challenge and don't let it bother me</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>37. Although it hurts, I just keep on going</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>38. I feel I can't stand it anymore</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>39. I try to be around other people</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>When I feel pain...</td>
<td>Never</td>
<td>Sometimes</td>
<td>Always</td>
</tr>
<tr>
<td>---------------------</td>
<td>-------</td>
<td>-----------</td>
<td>--------</td>
</tr>
<tr>
<td>40. I ignore it</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>41. I rely on my faith in God</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>42. I feel like I can't go on</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>43. I think of things I enjoy doing</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>44. I do anything to get my mind off the pain</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>45. I do something I enjoy, such as watching TV or listening to music</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>46. I pretend it's not a part of me</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>47. I do something active, like household chores or projects</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
<tr>
<td>48. I use a heating pad</td>
<td>0 1</td>
<td>2 3 4 5 6</td>
<td></td>
</tr>
</tbody>
</table>

**During the past week**, how many days did you use each of the following at least once in the day to cope with your pain? (Note: Please indicate the number of days you used each strategy for pain, whether or not you were experiencing pain at the time (i.e. include times when you used the strategy to minimise or prevent pain in the future).)

<table>
<thead>
<tr>
<th>Number of days used</th>
<th>1. Imagined a calming or distracting image to help me to relax</th>
<th>0 1 2 3 4 5 6 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Kept on doing what I was doing</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>3. Stretched the muscles in my legs and held the stretch for at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>4. Ignored the pain</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>5. I took a rest</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>6. Made arrangements to see a friend or family member</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>7. I went to bed early to rest</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>8. I got support from a friend</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>9. Asked someone to do something for me</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>10. Reminded myself that things could be worse</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>11. Avoided using part of my body (e.g., hand, arm, leg)</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>12. Focused on relaxing my muscles</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>13. Set on the floor, stretched and held the stretch at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>14. Told myself things will get better</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>15. Held on to something when getting up or sitting down</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>16. I got support from a family member</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>17. Exercised to strengthen the muscles in my arms for at least 1 minute</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>18. I rested as much as I could</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>19. Thought about someone with problems worse than mine</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>20. I talked to someone close to me</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>21. Told myself that I am adjusting to my pain problem better than many other people</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>22. Called a friend on the phone to help me feel better</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>23. Thought about all the good things I have</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>24. Listened to music to relax</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>25. Asked for help with a chore or task</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>26. Stretched the muscles in my neck (and held the stretch) for at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>27. Told myself my pain will get better</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>28. I didn't let the pain interfere with my activities</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>29. Exercised to strengthen the muscles in my legs for at least 1 minute</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>30. Thought about a friend who has coped well with a problem</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>31. Listened to a relaxation tape to relax</td>
<td>0 1 2 3 4 5 6 7</td>
<td></td>
</tr>
</tbody>
</table>

Please continue to the next page
<table>
<thead>
<tr>
<th></th>
<th>Number of days used</th>
</tr>
</thead>
<tbody>
<tr>
<td>32. Engaged in aerobic exercise (exercise that made my heart beat faster) for at least 15 minutes</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>33. Limited my walking because of pain</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>34. Just didn’t pay attention to the pain</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>35. Walked with a limp to decrease the pain</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>36. Meditated to relax</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>37. Reminded myself that I had coped with the pain before</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>38. Lay on my back, stretched, and held the stretch at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>39. Held part of my body (e.g., arm) in a special position</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>40. Rested in a chair or recliner</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>41. Avoided putting weight on feet or legs</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>42. Asked for help in carrying, lifting or pushing something</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>43. Exercised to improve my overall physical condition for at least 5 minutes</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>44. Talked to a friend or family member for support</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>45. Reminded myself that there are people who are worse off than I am</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>46. Limited my standing time</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>47. Lay down on a bed</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>48. Avoided some physical activities (lifting, pushing, carrying)</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>49. Reminded myself about things that I have going for me such as intelligence, good looks, and good friends</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>50. Used self-hypnosis to relax</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>51. I just kept going</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>52. Exercised to strengthen the muscles in my stomach for at least 1 minute</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>53. Got together with a friend</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>54. Reminded myself that others have coped well with pain problems</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>55. Stretched the muscles where I hurt and held the stretch for at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>56. Avoided activity</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>57. Got together with a family member</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>58. Went into a room by myself to rest</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>59. Used deep, slow breathing to relax</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>60. Exercised to strengthen the muscles in my back for at least 1 minute</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>61. Stretched the muscles in my shoulders or arms, and held the stretch for at least 10 seconds</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>62. Asked someone to get me something (e.g., medicine, food, drink)</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>63. Did not let the pain affect what I was doing</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>64. Lay down on a sofa</td>
<td>0 1 2 3 4 5 6 7</td>
</tr>
</tbody>
</table>

66. Based on all the things you do to cope, or deal with your pain, on an average day, how much control do you feel you have over it? Please circle the appropriate number.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no control</td>
<td>some control</td>
<td>complete control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

66. Based on all the things you do to cope, or deal with your pain, on an average day, how much are you able to decrease it?

Please circle the appropriate number.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>can't decrease</td>
<td>can decrease it somewhat</td>
<td>can decrease it completely</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please continue to the next page
Part 6: Effect of Pain on Life Activities

This part is designed to measure whether your pain is preventing you from doing what you would normally do, or from doing it as well as you normally would. Think about the overall impact of your pain, not just when the pain is at its worst, by circling a number between 0 and 10, where 0 means no disability at all and 10 means your activities are totally disrupted or prevented by your pain.

1. **Family / home responsibilities**
   This category refers to activities related to the home or family, including chores or duties around the house (e.g., yard work) and errands or favours for other family members (e.g., driving the children to school).

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

3. **Social activity**
   This category refers to participating in activities with friends and acquaintances (other than family members), including parties, theatre, concerts, dining out, and other social functions.

4. **Occupation**
   This category refers to activities that are a part of or directly related to your job. This includes non-paying jobs as well (e.g. housewife or a volunteer worker).

5. **Sexual behaviour**
   This category refers to the frequency and quality of your sex life.

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

7. **Life support activity**
   This category refers to basic life-supporting behaviours such as eating, sleeping and breathing.

Please continue to the next page
Part 7: Mood

For each of these statements, please indicate which answer best describes how you have been feeling in the past four weeks, by circling a number between 0 and 3, using the following scale:

<table>
<thead>
<tr>
<th>Statement</th>
<th>Rarely or none of the time</th>
<th>Some or a little of the time</th>
<th>A moderate amount of time</th>
<th>Most of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I feel downhearted and sad</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Morning is when I feel best</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I have crying spells or feel like it</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I have trouble getting to sleep at night</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I feel that nobody cares</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I eat as much as I used to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I still enjoy sex</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I notice that I am losing weight</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I have trouble with constipation</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>My heart beats faster than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I get tired for no reason</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>My mind is as clear as it used to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I tend to wake up too early</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I find it easy to do the things I used to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I am restless and can’t keep still</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>I feel hopeful about the future</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I am more irritable than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I feel quite guilty</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I feel that I am useful and needed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>My life is pretty full</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I feel that others would be better off if I were dead</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>I still enjoy the things I used to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Please indicate how much you agree with the following statements by circling a number between 0 and 3, using the following scale:

<table>
<thead>
<tr>
<th>Statement</th>
<th>Very little</th>
<th>A little</th>
<th>Some</th>
<th>Much</th>
</tr>
</thead>
<tbody>
<tr>
<td>I feel downhearted and sad</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Morning is when I feel best</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
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<td>0</td>
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<td>3</td>
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</tbody>
</table>

Please continue to the next page
Part 8: Information about the circumstances that resulted in your injury / pain

Below are some questions about the circumstances that resulted in your injury or pain. Please answer them by circling Yes or No.

1. Did your pain begin as a result of a specific injury, illness, accident, incident or event? Yes  
   If you answered Yes, please complete the remainder of Part 8.  
   If you answered No, please go to Part 9.

2. Under the circumstances that caused your injury/pain:
   a. Was someone else physically injured? Yes  
   b. Did you think that your life was in danger? Yes
   c. Did you think that someone else's life was in danger? Yes
   d. Did you feel helpless? Yes
   e. Did you feel terrified? Yes

Below is a list of problems that people sometimes have after experiencing a traumatic event. In relation to the circumstances that resulted in your current injury / pain, please indicate whether, and if so how often, each problem listed below has bothered you in the past month, by circling a number between 0 and 3 using the following scale:

<table>
<thead>
<tr>
<th>Problem</th>
<th>0 = not at all or only one time</th>
<th>1 = once a week or less/once in a while</th>
<th>2 = 2-4 times a week/half the time</th>
<th>3 = 5 or more times a week/ almost always</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. Having upsetting thoughts or images about the event that came into your head when you didn't want them to</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Having bad dreams or nightmares about the event</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Reliving the event, acting or feeling as if it was happening again</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Feeling emotionally upset when you were reminded of the event (for example, feeling scared, angry, sad, guilty, etc)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Experiencing physical reactions when you were reminded of the event (for example, breaking out in a sweat, heart beating fast)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Trying not to think about, talk about, or have feelings about the event</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Trying to avoid activities, people or places that remind you of the event</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Not being able to remember an important part of the event</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Having much less interest or participating much less often in important activities</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
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<tr>
<td>12. Feeling distant or cut off from people around you</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Feeling emotionally numb (for example, being unable to cry or unable to have loving feelings)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
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<tr>
<td>15. Having trouble falling or staying asleep</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>16. Feeling irritable or having fits of anger</td>
<td>0 1 2 3</td>
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<tr>
<td>17. Having trouble concentrating (eg, drifting in and out of conversations, losing track of a story on television, forgetting what you read)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
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<tr>
<td>18. Being overly alert (for example, checking to see who is around you, being uncomfortable with your back to a door, etc)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Being jumpy or easily startled (for example, when someone walks up behind you)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please continue to the next page
20. How long have you experienced the problems that you reported in the section above? (Please circle one)
   1. less than 1 month
   2. 1 to 3 months
   3. more than 3 months

21. How long after the event did these problems begin? (Please circle one)
   1. less than 6 months
   2. 6 or more months

22. Indicate below if the problems you rated in the section immediately above have interfered with any of the following areas of your life during the past month, by circling either Yes or No.
   a. Work
   b. Household chores and duties
   c. Relationships with friends
   d. Fun and leisure activities
   e. Schoolwork
   f. Relationships with your family
   g. Sex life
   h. General satisfaction with life
   i. Overall level of functioning in all areas of your life

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>a.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b.</td>
<td></td>
<td></td>
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<tr>
<td>c.</td>
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<tr>
<td>d.</td>
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<td>e.</td>
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<tr>
<td>f.</td>
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<tr>
<td>g.</td>
<td></td>
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<tr>
<td>h.</td>
<td></td>
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<tr>
<td>i.</td>
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</tr>
</tbody>
</table>

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**Part 9: Information about other life experiences**

Some people have lived through or witnessed a very stressful and traumatic event at some other point in their lives. Below is a list of traumatic events. Please circle ALL of the events that have happened to you or that you have witnessed. **Do not include the incident or circumstances that resulted in your current injury/pain.**

1. Serious accident, fire or explosion (for example an industrial, farm, car, plane, or boating accident)
2. Natural disaster (for example, tornado, hurricane, flood or major earthquake)
3. Non-sexual assault by a family member or someone you know (for example, being mugged, physically attacked, shot, stabbed, or held at gun-point)
4. Non-sexual assault by a stranger (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)
5. Sexual assault by a family member or someone you know (for example, rape or attempted rape)
6. Sexual assault by a stranger (for example, rape, or attempted rape)
7. Military combat or a war zone
8. Sexual contact when you were younger than 18 with someone who was 5 or more years older than you (for example, contact with genitals, breasts)
9. Imprisonment (for example, prison inmate, prisoner of war, hostage)
10. Torture
11. Life threatening illness
12. Other traumatic event (please specify)______________________________________________

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Please continue to the next page
Part 10: Work

And now finally, some questions about your work.

1. What is your main occupation? Please specify the nature and level of your position. If you are not currently employed, what was your main occupation prior to ceasing work?

2. What is your current work status?
   (please circle)
   1. full time work
   2. part-time work (include graduated return to work plans)
   3. voluntary work
   4. home duties
   5. studying
   6. retraining
   7. retired
   8. unemployed due to pain
   9. unemployed due to other reasons

3. What is your current source of income? (you may circle more than one)
   1. wages / salary
   2. workers compensation
   3. sickness benefits
   4. invalid or disability benefits
   5. unemployment benefits
   6. supporting parents' benefits
   7. spouse's / partner's / parents' earnings
   8. other (please specify) ____________________

The next questions are about paid work. Answer Section A if you are currently working OR Section B if you are not currently working.

Section A. Answer if you are currently working...

a. are you on restricted duties? (please circle) yes / no / not applicable
b. how much time have you had off work due to pain in the last 12 months? ________________
c. how well do you think you are currently able to do your normal job? (please circle)
   1. as well as I could before my injury / pain
   2. only with reduced hours and/or restricted duties
   3. not at all in my current condition
   4. not applicable

Section B. Answer if you are not currently working...

a. how long has it been since you last worked? ________________________
b. do you have a job to go back to? (please circle) yes / no / not applicable
c. have you tried to return to work? (please circle) yes / no / not applicable

   number of attempts ________________
d. how much time have you had at work in the last 12 months? ________________
e. how well do you think you are currently able to do your normal job? (please circle)
   1. as well as I could before my injury / pain
   2. only with reduced hours and/or restricted duties
   3. not at all in my current condition
   4. not applicable

That concludes the questionnaire. Thank you very much for taking the time to complete it. Your assistance is appreciated.
ADJUSTMENT TO PHYSICAL INJURY AND CHRONIC PAIN STUDY

For your information

This is a research project investigating adjustment to physical injury and management of chronic pain. For this reason I wish to collect information about your thoughts and feelings about pain, your coping strategies, the impact of pain on your daily living and your psychological and emotional well-being, as well as general characteristics of your pain problem (e.g., how it started, diagnosis, intensity, frequency, duration and prior treatment).

I would like to seek your permission to include you in the research. I have attached a consent form for you to sign if you wish to participate. The research will require you to fill out a questionnaire, which you may complete at home. The questionnaire should take about half an hour to an hour to complete and the information obtained from these forms will be kept confidential, so far as the law provides. If any information is included in reports or publications you will not be personally identified.

An ethics protocol covering this research has been approved by the ACT Department of Health Ethics Committee and the Australian National University Human Research Ethics Committee. There are no foreseen adverse effects or risks related to this project.

You do not have to take part in this study, however, if you do agree to participate please sign the attached Consent Form. Whether or not you decide to participate in the study will not effect the availability or nature of treatment you receive at [insert name of referring organisation].

If you have any questions about the research project or your participation please contact me at the address or telephone number below. Alternatively, you may wish to direct questions to the other people or organisations listed. If for some reason you become distressed as a result of completing the questionnaires, a psychologist will be made available for counselling, where necessary, specifically only to address issues raised by the questionnaire.

Belinda Barker,
School of Psychology, The Australian National University, ACT 0200
Ph: 6125 0788; Mobile: 0410 629 511; fax: 6125 0499;
email: belinda.barker@anu.edu.au
Prof. Don Byrne,
Clinical Research Supervisor, The Australian National University, ACT 0200
Ph: 02 6125 3974; fax: 6125 0499

The Secretary, ACT Department of Health Ethics Committee
Second Floor, North Building, London Circuit, Canberra City, ACT 2601
Ph: 02 6205 0846

The Human Ethics Officer, Research Services Office
The Australian National University, ACT 0200
Ph: 02 6125 2900

[Insert contact details for referring organisation]

Thank you for your time,

Yours sincerely,

Belinda Barker
Psychologist MAPS
B.Sc. (Hons-Psychology), LLB.
Consent Form to Participate in a Research Project

I, _______________________________________________________

(name of participant)

of _______________________________________________________

__________________________ _______________________________

(street) (suburb/town) (state & postcode)

have been asked to consent to participation in a research project entitled ‘Adjustment to Physical Injury and Chronic Pain’. In relation to this project I have read the Patient Information Sheet, which I have initialed. I have been informed of the following points:

1. Approval of an ethics protocol submitted for this research has been granted by the ACT Department of Health & Community Care Human Research Ethics Committee and the Australian National University Human Research Ethics Committee.

2. The aim of the project is to investigate adjustment to physical injury and management of chronic pain.

3. The results obtained from the study may or may not be of direct benefit to my medical management.

4. The procedure will involve completing questionnaires after referral to the [relevant organisation].

5. There are no foreseen adverse effects or risks related to this project.

6. Should I develop a problem which I suspect may have resulted from my involvement in this project, I am aware that I may contact: Belinda Barker, Division of Psychology, Australian National University ACT 0200 or on 02 6125 0788 OR 0410 629 511 or Prof. Don Byrne, Clinical Supervisor, Australian National University ACT 0200 or on 02 6125 3974 or [insert contact details for referring organisation].

7. Should I become distressed as a result of completing the questionnaire, I understand that a psychologist from [insert organisation as applicable] will be made available for counselling, where necessary, specifically only in relation to the issues raised by the questionnaire.

8. Should I have any problems or queries about the way in which the study was conducted, and I do not feel comfortable contacting the research staff, I am aware that I may contact the ACT Department of Health Ethics Committee Secretary on Second Floor, North Building, London Circuit, Canberra City, ACT 2601 or phone number 02 6205 0846 OR The ANU Human Ethics Officer, Australian National University, ACT 0200 or phone number 02 6125 2900.

9. I can refuse to take part in this project or withdraw from it at any time without affecting my medical care.

10. Participation in this project will not result in any extra medical or other costs to me.

11. I understand that where the results of the research are made accessible, my involvement and my identity will not be revealed in formal publications or general reports and information obtained from my participation will remain confidential, in so far as the law provides.

12. In giving my consent, I acknowledge that the relevant Health Department Officials directly involved in the study (including Belinda Barker) may examine my medical records but only as they relate to this project.
After considering all these points, I accept the invitation to participate in this project.

I also state that I have not participated in any other research project in the past 3 months. If I have, the details are as follows:

Please sign below if consent is given to participate:

Date: ___________   Witness: ____________________________
       (please print name)

Signature: ____________________________  ____________________________
           (of participant/volunteer)         (of witness)

Investigator's Signature: ____________________________  v 22.08.01
APPENDIX C

LETTERS APPROVING ETHICS PROTOCOL FOR STUDY 1
Dear Ms Barker,

The ACT Health and Community Care Human Research Ethics Committee considered the proposed study ‘the Impact of Psychological Trauma on Adjustment to Physical Injury and Management of Chronic Pain’ study at the meeting held on 13 August 2001. Ethics Committee Submission No ETH.7/01 240 refers.

The Committee agreed that amendments are required before the study can be approved.

Concerns raised by the Committee included:
- the Patient Information Sheet, paragraph one, first sentence, “My name is Belinda Barker and I am completing a Doctor of Philosophy (Clinical Psychology) at the Australian National University. As part of that Doctorate” should be depersonalised and changed to “This is a research project into ...”;
- the Patient Information Sheet, contact details listing, should include the name of Mr Tony Corless, Acting Director, Psychology, The Canberra Hospital, and his telephone number, under the Pain Management Unit paragraph; and
- the Patient Information Sheet and Consent Form should be on the appropriate ACT Community Care or The Canberra Hospital letterhead.

The Committee agreed that the Chair may approve the study out of session, provided the above concerns are addressed and a copy of the amended Patient Information Sheet and Consent Form are received by the Committee.

Yours sincerely,

Elizabeth Grant
Chair
Ethics Committee
2x August 2001
Ms Belinda Barker  
School of Psychology  
Australian National University  
ANU 0200

Dear Ms Barker

Thank you for your letter of 22 August 2001, clarifying the concerns raised by the Committee at the meeting held on 13 August 2001, in respect of the proposed study titled ‘The Impact of Psychological Trauma on Adjustment to Physical Injury and Management of Chronic Pain’. Ethics Committee Submission No ETH.7/01.240 refers.

I am pleased to advise you that the study has now been approved, including the Patient Information Sheet (Version: 22.08.01), Consent Form (Version: 22.08.01) and Questionnaire.

I attach for your records an Outcome of Consideration of Protocol form.

You may recall that the ACT Health and Community Care Human Research Ethics Committee Guidelines of Application require you to complete payment of the levy when approved by the Ethics Committee.

Please forward $27.50 levy fee to the Secretariat, ACT Health and Community Care Human Research Ethics Committee, GPO Box 825, Canberra ACT 2601 as soon as possible. An invoice is attached for your attention.

Yours sincerely

[Signature]

Elizabeth Grant  
Chair  
Ethics Committee

30 August 2001
Outcome of Consideration of Protocol

Submission No: ETH.7/01.240 Date of Approval: 30 August 2001

Project Title:
The Impact of Psychological Trauma on Adjustment to Physical Injury and Management of Chronic Pain

Submitted by:
Ms Belinda Barker

Your project was considered by the ACT Health and Community Care Human Research Ethics Committee and approved for a period of two years.

Further Action required:

Review due: August 2002

The Ethics Committee require as part of the review process that:

• At regular periods, and not less frequently than annually, Principal Investigators are to provide reports on matters including:
  - security of records
  - compliance with approved consent procedures and documentation
  - compliance with other approved procedures.
  - as a condition of approval of the protocol, that Investigators report immediately:
    - adverse affects on subjects
    - proposed changes in the protocol
    - unforeseen events that might affect continued ethical acceptability of the project.

• All published reports to carry an acknowledgement stating:
  - approved on 13 August 2001 by the ACT Health and Community Care Human Research Ethics Committee.

MS ELIZABETH GRANT AM, CHAIR Date: 30 August 2001
Ms Belinda Barker  
School of Psychology  
Australian National University  
ANU 0200

Dear Ms Barker,

Thank you for your letter of 27 February 2002, and the amended Patient Information Sheet and Consent Form for the study The Impact of Psychological Trauma on Adjustment to Physical Injury and Management of Chronic Pain. Ethics Committee Submission No ETH.7/01.240 refers.

The Committee approved the Patient Information sheet (Version: 20.02.02) and Consent Form (Version: 20.02.02) at the meeting held on 8 April 2002.

Yours sincerely,

Elizabeth Grant AM  
Chair  
Ethics Committee  

23 April 2002
28 August 2001

Ms Belinda Barker
PhD (Clinical Psychology) student
Department of Psychology
Faculty of Science
The Australian National University
ACT 0200

Dear Ms Barker,

Protocol 2001/89
The impact of psychological trauma on adjustment to physical injury and chronic pain

On behalf of the Human Research Ethics Committee I am pleased to advise that the above protocol has been approved as per the attached Outcome of Consideration of Protocol. Please note that as a formality this approval is subject to formal ratification by the Committee at its next meeting.

For your information:
1. Under the NHMRC/AVCC National Statement on Ethical Conduct in Research Involving Humans we are required to follow up research that we have approved. Once a year (or sooner for short projects) we shall request a brief report on any ethical issues which may have arisen during your research and whether it proceeded according to the plan outlined in the above protocol.
2. Please notify the Committee of any changes to your protocol in the course of your research, and when you complete or cease working on this project.
3. The validity of this current approval is five years' maximum from the date shown on the attached Outcome of Consideration of Protocol form. For longer projects you are required to seek renewed approval from the Committee.

Yours sincerely,

Sylvia Deutsch
Secretary, Human Research Ethics Committee
Outcome of Consideration of Protocol

Researcher: Ms Belinda Barker
Contact details: PhD (Clinical Psychology) student, Department of Psychology, Faculty of Science
Protocol No.: 2001/89
Title: The impact of psychological trauma on adjustment to physical injury and chronic pain
Date on application: Date received in Research Services Office: 29 June 2001

On behalf of the Human Research Ethics Committee,

I approve/do not approve the above protocol.

Approval is subject to the following conditions:

Reasons for non-approval:

Review due:

Chairperson: (Dr Peter McCullagh) Date: 28-8-01
17 April 2002

Ms Belinda Barker  
PhD student  
Department of Psychology  
Faculty of Science  
The Australian National University  
Canberra  
ACT 0200

Dear Ms Barker,

Protocol 2001/89  
The impact of psychological trauma on adjustment to physical injury and chronic pain  
Variation: Recruitment of additional participants from selected rehabilitation providers in the ACT

I am pleased to advise that the above variation to the above protocol, as described in your email to me of 27 February 2002, has been approved by the Chair of the Human Research Ethics Committee Dr. Peter Hiscock. The approval of the variation will routinely be reported to the Committee at its next meeting on 19 April 2002.

Please do not hesitate to contact me if you have any queries.

Yours sincerely

Sylvia Deutsch  
Secretary, Human Research Ethics Committee
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\( r = r_{pb} \) when correlated with a continuous variable or \( r_p \) when correlated with another dichotomous variable.

* \( p < .05 \), ** \( p < .01 \), *** \( p < .001 \)
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APPENDIX E
STUDIES 2, 3 & 4 INFORMATION SHEET & CONSENT FORM
ADJUSTMENT TO PHYSICAL INJURY AND CHRONIC PAIN STUDY

PARTICIPANT INFORMATION & CONSENT FORM

This is a research project investigating how people adjust after a physical injury and how they manage chronic pain. Before you decide to participate in any research project, it is important that you understand what is involved. If there is anything in this information sheet that you do not understand, please ask the health practitioner who told you about the study or contact the researchers.

In this project, we wish to collect general information about your pain problem, as well as information about your thoughts and feelings about your injury and pain, and the impact the injury and pain has had on your daily living and on your mental and emotional well-being.

Participation in this project involves three stages. In the first stage you will need to fill in some brief questionnaires (about 10 minutes), which you can do at home and bring to the second stage. In the second stage you will be asked to fill in another brief questionnaire, view some pictures of daily activities and have an interview with a psychologist about your reactions to the injury (between one hour and one and a half hours). During this second session, it will be necessary to take readings of your heart rate, blood pressure, skin conductance, and muscle tension using specialised equipment. You will be asked to attend twice during the third stage, for about half an hour each time. During these sessions you will be asked to complete a short physical task and provide some ratings, and you may be asked some further questions about your injury. The task will be selected in conjunction with the practitioner who referred you to the study and will take into account your physical capacity and ratings you made during the second stage.

Information obtained during the research project will be kept confidential, in so far as the law provides. This means that you won’t be personally identified and your personal information will not be used for any other purpose other than this project. Information will not be released to your referring practitioner, except to assist with the selection of the physical task, unless you otherwise provide consent for us to do so.

If for some reason you become distressed as a result of participating in the research project, a psychologist will be made available for counselling, where necessary, specifically only to address issues raised by the research participation.

Whether or not you decide to participate will not affect the availability or nature of assessment or treatment you receive at [insert name of referring organisation]. You do not have to take part in this project and if you wish to withdraw from this project or do not wish to continue with any part of the study, at any time, for whatever reason, you are completely entitled to do so. Your withdrawal would not be held against you in any way. If you do agree to participate, please sign the Consent Form and retain the copy for yourself.

A-27
If you have any questions about the research project or your participation, please contact:

- **Ms Belinda Barker (Primary Investigator)**
  School of Psychology, The Australian National University, ACT 0200
  Ph: (02) 6125 0788; mobile: 0410 629 511; fax: (02) 6125 0499; email: belinda.barker@anu.edu.au

- **Prof. Don Byrne (Clinical Research Supervisor)**
  School of Psychology, The Australian National University, ACT 0200
  Ph: (02) 6125 3974; fax (02) 6125 0499; email: don.byrne@anu.edu.au

Approval of an ethics protocol submitted for this research has been granted by the Australian National University Human Research Ethics Committee. Should you wish to discuss the study with someone not directly involved, in particular in relation to matters concerning policies, information about the conduct of the study or your rights as a participant, or should you wish to make an independent complaint, you can contact: The Human Ethics Officer, Research Services Office, The Australian National University, ACT 0200, or on (02) 6125 2900.
CONSENT FORM

I, ____________________________

(your name)

of ____________________________

(street) _______________________

(suburb/town) __________________

(state) (postcode) _____________

consent to participate in a research project entitled ‘Adjustment to Physical Injury and Chronic Pain’. In relation to this project I have read the Participant Information and Consent Form, and acknowledge that I have been informed of the following points:

1. Approval of an ethics protocol submitted for this research has been granted by the ANU Human Research Ethics Committee.

2. The aim of the project is to investigate adjustment to physical injury and experience of chronic pain.

3. The results obtained from the study may or may not be of direct benefit to my medical, physical or psychological management.

4. The procedure will involve completion of questionnaires and rating scales, a clinical interview with a psychologist, two brief physical tasks and measurement of physiological reactions (e.g., heart rate, blood pressure, skin conductance, muscle tension).

5. I may or may not experience some mild discomfort or anxiety while completing the research procedure, however the extent to which this occurs will be limited wherever possible and I can choose not to continue at any time if I feel too distressed.

6. Should I develop a problem that I suspect may have resulted from my involvement in this project, I am aware that I may contact research staff (Belinda Barker, Prof. Don Byrne), according to the contact details above.

7. Should I become distressed as a result of completing the questionnaire, I understand that a psychologist will be made available for counselling, where necessary, specifically only in relation to the issues raised by the research procedures.

8. Should I have any problems or queries about the way in which the study was conducted, and I do not feel comfortable contacting the research staff, I am aware that I may contact the Human Research Officer, Research Services Office, The Australian National University, ACT 0200, or on (02) 6125 2900.

9. I can refuse to take part in this project or withdraw from it at any time, for whatever reason, without affecting my usual assessment, treatment or care.

10. Participation in this project will not result in any extra medical or other costs to me.

11. I understand that where the results of the research are made accessible, my involvement and my identity will not be revealed in formal publications or general reports, and information obtained from my participation will remain confidential and anonymous, in so far as the law provides.

12. In giving my consent, I acknowledge that the research staff may consult with my referring practitioner in selecting a physical task that is appropriate in light of my physical capacity.

After considering all these points, I accept the invitation to participate in this project.

Please sign below if consent is given to participate:
Revocation of Consent

I hereby wish to WITHDRAW my consent to participate in the research project described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with the person who referred me to the project, or any other health practitioner.

Print Name: ___________________ Signature: ___________________ Date: ________

The section for Revocation of Consent should be forwarded to Ms Belinda Barker, School of Psychology, Bldg 39, The Australian National University, ACT 0200.
APPENDIX F
STUDY 2 QUESTIONNAIRE
Part 1: Background Information

Date completed: ________________________

1. Name: ______________________________

2. Sex: female / male

3. Date of Birth: ____________

4. a. Country of Birth: ________________________

b. If not Australia, how long have you lived here? ____________

4. Marital status: 1 Single 2 Married 3 Divorced/separated 4 Defacto 5 Widowed 6 Other (girlfriend/boyfriend)

6. Do you live... (please circle)

1 alone 
2 with a partner or spouse 
3 with a partner or spouse and child(ren) 
4 with children only 
5 with parents 
6 with other relatives 
7 with friends or flatmates

7. Highest level of education... (please circle)

1 university postgraduate 
2 university undergraduate 
3 T.A.F.E. 
4 HSC (Year 12) 
5 Year 11 
6 School Certificate (Year 10) 
7 Year 9 
8 Less than Year 9

Part 2: Pain History

1. When did your pain start? __/_____/______ (Be as specific as possible)

2. How did your pain begin? (Please circle only one. If more than one option applies, circle the best one.)

1 work-related accident 
2 at work, but not due to an accident (please describe) 
3 accident at home 
4 car, truck or motor-bike accident 
5 sporting injury 
6 after surgery 
7 after an illness 
8 pain just began, no clear reason 
9 other reasons (please describe)

3. What diagnosis have you been given, if any: ________________________________

4. Please list all the places in your body where you have felt pain in the past month, starting with the place that bothers you most:

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

5. In the past month, how often have you had pain? (Please circle one).

1 constant - always present, always the same intensity 
2 always present but the intensity varies 
3 usually present, but I have short periods without pain 
4 often present, but I have pain-free periods lasting up to several hours 
5 often present, but I am pain-free for much of the day 
6 occasionally present for brief periods, but not every day 
7 rarely present - I have pain every now and then, with days or weeks in between.

Please continue to the next page
6. Please indicate the intensity of your pain by circling a number on the line below:

a. How intense is your pain right now?
   - no pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

b. In the past month, what was your average pain level?
   - no pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

c. In the past month, what was the lowest pain level?
   - no pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

d. In the past month, what was the highest pain level?
   - no pain 0 1 2 3 4 5 6 7 8 9 10 worst pain imaginable

7. What do you realistically expect will happen to your pain in the future? [please circle]
   - 1 get worse 2 no change 3 improve 4 totally recover

Part 3: Treatment & Work History

1. Please list each medication you took for pain during the past week, and circle the number of days you took each medication during the past week. Please tick here [ ] if you do not take any medication.

2. Which people have you seen for your pain? Please circle.
   - acupuncturist 6 massage therapist 11 orthopaedic surgeon 16 rheumatologist
   - anaesthetist 7 naturopath 12 osteopath 17 pain clinic
   - chiropractor 8 neurologist 13 physiotherapist 18 general practitioner
   - homeopath 9 neurosurgeon 14 psychologist
   - hypnotherapist 10 occupational therapist 15 psychiatrist
   - other __________________________

3. How many times have you seen a professional about your pain in the last month? [please circle]
   - less than once per week
   - once or twice per week
   - two or three times per week
   - four or five times per week
   - more than five times per week

4. Please indicate which best describes your current work situation
   - full time work
   - undergoing a graduated return to work (light duties or reduced hours)
   - part-time work (at pre-injury level)
   - voluntary work
   - home duties
   - studying/retraining
   - retired
   - unemployed due to pain
   - unemployed due to other reasons

5. Do you receive compensation or are you seeking compensation (e.g., through litigation) for your injury/pain? Yes No
6. If you answered NO to Q5, have you previously received compensation or a settlement to a compensation claim? Yes No

Please continue to the next page
# Part 4: General Feelings

For each of the statements below, please circle the number that best indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any one statement.

- **0** = Did not apply to me at all
- **1** = Applied to me to some degree, or some of the time
- **2** = Applied to me a considerable degree, or a good part of the time
- **3** = Applied to me very much, or most of the time

<table>
<thead>
<tr>
<th>Number</th>
<th>Statement</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>I found it difficult to wind down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2.</td>
<td>I felt downhearted and blue</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3.</td>
<td>I was aware of the action of my heart in the absence of physical exertion</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>(e.g., sense of heart rate increase, heart missing a beat)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>I perspired noticeably (e.g., hands sweaty) in the absence of high</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>temperatures or physical exertion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>I felt sad and depressed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6.</td>
<td>I found it hard to calm down after something upset me</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7.</td>
<td>I was aware of dryness of my mouth</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8.</td>
<td>I found it difficult to relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9.</td>
<td>I experienced breathing difficulty (e.g., excessively rapid breathing,</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>breathlessness in the absence of physical exertion)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>I felt that I was using a lot of nervous energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11.</td>
<td>I was in a state of nervous tension</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12.</td>
<td>I could see nothing in the future to be hopeful about</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13.</td>
<td>I felt that I had nothing to look forward to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14.</td>
<td>I found myself getting upset rather easily</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15.</td>
<td>I had difficulty in swallowing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16.</td>
<td>I felt that life was meaningless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17.</td>
<td>I felt that life wasn’t worthwhile</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18.</td>
<td>I had a feeling of shakiness (e.g., legs going to give way)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19.</td>
<td>I experiencing trebling (e.g., in the hands)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20.</td>
<td>I felt I was pretty worthless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>21.</td>
<td>I found myself getting upset by quite trivial things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>22.</td>
<td>I found myself getting agitated</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>23.</td>
<td>I felt I wasn’t worth much as a person</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>24.</td>
<td>I was worried about situations in which I might panic and make a fool of</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>myself</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25.</td>
<td>I felt that I had lost interest in just about everything</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>26.</td>
<td>I was unable to become enthusiastic about anything</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>27.</td>
<td>I tended to over-react to situations</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>28.</td>
<td>I found that I was very irritable</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>29.</td>
<td>I found myself in situations which made me so anxious I was almost</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>relieved when they ended</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30.</td>
<td>I feared that I would be “thrown” by some trivial but unfamiliar task</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>31.</td>
<td>I couldn’t seem to experience any positive feelings at all</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>32.</td>
<td>I couldn’t seem to get any enjoyment out of the things I did</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>33.</td>
<td>I felt I was close to panic</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

---

Please continue to the next page.
34. I felt that I was rather touchy 0 1 2 3
35. I felt terrified 0 1 2 3
36. I just couldn't seem to get going 0 1 2 3
37. I was intolerant of anything that kept me from getting on with what I was doing 0 1 2 3
38. I felt scared without any good reason 0 1 2 3
39. I had a feeling of faintness 0 1 2 3
40. I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic lights, being kept waiting) 0 1 2 3
41. I found it difficult to work up the initiative to do things 0 1 2 3
42. I found it difficult to tolerate interruptions to what I was doing 0 1 2 3

### Part 5: Effect of Pain on Life Activities

This part is designed to measure whether your pain is preventing you from doing what you would normally do, or from doing it as well as you normally would. Think about the **overall impact of your pain**, not just when the pain is at its worst, by circling a number between 0 and 10, where 0 means no disability at all and 10 means your activities are totally disrupted or prevented by your pain.

1. **Family / home responsibilities**
   - This category refers to activities related to the home or family, including chores or duties around the house (e.g., yard work) and errands or favours for other family members (e.g., driving the children to school).

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

2. **Recreation**
   - This category includes hobbies, sports, and other similar leisure time activities.

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

3. **Social activity**
   - This category refers to participating in activities with friends and acquaintances (other than family members), including parties, theatre, concerts, dining out, and other social functions.

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

4. **Occupation**
   - This category refers to activities that are a part of or directly related to your job. This includes non-paying jobs as well (e.g., house-wife or a volunteer worker).

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

5. **Sexual behaviour**
   - This category refers to the frequency and quality of your sex life.

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

7. **Life support activity**
   - This category refers to basic life-supporting behaviours such as eating, sleeping and breathing.

<table>
<thead>
<tr>
<th>No disability</th>
<th>Total disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>
Part 6: Information about the EVENT that resulted in your injury / pain

1. Did your pain begin as a result of a specific injury, accident, incident or event?  
   Yes   No
2. During that event:
   a. Was someone else physically injured?  
      Yes   No
   b. Did you think that your life was in danger?  
      Yes   No
   c. Did you think that someone else’s life was in danger?  
      Yes   No
   d. Did you feel helpless?  
      Yes   No
   e. Did you feel terrified?  
      Yes   No

Below is a list of problems that people sometimes have after experiencing a traumatic event. In relation to the EVENT that resulted in your current injury / pain, please indicate whether, and if so how often, each problem listed below has bothered you in the past month, by circling a number between 0 and 3 using the following scale:

- 0 = not at all or only one time
- 1 = once a week or less / once in a while
- 2 = 2 - 4 times a week / half the time
- 3 = 5 or more times a week / almost always

<table>
<thead>
<tr>
<th>Problem</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Having upsetting thoughts or images about the event that came into your head when you didn’t want them to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Having bad dreams or nightmares about the event</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Reliving the event, acting or feeling as if it was happening again</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Feeling emotionally upset when you were reminded of the event (for example, feeling scared, angry, sad, guilty, etc)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Experiencing physical reactions when you were reminded of the event (for example, breaking out in a sweat, heart beating fast)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Trying not to think about, talk about, or have feelings about the event</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Trying to avoid activities, people or places that remind you of the event</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Not being able to remember an important part of the event</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Having much less interest or participating much less often in important activities</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. Feeling distant or cut off from people around you</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. Feeling emotionally numb (for example, being unable to cry or unable to have loving feelings)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. Having trouble falling or staying asleep</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. Feeling irritable or having fits of anger</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. Having trouble concentrating (eg, drifting in and out of conversations, losing track of a story on television, forgetting what you read)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16. Being overly alert (for example, checking to see who is around you, being uncomfortable with your back to a door, etc)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17. Being jumpy or easily startled (for example, when someone walks up behind you)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

18. How long have you experienced the problems that you reported on this page? (Please circle one)
   1. less than 1 month
   2. 1 to 3 months
   3. 3 to 6 months
   4. more than 6 months

19. How long after the event did the problems on this page begin? (Please circle one)
   1. less than 6 months
   2. 6 or more months

Thank you very much for filling in the questionnaire.
Please bring it with you to the first research appointment.
APPENDIX G

LETTERS APPROVING ETHICS PROTOCOLS FOR STUDY 2, 3 & 4
Dear Ms Barker,

Protocol 2002/166
The impact of psychological trauma on adjustment to physical injury and chronic pain

On behalf of the Human Research Ethics Committee I am pleased to advise that the above protocol has been approved as per the attached Outcome of Consideration of Protocol. Please note that as a formality this approval is subject to formal ratification by the Committee at its next meeting on 29 November 2002.

For your information:
1. Under the NHMRC/AVCC National Statement on Ethical Conduct in Research Involving Humans we are required to follow up research that we have approved. Once a year (or sooner for short projects) we shall request a brief report on any ethical issues which may have arisen during your research and whether it proceeded according to the plan outlined in the above protocol.
2. Please notify the Committee of any changes to your protocol in the course of your research, and when you complete or cease working on this project.
3. The validity of this current approval is five years' maximum from the date shown on the attached Outcome of Consideration of Protocol form. For longer projects you are required to seek renewed approval from the Committee.

Yours sincerely,

Sylvia Deutsch
Secretary, Human Research Ethics Committee
On behalf of the Human Research Ethics Committee,

I approve/do not approve the above protocol.

Approval is subject to the following conditions:

Reasons for non-approval:

Review due: .......................... Date: 13/11/02

Chairperson: (Professor Hilary Charlesworth)
8 January 2003

Ms Belinda Barker
PhD student
School of Psychology
Faculty of Science
The Australian National University
ACT 0200

Dear Ms Barker,

Protocol 2002/166
The impact of psychological trauma on adjustment to physical injury and chronic pain
Variation: Payment of $10.00 per hour for participants

I am pleased to advise that the above variation to the above protocol, as described in your email to me of 17 December 2002, has been approved by the Chair of the Human Research Ethics Committee Professor Hilary Charlesworth. The approval of the variation will routinely be reported to the Committee at its next meeting.

Please do not hesitate to contact me if you have any queries.

Yours sincerely

Sylvia Deutsch
Secretary, Human Research Ethics Committee
Dear Ms Barker,

Protocol 2003/29
The impact of psychological trauma on adjustment to physical injury and chronic pain.

On behalf of the Human Research Ethics Committee I am pleased to advise that the above protocol has been approved as per the attached Outcome of Consideration of Protocol. Please note that as a formality this approval is subject to formal ratification by the Committee at its next meeting on 30 May 2003.

For your information:
1. Under the NHMRC/AVCC National Statement on Ethical Conduct in Research Involving Humans we are required to follow up research that we have approved. Once a year (or sooner for short projects) we shall request a brief report on any ethical issues which may have arisen during your research and whether it proceeded according to the plan outlined in the above protocol.
2. Please notify the Committee of any changes to your protocol in the course of your research, and when you complete or cease working on this project.
3. The validity of this current approval is five years’ maximum from the date shown on the attached Outcome of Consideration of Protocol form. For longer projects you are required to seek renewed approval from the Committee.

Yours sincerely,

Sylvia Deutsch
Secretary, Human Research Ethics Committee
# Outcome of Consideration of Protocol

<table>
<thead>
<tr>
<th>Researcher:</th>
<th>Ms Belinda Barker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contact details:</td>
<td>Postgraduate Student, School of Psychology, Faculty of Science</td>
</tr>
<tr>
<td>Protocol No.:</td>
<td>2003/29</td>
</tr>
<tr>
<td>Title:</td>
<td>The impact of psychological trauma on adjustment to physical injury and chronic pain</td>
</tr>
<tr>
<td>Date on application:</td>
<td>1 January 2003</td>
</tr>
<tr>
<td>Date received in Research Services Office:</td>
<td>14 February 2003</td>
</tr>
</tbody>
</table>

On behalf of the Human Research Ethics Committee,

I approve/do not approve the above protocol.

Approval is subject to the following conditions:

- ...
- ...
- ...

Reasons for non-approval:

- ...
- ...
- ...

Review due: 

Chairperson: (Professor Hilary Charlesworth)
**APPENDIX H**

Table H.1 Means (standard deviations) and percentage frequencies for demographic, pain and self-report variables for the PTSD and No PTSD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>PTSD (n = 11)</th>
<th>No PTSD (n = 9)</th>
<th>Statistic(df)</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Females†</td>
<td>36%</td>
<td>89%</td>
<td></td>
</tr>
<tr>
<td>Age in years</td>
<td>39.50 (15.26)</td>
<td>43.71 (15.36)</td>
<td>t(18)=-.11</td>
</tr>
<tr>
<td>% reaching each educational level†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Less than Year 12</td>
<td>9%</td>
<td>22%</td>
<td></td>
</tr>
<tr>
<td>- Year 12</td>
<td>27%</td>
<td>22%</td>
<td></td>
</tr>
<tr>
<td>- TAFE/trade qualification</td>
<td>46%</td>
<td>33%</td>
<td></td>
</tr>
<tr>
<td>- Undergraduate qualification</td>
<td>9%</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td>- Postgraduate qualification</td>
<td>9%</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td>% currently working†</td>
<td>56%</td>
<td>64%</td>
<td></td>
</tr>
<tr>
<td>No. of prior trauma exposures</td>
<td>2.50 (1.72)</td>
<td>2.29 (1.60)</td>
<td>t(18)=.14</td>
</tr>
<tr>
<td>PDS</td>
<td></td>
<td></td>
<td>F(3,16)=5.89**</td>
</tr>
<tr>
<td>- PDS-B Re-experiencing</td>
<td>6.45 (3.67)</td>
<td>1.86 (0.90)</td>
<td></td>
</tr>
<tr>
<td>- PDS-C Avoidance/Numbing</td>
<td>8.30 (3.65)</td>
<td>3.43 (3.31)</td>
<td></td>
</tr>
<tr>
<td>- PDS-D Hyperarousal</td>
<td>9.90 (3.60)</td>
<td>4.71 (2.93)</td>
<td></td>
</tr>
<tr>
<td>- PDS Total</td>
<td>24.65 (9.71)</td>
<td>10.00 (6.68)</td>
<td></td>
</tr>
<tr>
<td>Pain duration (months)</td>
<td>24.50 (18.39)</td>
<td>22.29 (19.96)</td>
<td>t(18)=.20</td>
</tr>
<tr>
<td>No. of medications</td>
<td>1.20 (1.14)</td>
<td>1.29 (1.60)</td>
<td>t(18)=.60</td>
</tr>
<tr>
<td>No. of treatment providers</td>
<td>5.70 (2.21)</td>
<td>4.14 (2.61)</td>
<td>t(18)=1.66</td>
</tr>
<tr>
<td>PSI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline pain NRS</td>
<td>5.63 (2.12)</td>
<td>3.88 (1.47)</td>
<td>t(18)=2.54*</td>
</tr>
<tr>
<td>PDI Total</td>
<td>4.70 (2.78)</td>
<td>2.21 (1.58)</td>
<td>t(18)=3.01**</td>
</tr>
<tr>
<td>PHODA – Frequency of Activity</td>
<td>37.65 (15.83)</td>
<td>17.93 (12.10)</td>
<td>t(18)=3.55**</td>
</tr>
<tr>
<td>PHODA – Fear Rating</td>
<td>.49 (.23)</td>
<td>.37 (.16)</td>
<td>t(18)=1.98</td>
</tr>
<tr>
<td>TSK</td>
<td>32.60 (6.88)</td>
<td>28.43 (4.93)</td>
<td>t(18)=2.43*</td>
</tr>
<tr>
<td>PASS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- PASS Cognitive Symptoms</td>
<td>34.65 (11.25)</td>
<td>23.14 (12.64)</td>
<td></td>
</tr>
<tr>
<td>- PASS Fearful Appraisal</td>
<td>18.00 (8.89)</td>
<td>13.86 (12.27)</td>
<td></td>
</tr>
<tr>
<td>- PASS Escape and Avoidance</td>
<td>21.45 (9.62)</td>
<td>17.86 (13.45)</td>
<td></td>
</tr>
<tr>
<td>- PASS Physiological Symptoms</td>
<td>21.10 (11.55)</td>
<td>12.43 (9.29)</td>
<td></td>
</tr>
<tr>
<td>- PASS Total</td>
<td>95.20 (33.70)</td>
<td>67.29 (41.35)</td>
<td>t(18)=2.46*</td>
</tr>
<tr>
<td>DASS</td>
<td></td>
<td></td>
<td>F(3,16)=3.56*</td>
</tr>
<tr>
<td>- DASS Depression</td>
<td>17.00 (11.14)</td>
<td>7.43 (5.68)</td>
<td></td>
</tr>
<tr>
<td>- DASS Anxiety</td>
<td>12.80 (10.26)</td>
<td>9.57 (8.02)</td>
<td></td>
</tr>
<tr>
<td>- DASS Stress</td>
<td>18.90 (10.24)</td>
<td>16.71 (10.42)</td>
<td></td>
</tr>
<tr>
<td>- DASS Total</td>
<td>48.70 (29.35)</td>
<td>33.71 (21.64)</td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .05; **p < .01.

†Chi-square analyses were not computed due to insufficient cell counts (i.e. <5).
APPENDIX I
STUDY 4 QUESTIONNAIRES

PVAQ

Please indicate how often each statement was true of you over the past two weeks, on a scale ranging from 0 (never) to 5 (always):

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I am very sensitive to pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>2. I am aware of sudden or temporary changes in pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>3. I am quick to notice changes in pain intensity.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>4. I am quick to notice effects of medication on pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>5. I am quick to notice changes in location or extent of pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>6. I focus on sensations of pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>7. I notice pain even if I am busy with another activity.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>8. I find it easy to ignore pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>9. I know immediately when pain starts or increases.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>10. When I do something that increases the pain, the first thing I do is check to see how much pain was increased.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>11. I know immediately when pain decreases.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>12. I seem to be more conscious of pain than others.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>13. I pay close attention to my pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>14. I keep track of my pain level.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>15. I become preoccupied with pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>16. I do not dwell on pain.</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX J
STUDY 5 QUESTIONNAIRE

[For Health Professionals]

Please read the case study and then answer the questions below:

[Insert vignette]

1. What is wrong, if anything, with John? (state diagnosis if relevant)

2. What kind of pain do you think John would have?

(tick)

- constant – always present, always the same intensity
- always present, but the intensity varies
- usually present, but with short periods without pain
- often present, but with pain-free periods lasting up to several hours
- often present, but pain free for much of the day
- occasionally present for brief periods, but not every day
- rarely present, pain every now and then, with days or weeks in between

3. How strong do you think John’s pain would be, on average?

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Worst pain imaginable</td>
</tr>
</tbody>
</table>

4. Do you think John needs professional help? Yes / No
5. Do you think the following people would be helpful or harmful if John went to see them?

<table>
<thead>
<tr>
<th>Person</th>
<th>Helpful</th>
<th>Harmful</th>
<th>Neither</th>
</tr>
</thead>
<tbody>
<tr>
<td>A GP or family doctor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A pharmacist or chemist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A massage therapist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A physiotherapist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A chiropractor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A surgeon</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A medical specialist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>An osteopath</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A pain clinic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A counsellor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A social worker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A telephone counsellor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A psychiatrist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A clinical psychologist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A mental health nurse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Close friends and family</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naturopath or herbalist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clergy, minister or priest etc</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>John tries to deal with it on his own</td>
<td>helpful</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6. Do you think the following medications would be helpful or harmful for John?

<table>
<thead>
<tr>
<th>Medication</th>
<th>Helpful</th>
<th>Harmful</th>
<th>Neither</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamins &amp; minerals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herbal remedies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Analgesics (pain-killers)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-inflammatory agents</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-convulsants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedatives/Hypnotics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-psychotic agents</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-anxiety agents</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
7. Do you think the following activities or therapies would be helpful or harmful? (please circle)

<table>
<thead>
<tr>
<th>Activity</th>
<th>Helpful</th>
<th>Harmful</th>
<th>Neither</th>
</tr>
</thead>
<tbody>
<tr>
<td>Becoming more physically active</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoiding physical activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading self-help books</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time off work</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Getting out more and more</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Courses on relaxation (relaxation, stress management, meditation, yoga)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cutting out alcohol all together</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occasional alcohol to relax</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Counselling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive-behavioural therapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychodynamic psychotherapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed rest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinal manipulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical therapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular exercise /Gym</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Injections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acupuncture</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8. How soon after the accident would you expect John to return to work?

(tick)

- The next day
- <1 week after the accident
- <2 weeks after the accident
- <1 month after the accident
- <3 months after the accident
- <6 months after the accident
- <12 months after the accident
- More than 12 months after the accident
- Never
We need to know some brief information about you:

Sex:   Female / Male
Age:   _____ years
Occupation:  ___________________________

How many patients with chronic pain (pain > 3 months) have you treated in the past year?

_____  
About what percentage of your practice is this?  
%  

What percentage of patients with chronic pain had pain due to:

Sudden traumatic injury   ___%  
Sudden non-traumatic injury  ___%  
Gradual onset   ___%
Please read the case study and then answer the questions below:

1. What is wrong, if anything, with John?

2. What kind of pain do you think John would have?

   (tick)
   
   ___ constant – always present, always the same intensity
   ___ always present, but the intensity varies
   ___ usually present, but with short periods without pain
   ___ often present, but with pain-free periods lasting up to several hours
   ___ often present, but pain free for much of the day
   ___ occasionally present for brief periods, but not every day
   ___ rarely present, pain every now and then, with days or weeks in between

3. How strong do you think John’s pain would be, on average?

   0 1 2 3 4 5 6 7 8 9 10
   No pain
   Worst pain imaginable

4. Do you think John needs professional help?  Yes / No

5. Do you think the following people would be helpful or harmful if John went to see them?

   (please circle)
   
   o A GP or family doctor
   o A pharmacist or chemist
   o A massage therapist
   o A physiotherapist
   helpful    harmful    neither
A chiropractor  helpful harmful neither
A surgeon  helpful harmful neither
A medical specialist  helpful harmful neither
An osteopath  helpful harmful neither
A pain clinic  helpful harmful neither
A counsellor  helpful harmful neither
A social worker  helpful harmful neither
A telephone counsellor  helpful harmful neither
A psychiatrist  helpful harmful neither
A clinical psychologist  helpful harmful neither
A mental health nurse  helpful harmful neither
Close friends and family  helpful harmful neither
Naturopath or herbalist  helpful harmful neither
Clergy, minister or priest etc  helpful harmful neither
John tries to deal with it on his own  helpful harmful neither

6. Do you think the following medications would be helpful or harmful for John?

- Vitamins & minerals  helpful harmful neither
- Herbal remedies  helpful harmful neither
- Analgesics (pain-killers)  helpful harmful neither
- Anti-inflammatories  helpful harmful neither
- Anti-convulsants  helpful harmful neither
- Antidepressants  helpful harmful neither
- Antibiotics  helpful harmful neither
- Sedatives/Hypnotics (Sleeping pills)  helpful harmful neither
- Anti-psychotic agents  helpful harmful neither
- Anti-anxiety agents / Tranquilizers  helpful harmful neither

7. Do you think the following activities or therapies would be helpful or harmful?

- Becoming more physically active  helpful harmful neither
- Avoiding physical activity  helpful harmful neither
- Reading self-help books  helpful harmful neither
- Time off work  helpful harmful neither
- Getting out more and more: helpful, harmful, neither
- Courses on relaxation: helpful, harmful, neither
  (relaxation, stress management, meditation, yoga)
- Cutting out alcohol all together: helpful, harmful, neither
- Occasional alcohol to relax: helpful, harmful, neither
- Counselling: helpful, harmful, neither
- Cognitive-behavioural therapy: helpful, harmful, neither
- Psychodynamic psychotherapy: helpful, harmful, neither
- Hypnosis: helpful, harmful, neither
- Surgery: helpful, harmful, neither
- Bed rest: helpful, harmful, neither
- Spinal manipulation: helpful, harmful, neither
- Physical therapy: helpful, harmful, neither
- Regular exercise/Gym: helpful, harmful, neither
- Injections: helpful, harmful, neither
- Acupuncture: helpful, harmful, neither

8. How soon after the accident would you expect John to return to work?
   (tick)
   - The next day
   - <1 week after the accident
   - <2 weeks after the accident
   - <1 month after the accident
   - <3 months after the accident
   - <6 months after the accident
   - <12 months after the accident
   - More than 12 months after the accident
   - Never
We need to know some brief information about you:

Sex:     Female / Male
Age:     _____ years

Level of Education:
Year 9 or less     Year 10-11     Year 12     Trade/TAFE     Undergraduate     Post-graduate

Have you ever been injured in a car accident?       Yes / No
Have you ever had chronic pain (more than three months)?       Yes / No
Do you know anyone like the person in the case study?       Yes / No
APPENDIX K

VIGNETTES USED IN STUDY 5 QUESTIONNAIRE

[PTSD Vignette]

John is 30 years old. Six months ago he was involved in a car accident on his way home from work, when another car failed to give way at an intersection. Just before impact, John thought to himself “this is it”. He was trapped in the car for a short time after. He could smell petrol and was worried the car might explode. John was taken to hospital and X-rays showed he hadn’t broken any bones.

Since the accident, John has had pain in his neck, shoulders and lower back. His wife says he is irritable and gets angry very easily. He has stopped most of the activities he used to enjoy and prefers to be on his own rather than with other people. Since his injury, he is having trouble concentrating, even on simple tasks, and is more forgetful than usual. Even though he is tired, he can’t get to sleep at night. He usually tries to stay up as late as possible until he can’t keep his eyes open anymore. He often wakes up during the night in a sweat and finds it difficult to get back to sleep because of his pain. He feels on edge and is more jumpy than usual. Sometimes he finds he is thinking about the accident for no reason at all and he doesn’t like talking about it. He used to enjoy driving but now he avoids it as much as he can and takes a different route home from work. When he is driving he is “on the lookout” and his wife complains that he is more of a “back-seat driver” than ever before. If someone pulls out in front of John, he worries about having another accident and his heart beats fast, he breaks out in a sweat and he has trouble catching his breath. He complains that his pain is worse after driving longer distances. Before the accident he had clear goals about the future but now he takes it day by day.
[No PTSD Vignette]

David is 33 years old. Six months ago he was involved in a car accident on his way home from work, when another car hit him from behind while he was waiting to enter a roundabout. Dave remembers hearing the sound of screeching tyres just before the impact. He was taken to hospital and X-rays showed he hadn’t broken any bones.

Since the accident, Dave has had pain in his neck, shoulders and lower back. His wife says he is more tense and irritable than he used to be. He has stopped many of the activities he used to enjoy and is less tolerant when he is around other people. Since his injury, he is having trouble concentrating, even on simple tasks, and is more forgetful than usual. He is not getting much sleep and he feels tired and lacking in energy most of the time. He often wakes up during the night and finds it difficult to get back to sleep because of his pain. He spends a lot of his time watching television or resting on the couch. Dave is more wary of roundabouts and complains that his pain is worse after driving longer distances, but otherwise his driving has not been affected by the accident.
Dear Canberra Resident,

I am conducting a survey of knowledge about health problems, treatment and outcomes as part of a PhD research project at the ANU. In particular, I am interested in comparing similarities and differences between the public and health professionals’ level of knowledge and beliefs about how people adjust to and manage a physical injury, pain and related problems. The research is designed to investigate knowledge about the nature of problems arising from an injury, the usefulness of different treatments and the likely outcomes for an injured person. This information may assist in developing prevention, early intervention and evidence-based treatment strategies and assist individuals in seeking help for their health conditions.

You have been chosen as a potential participant as a member of the public, selected at random from the ACT Telephone Directory. Your participation is important to provide a good cross-section of the knowledge and beliefs of the general public. Participation in this survey is entirely voluntary and anonymous. Participation simply involves filling in the enclosed questionnaire, which should take no more than 10-15 minutes. It involves reading a short case study and answering some questions. If you decide to participate, please fill in the questionnaire and return it in the reply-paid envelope provided.

You do not need to put your name anywhere on the questionnaire. Results of the survey may be published, however your name, any personal information and your involvement will not and cannot be revealed. In fact, if you return your questionnaire it will be completely anonymous because we have no way of telling who has completed it and, once the surveys have been returned, the list of potential participants will be destroyed. Your questionnaire will be securely stored in a locked filing cabinet. Any information entered into a password protected computer will only be identified by an ID number (that cannot be traced to you). Only the research supervisor and myself will be able to access the filing cabinet and computer data, in so far as the law provides.

If you have any questions or concerns about the research you can contact:
• Belinda Barker, PhD (Clinical Psychology) Candidate, School of Psychology, Australian National University, ACT 0200, phone 0410 629 511, fax 02 6125 0499 or email belinda.barker@anu.edu.au.
• Prof. Don Byrne, Research Supervisor, School of Psychology, The Australian National University, ACT 0200, phone 02 6125 3974 or email don.byrne@anu.edu.au

Otherwise, if you have any concerns about the research, you may contact the Human Research Ethics Committee, which has approved an ethics protocol submitted for this research:
• C/- Sylvia Deutsch, Human Ethics Officer, Research Services Office, The Australian National University, ACT 0200, Phone: 02 6125 2900, Fax: 02 6125 4807 or Email: Human.Ethics.Officer@anu.edu.au

Yours sincerely,

Belinda Barker
Psychologist
B.Sc. (Hons-Psych), LL.B., MAPS
Belinda Barker  
PhD (Clinical Psychology) Candidate, School of Psychology  
The Australian National University ACT 0200  
Ph: 0410 629 511; Fax: 02 6125 0499  
Email: belinda.barker@anu.edu.au

30 August 2004

Dear [Doctor/Chiropractor/Psychologist/Physiotherapist],

I am conducting a survey of knowledge about health problems, treatment and outcomes as part of a PhD research project at the ANU. In particular, I am interested in comparing similarities and differences between the public and health professionals' level of knowledge and beliefs about how people adjust to and manage a physical injury, pain and related problems. The research is designed to investigate knowledge about the nature of problems arising from an injury, the utility of different treatments and the likely prognosis for an injured person. This information may assist in developing prevention, early intervention and evidence-based treatment strategies and assist individuals in seeking help for their health conditions.

You have been chosen as a potential participant as a practicing [general practitioner /chiropractor/ psychologist/ physiotherapist] listed in the [telephone directory/ APA membership directory]. Your participation is important to provide a good cross-section of the knowledge and beliefs of [general practitioners/ chiropractors/ psychologists/ physiotherapists]. Participation in this survey is entirely voluntary and anonymous. Participation simply involves filling in the enclosed questionnaire, which should take no more than 10-15 minutes. It involves reading a short case study and answering some questions. If you decide to participate, please fill in the questionnaire and return it in the reply-paid envelope provided.

You do not need to put your name anywhere on the questionnaire. Results of the survey may be published, however your name, any personal information and your involvement will not and cannot be revealed. In fact, if you return your questionnaire it will be completely anonymous because we have no way of telling who has completed it and, once the surveys have been returned, the list of potential participants will be destroyed. Your questionnaire will be securely stored in a locked filing cabinet. Any information entered into a password protected computer will only be identified by an ID number (that cannot be traced to you). Only the research supervisor and myself will be able to access the filing cabinet and computer data, in so far as the law provides.

If you have any questions or concerns about the research you can contact:
- Belinda Barker, PhD (Clinical Psychology) Candidate, School of Psychology, Australian National University, ACT 0200, phone 0410 629 511, fax 02 6125 0499 or email belinda.barker@anu.edu.au.
- Prof. Don Byrne, Research Supervisor, School of Psychology, The Australian National University, ACT 0200, phone 02 6125 3974 or email don.byrne@anu.edu.au

Otherwise, if you have any concerns about the research, you may contact the Human Research Ethics Committee, which has approved an ethics protocol submitted for this research:
- C/- Sylvia Deutsch, Human Ethics Officer, Research Services Office, The Australian National University, ACT 0200, Phone: 02 6125 2900, Fax: 02 6125 4807 or Email: Human.Ethics.OFFicer@anu.edu.au

Yours sincerely,

Belinda Barker  
Psychologist  
B.Sc. (Hons-Psych), LL.B., MAPS
13. The research aim is to investigate the public and health professionals' level of knowledge and beliefs about how people adjust to and manage a physical injury, pain and related problems. The research is designed to investigate knowledge about the nature of problems arising from an injury, the usefulness of different treatments and the likely outcomes for an injured person.

14. The research involves completing and returning a questionnaire.

15. Participation is completely voluntary and anonymous.

16. Information from my questionnaire will only be identified by an ID number unrelated to me and will be securely stored in a locked filing cabinet and a password protected computer, accessible only by Belinda Barker or her research supervisor. My responses are thus confidential, in so far as the law provides.

17. The results of the study may be published. If so, my involvement and my personal identity will not and cannot be revealed.

18. If I have any questions or problems about the research I can contact:
   - Belinda Barker, PhD Candidate, School of Psychology, Australian National University ACT 0200, phone 02 0410 629 511, fax 02 6125 0499 or email: belinda.barker@anu.edu.au
   - Prof. Don Byrne, Research Supervisor, Australian National University ACT 0200, phone 02 6125 3974 or email: don.byrne@anu.edu.au

19. Alternatively, questions or concerns about the way the research is conducted can be directed to the:
   • Human Research Ethics Committee, c/- Sylvia Deutsch, Human Ethics Officer, Research Services Office, The Australian National University, ACT 0200, Phone: 02 6125 2900, Fax: 02 6125 4807 or email: Human.Ethics.Officer@anu.edu.au

20. If for some reason I experience discomfort or distress from reading or filling in the questionnaire, I understand that I could contact the following organizations to discuss my concerns:
   • Lifeline, 24 hour telephone counselling line, Phone: 13 11 14
   • Psychology Clinic, ANU, Phone: 6125 0412

After considering all these points, I agree that by completing the questionnaire and returning it in the reply-paid envelope I have consented to participate in this survey.
Ms Belinda Barker  
Postgraduate Student,  
School of Psychology  
Faculty of Science  
The Australian National University  
ACT 0200

Dear Ms Barker,

Protocol 2004/196  
Impact of psychological trauma on adjustment to injury and management of chronic pain

On behalf of the Human Research Ethics Committee I am pleased to advise that the above protocol has been approved as per the attached Outcome of Consideration of Protocol.

For your information:
1. Under the NHMRC/AVCC National Statement on Ethical Conduct in Research Involving Humans we are required to follow up research that we have approved. Once a year (or sooner for short projects) we shall request a brief report on any ethical issues which may have arisen during your research and whether it proceeded according to the plan outlined in the above protocol.
2. Please notify the Committee of any changes to your protocol in the course of your research, and when you complete or cease working on this project.
3. The validity of this current approval is five years' maximum from the date shown on the attached Outcome of Consideration of Protocol form. For longer projects you are required to seek renewed approval from the Committee.

Yours sincerely,

Yolanda Shave  
Acting Secretary, Human Research Ethics Committee
Researcher: Ms Belinda Barker
Contact details: Postgraduate Student, School of Psychology, Faculty of Science
Protocol No. 2004/196
Title: Impact of psychological trauma on adjustment to injury and management of chronic pain
Date on application: 1 July 2004  Date received in Research Office: 30 June 2004

On behalf of the Human Research Ethics Committee,

I approve/do not approve the above protocol.

Approval is subject to the following conditions:

Reasons for non-approval:

Review due: ............................................................ Date: 30-8-2004

Chairperson: ......................................................... (Dr Peter Hiscock)
### APPENDIX N

**Table N.1. Percentage frequency (count) of participants’ identification of problems as a function of Vignette and Profession in Study 5**

<table>
<thead>
<tr>
<th>Identified Problem</th>
<th>Vignette</th>
<th>No PTSD</th>
<th>(\chi^2) profession</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PTSD</td>
<td>No PTSD</td>
<td></td>
</tr>
<tr>
<td>Psychological</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None identified</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>0% (0)</td>
<td>8% (2)</td>
<td>14.53**</td>
</tr>
<tr>
<td>Psych</td>
<td>0% (0)</td>
<td>0% (0)</td>
<td>Min E = 4.63</td>
</tr>
<tr>
<td>Physio</td>
<td>17% (2)</td>
<td>15% (2)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>20% (1)</td>
<td>29% (2)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>PTSD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>100% (22)</td>
<td>11% (44)</td>
<td></td>
</tr>
<tr>
<td>Psych</td>
<td>100% (10)</td>
<td>5% (72)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>67% (8)</td>
<td>1% (8)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>60% (3)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>46% (10)</td>
<td>68% (17)</td>
<td>19.71***</td>
</tr>
<tr>
<td>Psych</td>
<td>30% (3)</td>
<td>57% (4)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>33% (4)</td>
<td>15% (2)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>20% (1)</td>
<td>43% (3)</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>32% (7)</td>
<td>8% (2)</td>
<td>8.13*</td>
</tr>
<tr>
<td>Psych</td>
<td>40% (4)</td>
<td>29% (2)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Physio</td>
<td>42% (5)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>40% (2)</td>
<td>14% (1)</td>
<td></td>
</tr>
<tr>
<td>Psychosomatic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>9% (2)</td>
<td>8% (2)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>0% (0)</td>
<td>14% (1)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>0% (0)</td>
<td>8% (1)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>40% (2)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Chronic pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>23% (5)</td>
<td>36% (9)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>50% (5)</td>
<td>57% (4)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>17% (2)</td>
<td>69% (9)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>20% (1)</td>
<td>43% (3)</td>
<td></td>
</tr>
<tr>
<td>Identified Problem</td>
<td>PTSD</td>
<td>No PTSD</td>
<td>$\chi^2$ profession</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------</td>
<td>--------------</td>
<td>---------------------</td>
</tr>
<tr>
<td><strong>Physical injury</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None identified</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>68% (15)</td>
<td>40% (10)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>50% (5)</td>
<td>43% (3)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>42% (5)</td>
<td>8% (1)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>0% (0)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Whiplash</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>18% (4)</td>
<td>24% (6)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>10% (1)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>33% (4)</td>
<td>46% (6)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>20% (1)</td>
<td>29% (2)</td>
<td></td>
</tr>
<tr>
<td>Soft-tissue/muscular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>23% (5)</td>
<td>24% (6)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>10% (1)</td>
<td>43% (3)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>25% (3)</td>
<td>23% (3)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>40% (2)</td>
<td>43% (3)</td>
<td></td>
</tr>
<tr>
<td>Spinal/nerve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP</td>
<td>5% (1)</td>
<td>16% (4)</td>
<td>Min E &lt; 5</td>
</tr>
<tr>
<td>Psych</td>
<td>20% (2)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Physio</td>
<td>8% (1)</td>
<td>15% (2)</td>
<td></td>
</tr>
<tr>
<td>Chiro</td>
<td>20% (1)</td>
<td>57% (4)</td>
<td></td>
</tr>
</tbody>
</table>

**Note.** $n$ for each cell is:

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>No PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP</td>
<td>22</td>
<td>25</td>
</tr>
<tr>
<td>Psych</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Physio</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>Chiro</td>
<td>5</td>
<td>7</td>
</tr>
</tbody>
</table>

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