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The Evolution of Chronic Pain:
Adjustment Status Following Treatment For Acute Low Back Pain

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A thesis submitted to the School of Graduate Studies of the
University of Ottawa as partial fulfillment of the requirements
for the degree of Doctor of Philosophy

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Dedication

I would like to dedicate this work to my parents and family, in loving memory of my father, Emile De Gagné, who was unable to share this experience with me. To my mother, Elizabeth De Gagné, who was able to share and support me through my education, I thank you for your devotion to your family, your constant support, your challenging intellect, and encouraging me in all my endeavors. Also, in loving memory of my aunt Hedwig Keukens-De Gagné, who was a constant source of encouragement and demonstrated that people can realize their goals if they believe in themselves. I also would like to dedicate this work to Scott Rafter, who was only with us in graduate school for a short time before he passed away. He remains forever in our thoughts and our hearts.

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Gathering data for this thesis was a combined effort on the part of many individuals. A collective of forty-one low back pain patients gave freely of their time, thoughts, and feelings to aid in our understanding of the developmental nature of chronic low back pain. Without the dedication of the staff at the Workwise Physiotherapy Clinic, Ottawa, Canada, this study would not have been possible. Thanks to Pat Rodriguez, Rhonda Nishio, Denise Chernier, David Follows, and many others at the clinic for their time and commitment. During graduate studies, I was fortunate enough to receive several scholarships and grants. In this regard, I sincerely acknowledge the support of the following granting agencies: Province of Alberta Graduate Scholarship, Ontario Graduate Scholarship, the University of Ottawa School of Graduate Studies, Labatt's Light Relay Fund, and the Social Sciences and Humanities Research Council of Canada.

Abstract

This study examined predictors of continued pain status in a sample of injured workers presenting with acute low back pain with no previous reported injury. Self-reported pain and disability, depressive symptoms, and coping strategies were assessed at admission to a treatment program in an initial sample of 41 injured workers. At discharge, 29 subjects were available for analyses, 27 at 3-months, and 20 at 6-months post injury. Coping and adjustment variables were examined as predictors of continued pain three months following the acute episode of low back pain. A logistic regression analysis using treatment change scores (self-reported disability and depressive symptoms) significantly predicted continued pain and correctly classified 91% of cases. Analyses describing the relationship among post-treatment change variables and continued pain revealed two factors, Diverting Attention, and Active Cognitive Coping, which correctly classified 90% of the cases. Hence, psychological variables measured close to injury onset were related to subsequent adjustment. Descriptive analyses indicated that somatic focus was synchronous with the temporal gradient of pain. Specifically, somatic focus was observed to increase as the pain experience persisted. The correlation among self-reported disability and present pain intensity was greater at discharge compared to admission. These findings indicated that variables impacted upon by treatment may serve as predictors of the development of chronic pain. The role of cognitive-behavioral interventions designed to impact on psychological adjustment variables is discussed.

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Pain plays an integral part of life. Not only does pain perform protective functions, it is the primary symptom that prompts individuals to seek medical attention and can greatly compromise the quality of life (Turk & Melzack, 1992). The view adopted by the International Association for the Study of Pain (IASP; 1986, p. S217) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." Such a definition reflects the multidimensional complexity of the experience of pain and emphasizes the psychological phenomenology of pain. This emphasis, in particular, speaks to the role of experience in the context of developing a set of thoughts, feelings, beliefs, and behaviors in response to pain (Eimer, 1989).

The full expression of pain has been linked to the highest level of the central nervous system (Iggo, 1987; Wall, 1990). In this regard, particular emphasis has been placed on higher cortical areas that are involved in discriminative and motivational systems (Weisenberg, 1987). Because these systems influence reactions based on cognitive evaluations and past experience, the role of psychological variables has become of tremendous import when assessing patients who present with pain complaints. For example, successful management of enduring pain complaints often involves altering cognitive-motivation components while the sensory component remains intact (Weisenberg, 1987). As noted by Leventhal and Nerenz (1983), the meaning of pain results from its coding or interpretation. Hence, how the patient conceptualizes the pain experience holds as much relevance for the experience of pain as does the physiological event itself. This integration of sensory and cognitive components in the pain experience speaks to the importance of assessing beliefs about pain and their relationship to treatment outcome (Jensen & Karoly, 1991; Jensen and Karoly, 1992; Jensen, Turner, Romano, & Lawler, 1994; Mikail, D'Eon, & De Gagné, 1996; Williams & Thorn, 1989).

Low back pain is considered to be one of the most common disabling conditions for those of working age (Waddell, 1982). The Nuprin Pain Report indicated that 56% of American adults presented with some back pain in the year prior to the survey (cf., Sternbach, 1986). Sternbach estimated that the societal losses in productivity (U.S.) due to the result of pain on full-time employees reached a staggering $55 billion. Frymoyer and Cats-Baril (1987) reported that the cost of compensation and medical payments for work related back disorders to be estimated at $7.2 billion (U.S.).

The Toronto Workers' Compensation Board reported that in 1982, back pain accounted for 28% of work-related injuries and is the most frequently cited reason for compensation. In 1982, Ontario alone processed 41,102 claims for back pain (Lonetto, Kumuchy & Robinson, 1986). The Spitzer (1987) report of the Quebec Task Force on Spinal Disorders revealed that 3,077 claims were related to the back and neck in 1981. Back pain was the most common anatomical site, accounting for 70% of compensated claims. Total compensation costs for spinal disorders in 1981 were $150 million. Of this, $129 million was spent for salary replacement due to work disability, with the remaining $21 million directed toward medical expenses. Workers who remained absent from work after three months had a greater tendency to remain absent for extended periods, thus accounting for a greater percentage of compensation costs. The social consequences related to quality of life and massive loss in productivity related to the development and continuance of back pain call for a continued effort to delineate the nature and course of chronic disabling back pain.

Pain: A Historical Overview

Historically, the investigation of the pain experience has been surrounded by considerable controversy about whether pain belongs to the domain of sensory physiology or to that of perceptual psychology (Chapman, 1978). As a consequence, investigators of the pain experience proposed various theories pertaining to the nature of the experience. Nineteenth century and early 20th century conceptualizations on the nature of pain proposed that a positive relationship existed between the degree of pain and the extent of tissue damage (Barber, 1959). As noted by Bonica (1990a), the specificity and pattern theories of pain were two such theories that embraced the notion that pain was a purely sensory phenomenon.

Specificity theory presented pain as a specific sensation, with its own sensory apparatus independent to touch and other senses (Bonica, 1990). Hence, pain was presented as a unique consequence of excitation of nociceptive afferents (i.e., pain receptors; Wall, 1989). This theory suggested that a specific pain system carried messages from pain receptors in the skin straight through to a pain centre in the brain (Melzack & Wall, 1982). It was hypothesized that large fibers transmitted touch, and small fibers mediated pain impulses in separate, specific, direct pathways to touch and pain centers in the brain (Bonica, 1990). This theory was attractive because it suggested that pain was mediated by nociceptors, whereby, primary pain afferents terminated in the dorsal horn of the spinal cord (Wall, 1989). Three assumptions underlied this model; 1) receptors were specialized, 2) a single specific receptor lay beneath sensory spots on the skin, and 3) each psychological dimension of a sensory experience bears a linear relation to a single stimulus dimension and to a given type of skin receptor.

Alternatively, pattern theory proposed that the pattern of pain was produced by intense stimulation of nonspecific receptors. The major assumptions underlying this theory were that all fiber endings were alike and that all cutaneous sensations were produced by nerve impulses rather than by separate modality-specific transmission routes (Melzack & Wall, 1982).

Subsequent advances in pain research during the 1950s led Melzack and Wall (1965) to reappraise the relevance of previous theories of pain, whereupon, they presented their gate-control theory of pain (Mark I). Melzack and Wall concluded that the specificity theory was strongly supported by physiological evidence of specialization of the nervous system. However, its greatest weakness was the psychologic assumption that sensation is achieved via a fixed direct-line communication from the skin to the brain via a straight-through push-button fashion. Their gate-control model was one of the first theoretical models to incorporate psychological components of the pain experience. Melzack and Wall's multidimensional model of pain integrated sensory discriminative with cognitive or central control and motivational-emotion components. This model proposed that a neural mechanism in the dorsal horn of the spinal cord acts like a gate, modulating the sensation of pain. The gate can increase or decrease the flow of nerve impulses from peripheral fibers to the central nervous system which result in the sensation of pain. This model was later modified (Mark II) to include excitatory and inhibitory links from the substantia gelatinosa to the transmission (T) cells, as well as descending inhibitory control from brain-stem systems (Melzack & Wall, 1982). Thus, pain could be inhibited or exacerbated through higher cortical functioning. Hence, it was proposed that persons could impact directly on their pain experience by effortful strategies designed to control their anxiety levels (e.g., relaxation), and the cognitive appraisals (e.g., catastrophizing, diverting attention) of their pain experience. In this regard, pain perception was not exclusively dependent on the presence of tissue damage. For example, it is possible to experience pain in the absence of tissue damage, such as the occurrence of tension headaches. Conversely, the major tissue damage that occurs in the early stages of cancer is not associated with the presentation of pain (Melzack & Wall, 1982). Gate-Control theory is regarded as the most important development in the field of pain research and therapy (Bonica, 1990).

Categorizing pain. Pain can be categorized into two broad categories. This is based, primarily, on a continuum of duration, rather than on the severity of presentation (Turk & Melzack, 1992). *Acute pain* is typically associated with tissue change or damage, inflammation, or a disease process that is relatively brief in duration (Turk & Melzack, 1992). It is regarded as a temporary warning pain signaling physical changes, injury or disease (Hanson & Gerber, 1990). Examples include post-surgical pain, dental pain, and pain accompanying childbirth (Turk, Meichenbaum, & Genest, 1983). Applied to illness in general, acute and chronic illness represent different conceptual models (Hanson & Gerber, 1990). Leventhal, Zimmerman, and Gutmann (1984) noted that acute-illness models present with four features: the illness can be labeled; is caused by external disease agents; is short-term; and the underlying disease process can be cured with treatment. As noted by Hanson and Gerber (1990), at issue is whether these same assumptions should be applied to chronic pain.

In contrast, *chronic pain* challenges two major assumptions of the acute model (Hanson & Gerber, 1990). The first assumes that the pain is temporary, and the second posits that treatment eliminates symptoms and results in a cure. However, the vast majority of chronic pain patients conceptualize their experience within an acute model (Hanson & Gerber, 1990). Chronic pain is that which persists beyond the normal time of healing (Bonica, 1990) and is typically regarded as pain that will continue throughout an individual's lifetime. The point of division between acute and chronic pain, while having undergone some debate, has been established as three months (IASP, 1986).

Chronic pain can be categorized into several types (Turk, Meichenbaum, & Genest, 1983). *Chronic, periodic pain* is associated with pain syndromes that are characterized by intense episodes of pain interspersed with pain-free periods (e.g., migraine headaches, myofascial pain). The uses of medication, cognitive, and behavioral protocols have been employed to manage the pain experience. *Chronic, progressive pain* is the result of any number of medical conditions and is typically associated with tissue damage (e.g., rheumatoid arthritis, cancer). Cancer is the most common of these conditions that results in pain. Pharmacological approaches are typically used to attenuate the pain experience.

Chronic, intractable, benign pain is more commonly referred to as "chronic pain," according to the definition given above. Such chronic pain is present most of the time and varies in intensity (e.g., low back pain). As noted by Bonica (1990a), the term benign was initially introduced to differentiate between chronic pain associated with cancer, and chronic pain not associated with cancer. However, the notion of pain as benign (e.g., mild), does not truly represent the deleterious impact that chronic pain has on the patient, the family, and society. The term intractable refers to the longstanding presentation of pain that is likely to continue throughout the patient's lifetime.

The etiology of chronic pain is associated with chronic pathologic processes in somatic structures or viscera, which include prolonged dysfunction of the components of the central or peripheral nervous system, or both (Bonica, 1990b). In addition, chronic pain may be caused by psychologic or environmental factors, or both.

Finally, chronic pain may also be viewed as the extent to which it is regarded as an impairment, disability, or handicap. In this regard, the World Health Organization (WHO, 1980) introduced a manual of classification relating to the consequences of disease, whereby, the features of impairment and disability are presented as fundamentally different concepts. *Impairment* is characterized by a loss or abnormality of psychological, physiological, or anatomical structure or function. *Disability* is defined as any restriction of the ability to perform an activity within the expected normal range. Examples of disability include disturbances in personal care (e.g., excretory control) activities of daily living (e.g., sleep, fatigue), and locomotor activities. A *handicap* is a disadvantage resulting from an impairment or a disability, that interferes with the fulfillment of a role that is normal for that person. Hence, a handicap represents the socialization of an impairment or disability, and reflects the cultural, social, economic and environmental consequences that present as a result of the impairment and disability.

Adjustment to Pain

Adjustment is described in the coping and stress literature as psychological well-being, or adaptive mental functioning (Ellis, 1962). Jensen et al. (1994) noted that this definition has been expanded to include multidimensional aspects of interpersonal and intrapersonal functioning that include the ability to carry out normal physical and psychosocial activities of daily living. For example, Lazarus and Folkman (1984) suggested that adjustment comprises three domains: social functioning, morale, and somatic health. More recently, Jensen and Karoly (1991) assessed three related components of adjustment to chronic pain comprising activity level, psychological functioning, and medication/professional service utilization. These findings suggested that adjustment is a multidimensional concept with measurable outcomes and related intervention potential.

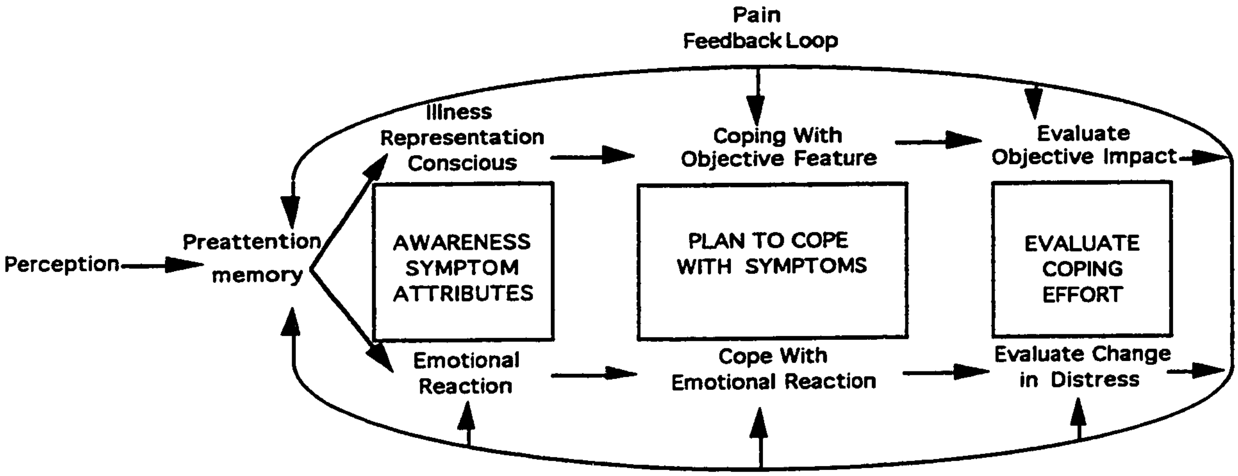
Numerous studies have indicated that patients' beliefs about their pain play a substantive role in their adjustment (e.g., Jensen, Turner, Romano, & Lawler, 1994; Lacroix, 1991; Mikail, D'Eon, & De Gagné, 1996; Williams & Thorn, 1989). Several related, yet distinct, theories have sought to explain the manner in which patients adaptively, or maladaptively, adjust to pain. For example, coping models of pain have been reviewed by Jensen, Turner, Romano and Karoly (1991) that speak to the role of psychosocial variables and adjustment to pain. In their review of the literature, Keefe, Dunsmore, and Burnett (1992) reported that chronic pain patients who remain passive, or utilize strategies that include catastrophizing, ignoring sensations, and diverting their attention to cope with their pain present with increased levels of physical and psychological disability. However, these data are mostly correlational, making it difficult to determine the temporal relationship between coping, pain, and disability. Prospective research that examines the development of chronic pain from acute onset would enable the identification of changes in cognitions that linked with changes in the temporal development of chronic pain.

A number of cluster analytic models have been presented to delineate the important dimensions associated with clinical outcome in chronic low back pain patients (Klapow et al., 1993; Sanders & Brena, 1993). For example, Klapow et al. (1993) used the Symptom Impact Inventory, the MPQ, and the BDI with 96 chronic pain patients to derive three groups. A "Chronic Pain Syndrome" cluster was characterized by high levels of impairment, pain, and depressive symptoms. A second cluster was denoted by high levels of pain, low levels of impairment and depressive symptoms ("Adaptive" cluster). A third cluster comprised individuals presenting with low pain intensity, impairment and depressive symptoms ("Good Pain Control" cluster). Sanders and Brena (1993) identified four clinical subgroups in a heterogeneous sample of 120 chronic pain patients. The results indicated that highly dysfunctional patients presented with greater self-reported depressive symptoms and disability and were less likely to be working prior to rehabilitation treatment. However, patients with moderate levels of physical pathology showed the least amount of improvement following treatment. In summary, the roles of self-reported disability, pain and depressive symptoms, and physical pathology has been significantly linked to poor outcome using these classification schemes.

Models of coping. Several coping models have been presented to explain adjustment to pain that include: 1) operant/ respondent conditioning (Fordyce, 1978); 2) cognitive perception processes such as appraisal of the threat (Lazarus & Folkman, 1984); and 3) self-efficacy and social learning theory (Bandura, 1977). The cognitive-behavioral model, discussed by Jensen et al. (1994) and others (Turk & Meichenbaum, 1989; Turk, Meichenbaum & Genest, 1983; Turner & Clancy, 1988), suggests that the manner in which patients interpret beliefs about their pain plays a substantive role in their subsequent adjustment to their condition. Interventions based on this approach are designed to help patients learn to live more effectively in their environment despite the presence of varying levels of discomfort (Turk & Meichenbaum, 1989). This type of remediation emphasizes patient acquisition of cognitive and behavioral skills for coping with pain (Turner & Clancy, 1988).

The self-regulation model (Leventhal, Meyer, & Nerenz, 1980; Leventhal, Nerenz & Steele, 1984) posits that adjustment to illness danger is related to a person's understanding of his or her condition, and subsequently affects future adjustment-be it adaptive or maladaptive (see Figure 1). This model suggests that there are four levels of processing, against which the individual evaluates changes in physiological status. Emphasis is placed on the major attribute of illness representation: identification, cause, consequence and duration of symptoms. Referring to Figure 1, four assumptions explain the response to a health threat. The initial perception of the stimulus results in the interpretation and reaction to information in the body on a moment to moment basis. This processing is enhanced by the integration of current stimulus information (e.g., pain) with both innate and acquired codes or memories. This processing of moment to moment information organizes experience and behaviors in an episodic fashion.

Figure 1. Hierarchical Model of Symptom Processing: Adapted From Leventhal et al., (1980, 1984).



Subsequent to the activating event, the system divides into two parallel pathways. One pathway (top of figure) involves the activation of objective features of an event (pain) and the subsequent plan to cope with the threat. The second pathway comprises an emotional response to the problem, the development of a plan to cope with the emotion, and evaluate the change in distress. The two pathways interact at the conscious and preconscious level, resulting in adaptation to each situation. Hence, individuals attempt to integrate both the schematic components (of the concrete symptoms) and abstract conceptualizations (labels) to impute the identity of an illness episode. From an acute perspective, illness is thought to be related to emotional considerations. Alternatively, chronicity implies a lack of control over the self and over the illness. This is related to a final acceptance of a permanent, negative change in the self (Leventhal et al., 1984).

Hierarchical processing is the fourth assumption of this model and occurs at both schematic and abstract-conceptual levels. For example, coping with a headache comprises concrete aspects (e.g., headache pain) and conceptual abstractions (e.g., I am having a stroke) that occur during the representation, coping and appraisal stages. In the case of low back pain, a pain activates the schema, and the conceptual abstraction leads to a label (e.g., a bone chip is rubbing my spine; muscle tension). This hierarchical system enables the possibility of consistency or inconsistency between the concrete and abstract levels. For example, a patient may be treated for low back pain with an exercise regime to maximize function and improve tolerance to activity. Before treatment, the patient may have been quite sedentary and avoided any activity that would exacerbate his or her symptoms. The patient is told that he or she has made significant improvement, however, the patient reports feeling worse (pain has not subsided) during and after treatment, than prior to treatment. The information that the patient is improving is inconsistent with concrete experience. This inconsistent information activates schematic processes that provide a dynamic and self-regulating function (Markus & Wurf, 1987). These processes guide the processing of new information and retrieval of stored information (Fiske & Linville, 1980). These templates allow the person to carry out active searches for symptoms in the relevant body system and to reify existing hypotheses about causes and consequences of illness. They also serve to resist information in favor of reifying a hypothesis about the symptom event. This may set the stage for maladaptive processes.

In the above example, ambiguous information also would be processed as relevant to the existing schema (Markus, 1977). As reported by Leventhal et al. (1984) emotional responses appear to be related to concrete processing or the automatic combination of stimuli with perceptual memories (Leventhal, 1984). Hence, ongoing pain may be interpreted as tissue damage, and previously learned responses may inhibit more adaptive functioning, or acceptance of a permanent change in the self, as noted above.

Edwards and Pearce (1994) reported that schematic processing of pain information is clearly present in chronic pain patients. Their results indicated that chronic pain patients produce significantly more pain-related word completions than non-patient controls. In the context of a self-regulation model, this would suggest that selective information processing may play a role in the maintenance of health problems (cf., Edwards & Pearce, 1994). Edwards and Pearce (1994), investigated the schematic representation of pain information in a sample of chronic pain patients attending an outpatient rheumatology clinic. Patients were asked to complete pain related three-letter word stems. This methodology was reported elsewhere to establish the existence of schematic processing that invokes dynamic, rather than passive responding (e.g., Markus 1977, 1990). In this paradigm, it is hypothesized that schematic individuals have an elaborated, well-articulated cognitive structure that enables them to process relevant information about themselves quickly and efficiently. Hence, faster reaction times for schematic relevant information are usually taken as evidence for schematic representation of the construct under investigation. In word completion tasks, it is hypothesized that chronic pain patients will engage in a selective recall of pain-related information. Such a method has been criticized for the reactivity of a limited set of stimulus sets. Thus, respondents examine the self based on organizations developed by the experimenter—arguably not self-knowledge. Deutsch, Kroll, Weible, Letourneau, and Goss (1988) noted that this reactive procedure permits untapped traits to be left unaddressed. Mood-congruent endorsement of checklist items, which mimics features of schematic information processing, has been linked to validation problems in this procedure (Safran, Segal, Hill, &

Whiffen, 1990). Edwards and Pearce (1994) reported that chronic pain patients completed more pain-related word stems than control groups comprising community volunteers and mental health professionals. Hence, it was argued that the content of chronic pain patients' schema was related to the specific pain experiences of the individual.

Self-regulatory beliefs about the extent to which a person takes responsibility for improvement of functioning, and control over symptoms also has been implicated in the literature as markers of adjustment to pain (e.g., Lawson et al., 1990; Leventhal , Meyer, & Nerenz; Turk, Rudy, & Salovey, 1986). In this regard, Leventhal and his colleagues (Leventhal et al., 1980; Leventhal & Nerenz, 1983) reported that persons attempt to make sense of physical symptoms because such experiences represent a threat to one's state of well-being. Hence, people develop common-sense representations of illness that are influenced by self-regulatory mechanisms referred to as implicit models, or schemata. The importance of understanding patients' implicit model of their illness has been researched by a variety of investigators (Lacroix, 1991; Lacroix et al. 1990, Lacroix et al., 1991; Leventhal & Nerenz, 1983; Leventhal et al., 1980; Mechanic, 1962). For example, through structured interviews, Leventhal and colleagues explored implicit models that patients hold about their illness. This work, buttressed by that of Lau and Hartman (1983) and Lau, Bernard, and Hartman (1989), suggested that schemata of illness include five basic components, in addition to the reported symptoms: label, consequences, timeline, cause, and cure. Schober and Lacroix (1991) reported the invariance of these components in their historical analysis of illness schemata of patients from the 17th and 18th century. Turk, Rudy and Salovey (1986) also reported similar findings, citing evidence of a four-dimensional structure of illness comprising seriousness, personal responsibility, controllability, and changeability.

Pain and self-reported disability. The relationship between control over pain and self-reported disability also has been noted in previous research (Jensen & Karoly, 1991; Jensen & Karoly, 1992; Strong, Ashton, Cramond, & Chant, 1990). Several predictor variables have consistently shown their relatedness to chronic pain that include the use of self-regulating coping styles (Jensen et al, 1991; Rosenstiel & Keefe; Lawson et al., 1990), disability status (Block, Kremer, & Gaylor, 1980; Fordyce, 1976; Roland & Morris, 1983), the duration of pain (Hinkley

& Jaremko, 1994), number of previous lumbar surgeries (Keefe, 1982), perceived control over pain (Strong et al., 1990; Toomey, Mann, Abashian, Carnrike, & Hernandez, 1993; Toomey, Mann, Abashian, & Thompson-Pope, 1991), and personality variables that include somatization (Fordyce, 1976; Sternbach, Wolf, Murphy, & Akeson, 1973).

In a study of 50 chronic pain patients, Strong et al. (1990) reported that attitudes, perceived ability to control symptoms, and beliefs about pain were related to self-reported disability. Consistent with the previous literature, Jensen and Karoly (1992) reported that patients' self-reported ratings of disability were correlated with low levels of psychological functioning. Furthermore, they noted that patients' belief in themselves as disabled was inversely related to activity level for patients reporting low and medium levels of pain severity. These data supported the hypothesis that implicit beliefs about chronic pain were associated with multidimensional aspects of adjustment to pain. The authors emphasized the importance that these beliefs have on one's perception of being disabled by pain. Roland and Morris (1983) reported that high self-reported disability at presentation to primary care among low back patients was significantly related to poor outcome. Their results suggested that a continuation of self-reported disability (scores of 14 or more) four weeks following presentation defined poor outcome. Hinkley and Jaremko (1994) argued for the importance of early psychological intervention in a group of patients presenting with pain for 7.3 months. In their sample of 635 orthopedic patients, the authors reported that patients with longer pain duration presented with higher pain intensity, less effective coping ability, and greater psychological disturbance.

Pain and depression. A number of investigators in recent years have reported that a large percentage of chronic pain patients experience symptoms of depression or are diagnosed with major depression (Dworkin & Gitlin, 1991; Romano & Turner, 1985; Sullivan & D'Eon, 1990; Sullivan, Reesor, Mikail, & Fisher, 1992; Ward, 1990). Independent of a specific diagnosis, the prevalence of depressive symptomatology in chronic pain patients has also been reported in epidemiologic studies to be between 13% and 20% (Boyd & Weissman, 1982). In a review of the literature, Romano and Turner (1985) cited a number studies reporting prevalence rates ranging from 10% to 100%. Dworkin and Gitlin (1991) summarized the results of 29 studies that examined the prevalence of current major depression and dysthymia among chronic pain patients diagnosed by the DSM-III criteria. These investigators reported that the prevalence of concurrent depressive disorders varied widely from 1.5% to 57.1%. Factors contributing to these discrepancies were purportedly related to; 1) studies that examined heterogeneous samples of chronic pain patients; and 2) the type of clinical setting in which patients were recruited. Despite these incongruencies, the presence of concurrent depressive episodes was, on average, substantial (M = 24%).

In a study of 758 patients presenting with six chronic illnesses (arthritis, depression, diabetes, cancer, renal dermatologic) Cassileth et al. (1984) reported that psychological adaptation among these patients improved after the initial three month diagnostic period. Turner and Romano (1984) hypothesized that the course of depression in chronic pain may manifest in rapid onset with a slow dissipation over 3 to 5 years. Von Korff, Le Resche, and Dworkin (1993) have recently called for longitudinal research in this area. Hence, the prospective evaluation of the development of chronic pain would be able to address whether change in depression status follows, or is synchronous with, change in pain status. Such results are of great import as Sullivan et al. (1992) noted that depressive affect has been a neglected topic in the management of low back pain.

The pain-depression link. In an attempt to clarify existing conceptualizations of the pain-depression link in pain patients, several theories regarding etiology have been noted in the literature. For example, Rudy, Kerns and Turk, (1988) reported that the search to identify concomitants of the pain-depression link has resulted in three general etiologic theories. Nonspecific biologic models posit that similar neurochemical mechanisms may contribute to both depressive disorders and chronic pain (Ward, et al., 1982). Blumer and Heilbronn (1982) have suggested that it is depression which causes the pain complaint. Specifically, chronic nonspecific pain is regarded as a variant of mood disorder. Finally, depression has also been explained as an understandable secondary reaction to a chronic disability (Hendler, 1984; Rudy et al., 1988; Kleinman, 1988).

In an attempt to further delineate the extant nature of depression as a secondary reaction to chronic pain, several investigators have proposed a cognitive-behavioral mediation model. To summarize, this model proposes that depressive symptoms arise from a sustained reduction in instrumental activities and related decreases in social rewards. As noted by Rudy et al. (1988), the basis for the cognitive-behavioral mediation model is derived from substantive research on the contribution of cognitive and behavioral factors related to the pain experience.

In the last ten years, two published studies have used causal modeling techniques to address weaknesses in previous correlational research on the pain-depression link. Rudy et al. (1988) used covariance structural modeling (LISREL-V) to test the hypothesis that cognitive-behavioral variables mediated the link between pain and depression. These investigators found that the direct link between pain and depression was non-significant. However, measures of perceived life interference and self-control were reported as significant moderators between pain and depression. Brown (1990) used a prospective cross-lagged design to test the hypothesis that pain caused depression in a sample of patients diagnosed with rheumatoid arthritis. Results supported a causal model in which pain predicted future depressive affect. This is one of the first studies to directly examine the temporal relationship between pain and depression and suggested that chronic pain is a cause rather than a consequence of depressive affect. Recently, De Gagné (1995) used covariance structural modeling (LISREL-VII) to summarize the findings of these two studies within one conceptual framework in a large sample of chronic pain patients ($\underline{N} = 306$). This model predicted that the pain-depression link is mediated by perceived interference and spousal support. These data also supported a significant direct path from pain to depressive affect.

Returning to work. As noted by Lacroix et al. (1990) and Fishbain et al. (1993), recovery from low back pain has been linked not only to physical indicators, but also to psychosocial factors. Exclusive reliance on physical examination to determine level of disability has not been empirically demonstrated to predict return to work (Gallagher et al., 1989). However, the importance of psychosocial concomitants of low back pain and self-reported pain (Fishbain et al., 1993) have been used to predict work status in a number of studies.

In a retrospective study of low back pain patients, Lacroix et al. (1990) reported that orthopedic evaluations of severity and prognosis did not significantly predict return to work. Results indicated that the number of nonorganic physical signs, scales 1 and 3 of the MMPI, and age failed to predict work status. Conversely, the accuracy of the patients' understanding of their medical condition as measured by the Schema Assessment Inventory (Lacroix, 1991) significantly correlated with return to work.

Fishbain et al. (1993) summarized the findings of 26 studies examining return to work variables in patients with low back pain. These authors indicated that no organic factors have been found to be predictive of return to work and only a few demographic characteristics seem relevant (e.g., higher pre-injury income). Job satisfaction was inconsistently related. Conversely, perceptions of, and control over pain, self-reported disability, affect, self-reported seriousness of condition, compensation, and litigation variables were reported to be related to return to work status. Although all 26 studies had a follow-up component, only three utilized a three-month follow-up and the others ranged from six months to one-year follow-up points. In addition, it appears that the definition of acute pain was inconsistent with at least one study defining acute pain as pain of less than seven months in duration. Given the guidelines set forth by the IASP (1986), chronic pain is described as pain that persists beyond three-months. Hence, the temporal impact (e.g., from pain onset to seven-months) that these variables had on return to work, from a truly acute state, remain unclear in these analyses.

In a recent study by Dozois, Dobson, Wong, Hughes, and Long (1995), the authors conducted a nine-month follow up of 117 male injured workers with chronic low back pain, who were seen at a work hardening program. Discriminant function analysis was used to predict return to work and correctly classified 79% cases based on psychological variables at admission. Predictor variables included coping strategies, psychological distress, and pain intensity.

<u>The developmental nature of chronic pain</u>. There is a paucity of studies that have prospectively examined psychological concomitants in the development of chronic pain. In two recent studies, Philips and her colleagues (Philips & Grant, 1991; Philips, Grant, & Berkowitz, 1991) evaluated the development of chronic back pain in 117 acute back pain patients. The investigators noted a substantial decrement in pain components (intensity, sickness impact, and disability) across a 6-month period. These decrements were not observed with pain cognitions, which remained relatively stable in individuals who developed chronic pain. Alternatively, the

disability index increased between the three and six month periods (Philips & Grant, 1991). These results suggested that the crucial interval constituting the greatest change in pain report lies between onset and three months. Philips and Grant (1991) reported that the initial presentation of maladaptive cognitions represented a constant for acute pain patients who later developed chronic pain. This is interesting in light of the work by Lacroix et al. (1991), who has noted that inaccurate symptom schemata are inversely related to an individual's general level of adaptive functioning.

Philips et al. (1991) reported that pain cognitions were significantly more maladaptive for those reporting pain at three-months than those who did not. Similarly, acute pain patients did not differ in reported pain cognitions at three-month and six-month intervals. Although not addressed further by Philips and her colleagues, it was reported that patients who went on to develop chronic pain, also reported cognitions that were related to exacerbating their experience (Philips, 1989). Specifically, the manner in which patients conceptualize their symptoms has been linked to adaptive functioning (Lacroix, 1991). For example, cognitive appraisals (beliefs and expectations) associated with chronic pain are argued to maintain the patient's preoccupation with bodily symptoms. Hence, it would be important to evaluate the role such beliefs have on patient functioning across the temporal gradient from acute to chronic stages of pain.

In a recent cross-sectional study, Hadjistavropoulos and Craig (1994) found that acute and chronic incongruent pain patients presented with similar characteristics. These characteristics included greater disability, catastrophizing cognitions, passive coping, and stronger emotionality. The term “incongruent pain” was defined by Reesor and Craig (1988) as a behavioral pattern that is not in keeping with expected medical findings on medical examination. Hadjistavropoulos and Craig (1994) suggested that acute pain patients who fail to cope with the initial injury through the use of passive coping strategies, and who become emotionally distressed, may continue this pattern and develop an excessive focus on pain. These patients constitute a group of individuals potentially at risk for the development of persistent pain and may be the most appropriate candidates for intervention strategies. Hinkley and Jaremko (1994) have argued for the delivery of early interventions in the treatment of pain complaints. In their sample of 635 orthopedic patients, they reported that duration of the pain complaint was related to increases in pathology.

Research Summary and Study Focus

The extent to which adjustment, or pain coping strategies, differ as a function of self-reported pain intensity, disability, affect, and pain duration have been identified as important areas of interest for research and clinical interventions (Jensen & Karoly, 1992; Jensen, Turner, & Romano, 1991; Lawson, Reesor, & Keefe, 1990; Rosenstiel & Keefe, 1983). Taken together, the aforementioned models suggest that interventions at the level of beliefs, coping strategies, affect, and behaviors related to pain may serve to impact on adjustment to injury. Furthermore, investigation of the natural unfolding of these behaviors, cognitions, and affects, at a point close to injury onset may lead to a better understanding of the course and severity of the development of chronic pain.

A number of psychological factors have been linked to successful return to pre-injury levels of functioning in acute and chronic pain populations (e.g., coping styles, depressive symptoms, self-reported pain, and self-reported disability). What is less clear, is the extent to which variables that have been shown to improve post-treatment in an acute population of low back pain patients also may be predictive of future functioning. Dozois et al. (1995) utilized such a strategy in a sample of chronic pain patients and reported that the same psychological variables affected by treatment, also were predictive of successful return to work. It would seem appropriate that the importance of these variables also be addressed in an acute pain population to determine the extent to which variables that predict continued pain, are also those that are affected by treatment. It follows that these treatment change variables would be important markers for clinical intervention and future research regarding the enhancement of subsequent functioning, and further indicate potential markers of the development of chronic pain status.

The aim of the present study was to investigate acute pain patients from a longitudinal perspective, and to examine correlates and predictors of continued pain. Specifically, how patients perceive and evaluate physiological change during a six-month period following injury was examined. The study of patients' symptom appraisal and resulting adjustment to injury was guided by Leventhal et al's (1980) self-regulation model of coping with health threats. This theory, as applied to the current study and previous research, speaks to the importance of assessing cognitive coping strategies such as diverting attention, cognitive coping, self-efficacy, depressive symptoms, self-reported disability, and personal responsibility as indicators of successful outcome of treatment protocols designed to maximize the physical functioning of persons who are injured. Hence, the study proposed that these implicit dimensions of illness, sometimes referred as illness schemata, were related to the subsequent development of a chronic condition and also to work status.

As noted by Leventhal (1984) many patients utilize concrete symptoms to guide their coping. Hence, the choice for measures used in this study was guided by the extent to which they allowed for the evaluation of implicit information on labeling effects by comparing coping, self-reported disability, depressive symptoms and pain ratings, both within and across acute and chronic pain categories. For example, use of the Implicit Models of Illness Questionnaires (IMIQ; Turk, Rudy, & Salovey, 1986) places importance on defining the problem instead of a complete focus on coping alone. The measurement of self-reported disability and pain, depressive affect and coping, and somatic focus, alongside problem definitions that include responsibility and control, speak to a more multidimensional representation of the evolution of the chronic pain experience. Rosenstiel and Keefe (1983) reported that higher coping scores were related to poorer outcome and higher pain levels. Hence, the Coping Strategies Questionnaire (Rosenstiel & Keefe, 1983) was considered useful to track the emotional appraisal of symptoms across a temporal gradient. Finally, Roland and Morris, (1983) reported that high disability scores, reported by low back pain patients at presentation to primary care, were significantly related to poor outcome. It was expected that similar results would be evidenced with the use of this scale in the current study. Thus, variables were chosen that would delineate the impact of the label as well as the symptom.

A mixed between and within-subjects design was used to examine the development of chronic pain and return to work in acute low back pain patients for six-months following injury. Patients completed a number of self-report inventories designed to assess implicit models of illness, symptom accuracy, depressive symptoms, coping strategies, disability, and job satisfaction. The primary advantage this prospective approach had over cross-sectional and retrospective studies is that the various dimensions were examined as they developed from the acute to the chronic stage of pain. Utilization of the six-month temporal gradient allowed for the examination of potentially large within-subject variability in pain status over time, resulting in an increased understanding of the spectrum of acute and chronic pain both across persons and within the same individual across time (Von Korf, 1992). As noted by Von Korff (1992), longitudinal research provides essential new information about the natural history of chronic pain and serves to identify factors regarding its associated development and maintenance.

Hypotheses were modified based on the ratio between number of variables to be analyzed and obtained group sample size, as well as previous research, theoretical, and practical meaningfulness of the variable combinations. For example, previous research was examined to determine the appropriate combination of variables used for analyses that would replicate as well as extend earlier work. An effort was maintained to create linear combinations of variables that did not include both adjustment and coping variables as recommended by Jensen et al. (1991). The Coping Strategies Questionnaire (Rosenstiel & Keefe, 1983) was analyzed using three primary factors (cognitive coping, diverting attention, and self-efficacy) as proposed by Lawson, Reesor, Keefe, and Turner (1990). The Schema Assessment Inventory failed to provide useful data, was difficult to administer, and was difficult for patients to complete. Given these limitations, this measure was not included in the following analyses. Specific hypotheses were as follows:

1. It was predicted that variables significantly affected by treatment also would be predictive of chronic pain at three-months. Based on previous research (Roland & Morris, 1983; Fishbain et al., 1993; Dozois et al., 1995) it was expected that self-reported disability and depressive symptoms would demonstrate a significant relationship with chronic pain status. Hence, treatment change scores were subjected to a logistic regression analysis. Minimal change in self-reported disability and continued depressive symptoms were hypothesized to positively predict continuing pain.

2. It was predicted that self-reported pain, disability, and depressive symptoms at admission would classify acute and chronic group membership using a logistic regression. Thus, it was hypothesized that those patients who later developed continuing pain would report greater levels of pain, disability, and depressive symptoms when they were assessed at admission compared to those whose pain dissipated over time.

3. It was predicted that implicit illness variables would classify chronic pain status. Hence, a logistic regression was performed using the IMIQ-severity, responsibility, and control scores. It was hypothesized that increased severity, decreased responsibility, and decreased control would classify those patients who developed continuing pain.

4. It was predicted that CSQ cognitive coping and diverting attention change scores subjected to a logistic regression would classify acute and chronic pain status. Thus, it was hypothesized that acute pain patients would be classified by exhibiting greater declines in the use of cognitive coping and attentional diversion strategies at discharge compared to patients who later developed chronic pain.

5. It was predicted that catastrophizing and self-efficacy would differ across and within groups at admission and 3-months. A MANOVA with one between-subjects (acute vs. chronic pain) and one within-subjects variables (Time 1, Time 3) was conducted on the catastrophizing and self-efficacy subscales of the CSQ. Main effects for group and time were anticipated. A group by time interaction also was anticipated. It was hypothesized that chronic pain patients would present with more catastrophizing cognitions and less perceived self-efficacy at Time 3 than acute patients.

6. It was predicted that changes in self-reported disability and depressive affect at discharge would predict acute and chronic pain status. A logistic regression was performed on the

RMDQ and BDI change scores. It was hypothesized that acute pain patients would report with greater reductions in self-reported disability compared to patients who later developed chronic pain. Depressive affect was hypothesized to remain higher at discharge for patients who later developed chronic pain compared to acute patients.

7. Using a descriptive analysis, it was predicted that severity and course of the pain complaint would be similar to that reported by Philips and Grant (1991). Accordingly, it was hypothesized that somatic symptom endorsement, as measured by the PILL (Pennebaker, 1982), would increase across the temporal gradient for patients who continued to present with ongoing pain symptoms, and that this endorsement would be related to depressive symptoms. It was also hypothesized that chronic pain patients would perceive themselves as having less personal responsibility and control than their acute counterparts, and that chronic pain patients also will perceive their symptoms as more serious and less changeable. This was expected to lead to greater frustration over time as measured by increased catastrophizing.

Method

Subjects

During sample recruitment, 130 patients were contacted through an introductory letter. Of these, 87 declined to be contacted for the study, 2 were not accepted due to their report of receiving treatment for a psychiatric condition, and the remaining 41 agreed to enter the study. Hence, a total of 41 acute low back pain patients, with no previous reported injury, were recruited from the Workwise Physiotherapy Clinic in Ottawa, Canada. Data were collected from consecutive admissions, over a 22-month period (October, 1994 to July 1996). Inclusion criteria included demonstration of English reading ability as indicated by a minimum grade eight reading level. Persons presenting with an unresolved previous injury, chronic pain, or those who reported a psychiatric condition were excluded from participation in the study. The initial sample comprised 15 women (Mean age = 39 years; SD = 11.8) and 26 men (Mean age = 37 years; SD = 10). Mean duration of time to enter clinic from time of injury for the entire sample was 14 days. Mean number of days from entry in program to discharge was 31.2 (SD = 19.04).

Twelve subjects dropped out of the study by discharge. Two subjects were withdrawn from follow-up treatment due to psychiatric conditions. The data from two additional subjects were not analyzed at the three-month period due to a comorbid condition and intermittent pain. The twelve subjects who dropped out of the project after discharge did not differ from participants who continued on the basis of variables observed in the study. Analyses of variance were used to examine all dependent variables across the three groups (acute, chronic, and drop out). The only significant difference found was for the IMIQ-Control. On the IMIQ-Control, individuals in the drop out group reported having less control than the acute pain group. Visual inspection of group means suggested that those who dropped out of the study scored similarly to chronic pain patients on the dependent variables. To compensate for small sample sizes, bonferroni corrections were not applied to these analyses. No significant demographic differences emerged among the groups.

Sample size varied across time resulting in 41 patients available for analysis at admission, 29 at discharge, 27 at three-months following admission, and 20 at 6-months following admission. At three-months, there were initially 29 subjects available for analyses. However, two of these subjects were excluded at this time period based on intermittent pain ratings and development of a comorbid heel spur condition. The resulting sample of 27 subjects comprised 7 acute pain patients (Mean age = 33.1 years; SD = 8.0) and 20 chronic pain patients (Mean age = 38.1 years; SD = 8.7).

Procedure

Participants entering the study were administered the Schema Assessment Inventory (SAI; Lacroix, 1991). In addition to the SAI, the Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982) was also completed. Previous research indicated that providing such a checklist serves to enhance priming of cognitive structures before testing and reduces confounds associated with mood congruent priming (Safran et al., 1990). During the initial assessment, patients were asked if they had a history of a psychiatric condition or were currently under treatment. If the patient was receiving treatment for a psychiatric condition, they were excluded from the study. This occurred on two occasions.

After the initial assessment, therapists completed the SAI on a staggered basis so that completing the data sheets did not conflict with their primary duties. Hence, the SAI was completed immediately after the initial physical therapy assessment, or later that day. A demographic information form (Appendix A) and the remaining questionnaires were given to the patients to complete either on site or over the weekend. Upon completion of the rehabilitation program, each patient was administered a discharge test battery comprising all questionnaires. Completion of the discharge battery required approximately 30 minutes. Confidentiality of client files was assured by using a three digit identification code to assist in matching data across the two testing sessions. Patients who failed to respond by mail were sent reminders by mail or telephone to enhance response rates. Those subjects who completed six-month questionnaires were compensated for miscellaneous expenses with a gift certificate valued at $20.00.

Criteria for establishing chronicity in this population was determined by IASP's (1986) definition of chronic pain. Hence, individuals presenting with pain at the three-month juncture were classified with chronic pain. This classification was verified in three ways. First, using the present pain intensity rating on the McGill Pain Questionnaire, patients reporting pain via this subscale at three-months were classified as chronic. Second, to cross validate the existence of low back pain, the SAI scale also was examined for patient self-reports of low back pain. Finally, the PILL also was examined for endorsement of the low back pain item. Chronic low back pain was classified upon satisfactory delineation across these three measures. The present pain intensity score was not used in analyses to prevent singularity among predictors (cf., Tabachnik & Fidell, 1989). The rehabilitation approach to low back patients at the Workwise Physiotherapy Clinic comprises an active self-management approach designed to maximize function and enhance return to pre-injury employment. Physiotherapists provided all patients with education about their injury, injury prevention, posture, body mechanics, manual therapy, and goal setting for their recovery.

Measures

Coping Strategies Questionnaire (CSQ). The CSQ (Rosenstiel & Keefe, 1983) is comprised of 42 items that measure six cognitive dimensions of coping with pain and two dimensions related to behavioral coping (Appendix B). Using multiple confirmatory factor analyses of the CSQ, Lawson, Reesor, Keefe and Turner (1990) reported three factors that accounted for most of the variance in cognitive coping activity; Cognitive Coping, Self-efficacy, and Externalized Coping. Two subscales were reported as being factorially independent; catastrophizing and increased behavioral activity. Jensen, Turner, and Romano (1992) reported that the use of individual scale scores of the CSQ, as opposed to composite scores, provided greater interpretability regarding potential washing out of combined variable effects. Factorial validity is considered the most robust test of scale construct composition (Anastasi, 1982; Gorsuch, 1983, 1990).

Schema Assessment Instrument (SAI). The SAI (Lacroix, 1991) is comprised of a series of five questions designed to assess patients' implicit models of their illness (Appendix C). Through a series of stepwise probings, the investigator is provided with a working model of: 1) the patient's presenting physical and psychological symptoms and the potential relatedness among these symptoms; 2) the patient's conceptualization (understanding) of his or her medical condition(s) and prognosis; and 3) the extent to which the patient understands the relationship between his or her medical condition and the presenting symptoms. On the basis of this information, the patient organizes his or her symptoms into a series of clusters referred to as "assessed" schemata.

Alternatively, an "expected" cluster is derived by reorganizing, if necessary, the patient's symptom list in terms of the groupings that would be expected on the basis of strict medical, orthopedic, psychological and other available assessments. Ratings based on medical evidence and the discrepancy between the assessed and expected clusters are scored in terms of seven-point scales. Medicalratings pertain to the severity of the presenting condition and to prognosis. Discrepancyratings are applied on the basis of: 1. the degree to which symptoms groupings concur (Differentiation); 2. whether the content of each of the assessed clusters concurs with medical and psychological evidence (Content); 3. whether the patient's understanding of the cause of each cluster fits with the medical and psychological evidence (Etiology); and 4. an overall rating which takes into consideration the patient's understanding of the condition (Global). Hence a total of six ratings may be derived as follows: Severity, Prognosis, Differentiation, Content, Etiology, Global. Combined, these scales measure the accuracy of the patient's illness schemata. Patients with low Global scores (< 3.0) are described as presenting with a poor understanding of their condition, which is orthogonal to the severity of their diagnoses and prognoses.

Using psychologists, physicians, and graduate students as raters, adequate interrater reliability has been demonstrated with two separate samples (Lacroix, 1991). Interrater correlations ranged from .72 to .99. The internal consistency of the SAI scales was also demonstrated to be adequate by the discrimination of items pertaining to disease aspects from those representative of psychological dimensions (r = -.44 to .05). Hence, this would suggest that the metric of schema accuracy is not simply a reflection of severity of medical condition (Lacroix, 1991).

Pennebaker Inventory of Limbic Languidness (PILL) The PILL (Appendix D) is a 54-item checklist that allows the researcher to assess the frequency of specific physical symptoms and sensations commonly experienced by an individual (Pennebaker, 1982). Higher scores are indicative of a greater number of reported symptoms occurring monthly or more frequently. Based on measures taken from five samples (N = 939) results yielded a mean score of 17.9 (SD = 6.9). Pennebaker (1982) reported adequate reliability estimates and high internal consistency (coefficient alpha = .88) for the PILL. Test-retest reliability (2 months) was .79 in a group of 177 adults. Geisser, Gaskin, Robinson and Green (1993) reported that somatic focus was related to depressive affect in a sample of arthritic and fibromyalgia patients.

Implicit Models of Illness Questionnaire (IMIQ). The IMIQ (Turk, Rudy, & Salovey, 1986) is a 24-item questionnaire designed to assess persons' implicit representations (schemata) of illness (Appendix E). As such, the questionnaire was designed to operationalize constructs based on earlier work by Leventhal and colleagues (Leventhal, Meyer, & Nerenz, 1980; Leventhal & Nerenz, 1983) and by Lau and Hartman (1983) on illness representations such as identity, cause, consequences, time-line, and cure. Respondents endorse items on a 9-point scale, indicating the degree to which they agree or disagree with statements describing a particular illness. Turk, Rudy, and Salovey (1986) reported a confirmatory factor analysis of the IMIQ that supported a four-factor solution. The primary factors (constructs) were 1) Seriousness, 2) Personal Responsibility, 3) Controllability, and 4) Changeability. The factor structure demonstrated adequate reliability, and discriminant validity was obtained through ratings of different illnesses by three experimental groups (Turk et al., 1986). Factorial validity is considered the strongest test of scale construct composition (Anastasi, 1982; Gorsuch, 1983, 1990).

Beck Depression Inventory (BDI). The BDI (Appendix F) is a 21-item self-report inventory that identifies symptoms and attitudes related to cognitive, behavioral, affective and somatic dimensions of depression (Beck et al. 1988; Beck et al. 1961). Items are comprised of four statements rated from zero to three in terms of intensity. Tanaka and Huba (1984) reported a confirmatory hierarchical factor analysis of the BDI on a clinical sample that revealed a general second-order factor of Depression. Three lower-order factors were comprised of Negative Attitude, Somatic Elements, and Performance difficulty. Estimates of the BDI's internal consistency reliability (coefficient alpha) for non-psychiatric populations have ranged from .73 to .92 (mean alpha =.81; Beck et al. 1988). De Gagné & Korol (1996) reported the factorial invariance of the second order hierarchical structure of the BDI across gender in a sample of 400 chronic pain patients.

McGill Pain Questionnaire (MPQ). The MPQ (Melzack, 1975) comprises 20 subclasses of qualitatively and quantitatively ordered pain descriptors (Appendix G). Three interrelated yet distinct dimensions of the pain experience (sensory, affective, and evaluative) are derived from the first 16 subclasses of these descriptors. Lowe et al. (1991) have supported the tripartite discrimination of acute pain into sensory, affective, and evaluative dimensions through multiple-sample confirmatory factor analysis. These and the remaining four sets of descriptors that comprise a miscellaneous dimension, are summed to yield a total pain rating index (PRI) based on the rank values of words chosen. Estimates of the MPQ's (first 16-subclasses of the PRI) internal consistency reliability (coefficient alpha) for two samples of female acute pain patients have ranged from .68 to .70 (mean alpha =. 69) for the sensory dimension and .71 to .81 (mean alpha =.76) for the affective dimension (Lowe et al. 1991). Test-retest reliability of the composite subscales were reported for a sample of 65 chronic low back pain patients (Love et al. 1989). Scale coefficients were reported as follows: Total Pain Rating Index = .83; Sensory subscale = .76; Affective subscale = .78; Evaluative subscale =. 47. Dubuisson and Melzack (1976) reported that the MPQ successfully discriminated among various pain syndromes.

Roland Morris Disability Questionnaire (RMDQ; Roland & Morris, 1983).The RMDQ comprises a subset of 24 items taken from the Sickness Impact Profile (Bergner, Bobbitt, Carter, & Gilson, 1981) that assess a person's ability to engage in activities of daily living while experiencing pain (Appendix H). The RMDQ is sensitive to improvement over time for acute low back pain patients (Roland & Morris, 1983) and has good test-retest reliability over a 6-week period (r = .67 to .77) for heterogeneous groups of chronic pain patients (Jensen et al., 1992). This measure is posited to be predictive of outcome status following treatment (Roland & Morris, 1983).

Minnesota Satisfaction Questionnaire (MSQ-short form) The Minnesota Satisfaction Questionnaire (short-form; Weiss, Dawis, England, & Lofquist, 1967) comprises a subset of 20 items take from the larger 100 item questionnaire and is a measure of a person's self-reported level of job satisfaction (Appendix I). The short-form can be divided into three subscales: Intrinsic

Satisfaction, Extrinsic Satisfaction, and General Satisfaction. The respondent indicates how satisfied he or she is with the reinforcer on his or her present job by endorsing items on a 5-point scale ranging from "Very Dissatisfied" to "Very Satisfied". Median reliability coefficients for the subscales across groups of engineers, electronics assemblers and machinists were as follows: Intrinsic Satisfaction = .86, Extrinsic Satisfaction = .80, and .90 for General Satisfaction. Construct validity for the short form is acceptable and comparable to the longer version which was derived from construct validation studies of the Minnesota Importance Questionnaire.

Results

This section addresses methodological issues and preliminary, descriptive results. Characteristics of the acute and chronic pain groups (as defined at the three-month evaluation), are described over the four testing periods. Correlation matrices of the major variables used to describe the evolution of chronic pain at each of the data collection time points are presented. Finally, variables at acute injury that are predictive of continuing pain are presented.

Data Approach

Correction for multiple analyses. To control for the inflation of Type I error, bonferroni corrections were applied to analyses involving multiple tests. There were no more than two levels to any factor in the multivariate analyses, hence, Type I error corrections for group contrasts following significant interactions were not required. In predictive and correlational analyses, relationships were identified only if they met the .05 alpha criterion. Background variables were examined for invariance across groups and to identify the existence of potential covariates. Hence, no adjustments for Type I error were conducted to analyze demographic characteristics, and alpha was retained at .05. In the event that differences were demonstrated among groups, separate variance estimates for t-tests were utilized as appropriate. MANOVAs were conducted using the default regression approach (SSTYPE-UNIQUE, SPSS Version 5.0). This method is appropriate for unequal numbers of cases in each cell, where all cells are equally important and an effect is adjusted for all other effects in the model (Norusis, 1990; Tabachnik & Fidell, 1989).

Treatment of missing data. In studies examining the unfolding of naturally occurring groups, a small proportion of missing data, for each of a large number of variables may significantly affect analyses. In the current study, data are missing for three reasons. The first includes instances where two respondents failed to answer a small number of questions on the CSQ. The second reason is due to subjects failing to return data questionnaire packages at specified time periods. Thirdly, some participants withdrew themselves from treatment or the study completely. To avoid regression to the mean while analyzing the smaller samples, replacement of missing data with mean substitution was not considered. This resulted in variable sample sizes across time periods.

In the case of subjects for whom low back symptoms resolved post-treatment, the Schema Assessment Inventory could not be completed because it evaluates the presentation of symptoms, not their absence. Hence, this inventory could only be assessed by those individuals who continued to present with low back symptoms. This resulted in limiting analyses of this variable to those individuals who continued to have low back pain. Occasionally, patients and physiotherapists reported that this measure was difficult to complete. Data analyses were conducted with this limitation in mind and relationships, or non-relationships of the SAI, with other variables were interpreted with caution.

Patient reclassification as acute/chronic. With exception of the SAI, all returned questionnaires were completed in full for analyses. As noted in the Subject section, two participants were excluded from the three-month analyses. One female participant reported a comorbid condition (heel spur) at three-months, but reported no back symptoms. Her questionnaires were reportedly completed based on this comorbid condition. At the six-month evaluation, this patient presented as pain free. A second female subject experienced intermittent chronic pain at the 3-month period, and was excluded from Time-3 and Time-4 analyses. This subject was classified as acute, although she was clearly presenting with intermittent chronic back pain. At six-months she reported the persistence of low back pain. One male subject failed to return questionnaires at the three-month period, but completed questionnaires at six-months. This subject was contacted by phone and reported that he presented with chronic low back pain at three-months that continued at six-months. Hence, he was classified as chronic for three-month predictive analyses.

Data analysis strategy. The prospective nature of the study dictated that post-hoc evaluations be performed and are identified. Data analyses were chosen in keeping with their suitability for the constraints imposed by the characteristics of the naturally occurring sample that was obtained. Given the good test-retest reliability estimates of the measures used in this study, it is unlikely that variability of correlations in measures across time reflect measurement error.

Sample size estimates were based on previous studies that have examined symptom schemata, affective, and cognitive dimensions in acute and chronic back pain (cf., Hadjistravropoulos & Craig, 1994; Lacroix, 1990; Philips & Grant, 1991). The choice of recruiting subjects based on a large effect size was derived from previous research employing cross-sectional designs on the domains of interest, which yielded large effect sizes (e.g., Hadjistravropoulos & Craig, 1994; Lacroix, 1990). Effect size choice also was consistent with the notion that larger effect sizes would be more clinically meaningful.

Logistic regression analysis and discriminant function analysis (DFA) address the same question (Tabachnik & Fidell, 1989, Norusis, 1990). Logistic regression is more flexible because it has no assumptions about the distribution of the predictor variables. In logistic regression the predictors do not need to be normally distributed, linearly related, or present with equal variance in each group (Tabachnik & Fidell, 1996). In both regression and DFA, to avoid overfitting the model, the number of predictor variables cannot exceed the sample size of the smallest group (Tabachnik & Fidell, 1996). Unequal sample sizes do not pose any special problems in either technique. Tabachnik and Fidell (1989) reported ratios in discriminant function analysis of five to one that were robust. However, a key advantage to logistic regression over DFA is that highly unequal sample sizes are more proficiently handled by logistic regression (Tabachnik & Fidell, 1996). In this regard, the current data set had a three to one ratio regarding sample size differences. This issue, as well as the small sample size in the acute group, resulted in the decision to utilize a logistic regression approach to accommodate the data. Model fit was interpreted by examining the presence of a significant chi-square when comparing the constant-only model with the Model Chi-Square (i.e., the constant plus all predictors; cf. Norusis, 1990; Tabachnik & Fidell, 1996). This was validated by the evaluation of a non-significant chi-square comparing the combination of all predictors in the model with the perfect model (cf. Norusis, 1990). In this latter case, a non-significant chi-square indicates that the proposed model adequately duplicates the observed frequencies at various levels of outcome. In summary, the data set was found to be statistically appropriate to analyze with logistic regression.

It was of clinical relevance to determine the extent to which changes or gains on the dependent variables at discharge from treatment constituted predictive power regarding chronic pain development outcome (cf. Dozois et al., 1996). Examining change scores is considered an appropriate statistical procedure to address post-treatment clinical changes in light of subjects presenting with similar characteristics prior to treatment (Huck & McLean, 1975; Jamieson, 1994). Change scores compensate for decreases in power for the main effect across time periods when initial scores are not effected by treatment (Huck & McLean, 1975). This procedure has been shown to compensate for the results of non-significant main effects in a repeated measures ANOVA, where the F-value has been shown to be one-half of its true size, which typically results in nonsignificant findings. In this model, the interaction effect has been shown to represent the main effect for group differences and is equal to the one way analysis of change scores. The use of change scores increases the power of the following tests and reduces the number of post-hoc comparisons required. Jamieson (1994) reported that the utilization of a change score method to describe relationships with a third variable was superior to the regression method, and resulted in a reduction of Type I error.

Data transformations. Evaluation of univariate and multivariate outliers, assumptions of linearity, normality, multicollinearity or singularity, and homogeneity of variance-covariance matrices revealed no threat to multivariate analyses. In the context of naturally occurring groups, univariate deviations from normality were tolerated within three standard deviations of the mean and then evaluated for normative relevance. Transformation of the BDI was deemed appropriate due to the significant positive skew of the distribution ($Z = 3.9$), despite its natural and expected occurrence. Hence, a square root transformation was performed to normalize the distribution (cf., Tabachnik & Fidell, 1989). In the current data, a test for gender differences on the BDI was not significant, hence, no correctional analyses were conducted in this regard, $t\ (23.51) = -1.84$, $p > .05$. Strong, Ashton and Stewart (1994) also noted that men and women with chronic low back pain were invariant in their expression of depressive symptoms.

It was observed that female participants scored higher than men on the CSQ Catastrophizing subscale at admission. In a review of the literature, Unruh (1996) reported that women pay more attention to symptoms and respond more aggressively to symptoms of ill heath. They also catastrophize more in response to pain than men (cf., Jensen, Nygren, Gamberale, Goldie & Westerholm, 1994). Unruh also reported that women develop a greater repertoire of coping strategies, whereas, men display more action oriented coping that includes denial and problem focused coping. This difference was not statistically significant, however, descriptive aspects of this observed difference were noted in the discussion as appropriate.

Summary. The data analytic approach took into consideration a number of sample specific characterics. Bonferroni corrections were applied to analyses involving multiple tests with exception to the examination of background variables. Analyses were not modified with mean replacement due to sample size considerations. Two participants were excluded from three- and six-month analytic procedures. One of these participants was excluded due to the presentation of a comorbid condition. The other participant was excluded due to the report of intermittent pain. The logistic regression approach to data analysis was chosen based on evaluation of normality assumptions, the small and unequal sample sizes, previous research, the prospective nature of the data, and the associated power of performing this type of analysis. The calculation of effect sizes was performed to aid in interpretation of the results. The BDI was transformed using a square root transformation due to the positive skew associated with this variable.

Descriptive Results

At three-months, patients were classified as having acute pain, if their pain resolved, and as having chronic pain, if their pain continued as noted earlier. Table 1 describes the acute and chronic groups in terms of their physical and demographic characteristics when they presented at admission for treatment. Results indicated that the 7 acute and 20 chronic pain patients did not differ significantly at admission in terms of physical or demographic characteristics (alpha = .05). However, examination of group means indicated that, on average, the patients with continuing pain initially presented for treatment one week later than the patients whose pain resolved. There were two univariate outliers in this regard. However, neither of these subjects presented with chronic pain during admission for treatment and it was decided to retain them as representative of a naturally occurring clinical presentation. Objective severity of physical condition at admission was not rated differently for individuals whose pain resolved compared to those whose pain did not, $t(13.66) = -.36$, $p > .05$. Mean physiotherapist severity ratings for acute patients ($M = 4.3$; $SD = .95$) and those who later developed chronic pain ($M = 4.5$; $SD = 1.2$) were relatively indistinguishable at admission. As seen in Table 2, a relationship between the RMDQ and time since injury (TSI) was noted, where r (38) = -.27, p =.051. This suggested that higher levels of self-reported disability were reported closer to injury onset. No other significant correlations between TSI and the dependent variables were found.

Tables 2, 3, and 4 represent cross-product correlation matrices of the key variables using pair-wise deletion of data. This strategy was implemented to compensate for missing data related to the SAI, as noted above. These tables also indicate the maximum possible score attainable for each variable. It is noteworthy that the SAI Global score was significantly correlated with RMDQ at admission, r (38) = -.36 p < .012. As predicted, a poor understanding of the condition initially (Time 1) was related to increased reports of disability. However, this relationship failed to reach significance at other time periods. As indicated in Table 4, the relationships among self-reported disability, pain, and depressive symptoms becomes stronger over time, as interpreted by significance values. Higher correlations can be an artifact of smaller sample sizes, hence, significance values were used to measure the strength of association between the variables.

Course of Pain Complaint

The following descriptive analyses collapsed across all groups and examined changes in pain related indices over time. This strategy has been previously used to focus on the course and evolution of the pain complaint, rather than group differences (cf., Philips & Grant, 1991). In this regard, the focus of this perspective is on subjects who continued to present with pain for six-months after admission.

Table 5 depicts mean scores of predictor variables for subjects who present with continuing pain across all four time periods. At discharge from treatment, many subjects reported improvement in their condition. Somatic focus, as measured by the PILL, continued to increase over time compared to admission values. As hypothesized, depressive symptoms and somatic focus were synchronous across the four time periods (see Table 4). The PILL was increasingly significantly correlated with depressive symptoms across the six-month temporal gradient. A slight drop in job satisfaction was observed at discharge. However, this drop failed to continue over the months following discharge. Job satisfaction scores returned to admission values indicating that

Table 1

Percentages And Mean Values of Subject Characteristics by Group

	Group		
Characteristic	Acute (n =7)	Chronic (n = 20)	Statistic
Physical Characteristics	Mean (SD)	Mean (SD)	
Age	37.6 (10.8)	38.12 (11.82)	t (14.4) = -.11
Gender (Frequencies)	male 5 female 2	male 15 female 5	χ^2 (N=27, 1) = .33
Demographic Characteristics			
Time Since Injury (TSI)	13.0 (8.5)	22.0 (19.8)	t (19.46)= -1.57
two outliers removed	13.0 (8.5)	18.5 (14.3)	
Marital Status (%)			χ^2 (N=27, 2) = 3.18
Single	57.1, n =4	20.0, n =4	
Married	42.9, n =3	65.0, n =13	
Divorced	0.0	15.0, n =3	
Education (%)			χ^2 (N=26, 1) = 2.86
high school or less	42.9, n =3	15.0, n =3	
more than high school	42.9, n =3	85.0, n =17	
missing (n =1)	14.3, n =1	0.0	
Work Status Before Injury (%)			χ^2 (N=27, 1) =3.1
Full-time	100.0, n =7	80.0, n =16	
Part-time	0.0	20.0, n =4	
Occupation Type (%)			χ^2 (N=25, 3) =1.1
Professional	42.9, n =3	40.0, n =8	
Manager/Admin	14.3, n =1	25.0, n =5	
Services	0.0	5.0, n =1	
Laborers	42.9, n =3	20.0, n =4	
Missing (n =2)	0.0	10.0, n =2	
Income (%)			χ^2 (N=26, 1) =1.89
0 - 30,000	66.7, n =4	35.0, n =7	
> 30,000	33.3, n =2	65.0, n =13	
missing (n = 1)	14.3, n =1	0.0	

Note: No values were statistically significant with alpha set at .05.

Table 2

Variable Correlations Within Each Time Period: Time 1 Below Diagonal and Time 2 Above Diagonal

	SAI	BDI	DIV	COG	EFF	CAT	CTL	DEC	PPI	DIP	PILL	RMDQ	S	R	CTL	CHG	PRI	SAT	TSI
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1	1	0.05	-0.5	0.26	0.84	-0.54	0.77	0.79	-0.54	-	-0.17	-0.29	-0.3	-0.01	0.72	0.5	-0.5	-0.02	-
2	-0.28	1	0.18	0.18	-0.5	0.38	-0.45	-0.48	0.43	-	0.46	0.47	0.26	0.21	-0.33	0.16	0.46	-0.37	-
3	-0.13	0.03	1	0.56	-0.35	0.75	-0.31	-0.34	0.14	-	0.55	0.14	-0.03	0.08	-0.11	-0.45	0.33	-0.01	-
4	0.04	-0.08	0.47	1	0.11	0.11	0.16	0.04	0.04	-	0.25	0.19	-0.06	-0.08	0.13	-0.05	0.12	-0.04	-
5	0.23	-0.49	0.07	0.5	1	-0.67	0.93	0.92	-0.65	-	-0.35	-0.36	-0.06	-0.13	0.62	0.31	-0.64	0.05	-
6	-0.24	0.57	0.51	-0.1	-0.44	1	-0.6	-0.63	0.26	-	0.61	0.24	0.09	0.27	-0.38	-0.45	0.48	-0.11	-
7	0.11	-0.34	0.1	0.54	0.9	-0.4	1	0.72	-0.6	-	-0.26	-0.22	-0.24	-0.04	0.67	0.29	-0.57	0.06	-
8	0.29	-0.54	0.03	0.38	0.93	-0.41	0.67	1	-0.6	-	-0.39	-0.46	0.14	-0.19	0.48	0.28	-0.61	0.03	-
9	-0.15	0.41	0.28	-0.03	-0.46	0.44	-0.34	-0.48	1	-	0.34	0.38	0.05	-0.18	-0.56	-0.05	0.43	-0.13	-
10	-0.01	-0.14	0.11	0.08	-0.09	0.08	-0.04	-0.14	0.25	1	-	-	-	-	-	-	-	-	-
11	-0.26	0.37	0.36	0.07	-0.24	0.5	-0.29	-0.17	0.3	-0.12	1	0.26	-0.04	0.16	-0.15	-0.05	0.54	-0.05	-
12	-0.36	0.43	0.29	0.01	-0.41	0.52	-0.26	-0.48	0.51	0.28	0.25	1	0.13	-0.06	-0.26	0.18	0.47	-0.04	-
13	0.07	0.02	0.09	-0.17	-0.28	0.37	-0.24	-0.26	0.14	0.33	0.15	0.14	1	0.03	-0.47	0.21	-0.02	0.02	-
14	0.03	-0.15	0.32	0.33	0.41	-0.04	0.32	0.42	-0.11	-0.22	-0.05	-0.23	-0.21	1	-0.05	-0.06	0.02	-0.07	-
15	0.28	-0.27	0.1	0.28	0.45	-0.32	0.29	0.52	-0.17	-0.18	-0.23	-0.43	-0.47	0.38	1	0.1	-0.45	0.26	-
16	0.17	-0.23	-0.09	-0.03	0.27	-0.2	0.18	0.3	-0.22	0.05	-0.27	-0.28	-0.17	0.11	0.46	1	-0.05	0.08	-
17	-0.15	0.57	0.24	-0.04	-0.45	0.59	-0.37	-0.44	0.65	0.23	0.59	0.43	0.25	-0.2	-0.36	-0.29	1	-0.23	-
18	-0.05	-0.31	0.21	0.18	0.17	-0.15	0.17	0.15	-0.03	0.1	0.12	-0.13	-0.11	-0.08	0.15	0.11	-0.21	1	-
19	-0.11	0.03	-0.07	-0.01	-0.15	-0.05	-0.08	-0.09	-0.06	-0.03	-0.09	-0.27	0.07	0.07	-0.03	0.21	-0.18	0.12	1

Note: Time 1 N = 41, If r = .27 then p=.047, one-tailed test). Time 2 N = 28 to 39, If p <.05 then correlations are in plain text

Variable #	Variable Name	Max. Pos. Score
1	SAI Global Score	07
2	Beck Depression Inventory	63
3	Diverting Attention Factor (CSQ)	36
4	Cognitive Coping Factor (CSQ)	36
5	Efficacy	06
6	Catastrophizing (CSQ)	36
7	Pain Control Ability (CSQ)	06
8	Pain Decrease Ability (CSQ)	06
9	Present Pain Intensity (MPQ)	05

Var. #	Vari. Name	Max Score
10	Calendar Days in Program	NA
11	Pennebaker Limpid Languidness (PILL)	54
12	Roland Morris Disability Scale	24
13	Implicit Models of Illness Scale-Severity	09
14	Implicit Models of Illness Scale-Responsible	09
15	Implicit Models of Illness Scale-Control	09
16	Implicit Models of Illness Scale-Changeable	09
17	Pain Rating Index (MPQ)	78
[1]18	Job Satisfaction	95
19	Time Since Injury	60

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[1] A clerical error resulted in the redundant inclusion of item 11. This item was deleted from the total score.

Table 3

Variable Correlations Within Each Time Period: Time3 Below Diagonal and Time 4 Above Diagonal

	SAI	BDI	DIV	COG	EFF	CAT	CTL	DEC	PPI	PILL	RMDQ	S	R	CTL	CHG	PRI	SAT
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	1	-0.14	-0.7	-0.81	-0.61	-0.38	-0.55	-0.24	-0.07	0.11	-0.37	-0.15	0.28	-0.24	0.38	-0.53	-0.29
2	-0.58	1	0.35	0.4	-0.24	0.55	-0.29	-0.01	0.63	0.55	0.44	-0.46	-0.18	-0.27	-0.43	0.64	-0.49
3	-0.12	0.29	1	0.76	0.12	0.8	0.16	0.04	0.09	0.25	0.34	0.15	-0.51	-0.16	-0.42	0.24	0.05
4	0.04	0.51	0.7	1	0.17	0.58	0.21	0.07	0.06	0.34	0.36	0.07	-0.49	-0.12	-0.32	0.28	0.11
5	0.29	-0.39	0.19	0.16	1	-0.22	0.91	0.87	-0.25	0.19	-0.38	-0.15	0.03	0.67	-0.03	-0.35	0.31
6	0.07	0.48	0.43	0.43	-0.4	1	-0.24	-0.14	0.38	0.23	0.38	0.04	-0.34	-0.24	-0.09	0.5	-0.23
7	0.19	-0.32	0.18	0.19	0.94	-0.31	1	0.58	-0.27	0.07	-0.34	-0.06	-0.17	0.48	-0.06	-0.29	0.34
8	0.33	-0.41	0.17	0.11	0.95	-0.44	0.79	1	-0.17	0.29	-0.34	-0.23	0.27	0.73	0.01	-0.33	0.19
9	-0.08	0.37	0.21	0.32	-0.59	0.4	-0.57	-0.54	1	0.4	0.58	-0.43	-0.08	-0.42	-0.3	0.77	-0.65
10	-0.09	0.6	0.17	0.28	-0.34	0.53	-0.25	-0.38	0.41	1	0.29	-0.1	-0.51	-0.15	-0.54	0.32	-0.23
11	-0.01	0.4	0.48	0.48	-0.02	0.25	0.03	-0.06	0.47	0.28	1	0.01	-0.24	-0.55	-0.47	0.67	-0.34
12	0.06	-0.07	0.22	0.28	0.01	0.2	-0.03	0.04	0.23	0.14	0.33	1	-0.41	-0.36	0.1	-0.27	0.27
13	-0.26	0.02	0.15	-0.17	0.1	0.02	0.13	0.07	-0.28	0.03	-0.29	-0.28	1	0.41	0.47	-0.11	0.09
14	0.05	-0.16	-0.15	-0.1	0.77	-0.55	0.67	0.78	-0.69	-0.16	-0.31	-0.2	0.21	1	0.36	-0.39	0.27
15	0.23	-0.25	-0.33	-0.25	0.34	-0.44	0.23	0.39	-0.49	0.03	-0.25	-0.04	0.17	0.55	1	-0.22	0.18
16	0.09	0.44	0.16	0.32	-0.49	0.51	-0.47	-0.46	0.83	0.51	0.44	-0.01	-0.16	-0.52	-0.25	1	-0.27
17	0.03	-0.15	-0.02	0.03	0.04	0.13	0.03	0.04	0.17	0.2	0.22	0.46	-0.19	-0.09	0.34	0.19	1

Note: Time 3 N = 26 (n = 19 SAI GLOB).
Time 3 if p < .05, then correlations are **bold**.

Time 4 N = 20, if p < .05 then correlations are in plain text.
SAI Gobal Score n=7. Correlations should be interpretted with caution due to small sample.

Variable #	Variable Name	Max. Pos. Score	Var. #	Vari. Name	Max Score
1	SAI Global Score	07	10	Pennebaker Limpid Languidness (PILL)	54
2	Beck Depression Inventory	63	11	Roland Morris Disability Scale	24
3	Diverting Attention Factor (CSQ)	36	12	Implicit Models of Illness Scale-Severity	09
4	Cognitive Coping Factor (CSQ)	36	13	Implicit Models of Illness Scale-Responsible	09
5	Efficacy	06	14	Implicit Models of Illness Scale-Control	09
6	Catastrophizing (CSQ)	36	15	Implicit Models of Illness Scale-Changeable	09
7	Pain Control Ability (CSQ)	06	16	Pain Rating Index (MPQ)	78
8	Pain Decrease Ability (CSQ)	06	[1]17	Job Satisfaction	95
9	Present Pain Intensity (MPQ)	05			

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[1] A Clerical error resulted in the redundant inclusion of item 11. This item was deleted from the total score.

Table 4

Summary of Table 2 and 3 Correlation Matrices

			MPQ-PRI		
		Admission	**Discharge**	**3-months**	**6-months**
RMDQ					
n=39	admission	.43(.003)			
n=34	discharge		.47(.002)		
n=26	3-month			.44(.013)	
n=20	6-month				.67(.009)

			MPQ-PRI		
		Admission	**Discharge**	**3-months**	**6-months**
BDI TOT					
n=40	admission	.57(.000)			
n=34	discharge		.46(.003)		
n=26	3-month			.44(.012)	
n=19	6-month				.64(.002)

			RMDQ		
		Admission	**Discharge**	**3-months**	**6-months**
BDI TOT					
n=39	admission	.43(.003)			
n=34	discharge		.47(.002)		
n=26	3-month			.40(.021)	
n=19	6-month				.44(.030)

			PILL		
		Admission	**Discharge**	**3-months**	**6-months**
BDI TOT					
n=39	admission	.37(.01)			
n=34	discharge		.46(.003)		
n=26	3-month			.60(.001)	
n=19	6-month				.55(.007)

Note: **MPQ-PRI** = McGill Pain Questionnaire pain rating index, **RMDQ** = Roland Morris Disability Questionnaire, **BDI TOT** = Beck Depression Inventory, **PILL** = Pennebaker Limbid Languidness Questionnaire.

Table 5

Means and Standard Deviations of Dependent Measures Across Four Time Points for Patients With Continuing Pain

	Admission n = 41	**Discharge** n =25	**3-Months** n=20	**6-Months** n=13
Severity of Condition	4.7 (1.3)			
Prognosis	6.1 (1.1)			
SAI Weeks in prog.	3.9 (1.7)			
SAI weeks to heal	5.6 (3.8			
% litigating	0%	0%	n=1 (WCB)	
Days in Program	31.2 (19.0)			
Dependent Measures				
PPI	2.8 (1.1)	2.4 (1.5)	2.1 (.97)	2.3 (1.4)
MPQ-PRI	25.0 (13.6)	22.9 (16.4)	20.6 (12.9)	27.5 (12.9)
RMDQ	15.8 (4.9)	8.4 (6.0)	7.1 (5.1)	6.1 (4.7)
BDI	9.1 (6.9)	7.9 (6.5)	8.7 (6.6)	8.1 (9.6)
IMIQ-S	7.0 (.87)	6.9 (.98)	7.2 (1.4)	6.6 (1.1)
IMIQ-R	3.4 (1.0)	4.3 (1.4)	4.1 (1.4)	4.5 (1.5)
IMIQ-C	4.5 (1.3)	5.3 (1.5)	4.9 (1.6)	4.4 (.97)
IMIQ-CH	6.5 (2.1)	6.5 (2.1)	6.5 (1.7)	5.4 (1.65)
SAI GLOB	5.3 (1.5)	4.7 (2.4)	4.4 (1.8)	4.2 (1.3)
CSQ CAT	9.1 (8.1)	6.7 (7.4)	4.6 (4.3)	9.8 (9.2)
males	6.9 (7.5)	3.7 (2.7)	5.0 (4.5)	8.2 (10.1)
females	12.1 (8.5)	11.2 (9.8)	3.6 (4.4)	10.0 (8.6)
CSQ CTL	3.2 (1.1)	4.0 (1.5)	3.4 (1.0)	3.6 (1.7)
CSQ DEC	2.9 (1.3)	3.6 (1.2)	3.2 (1.2)	3.2 (1.5)
EFFICACY	2.9 (1.0)	3.9 (1.2)	3.3 (1.1)	3.3 (1.4)
PILL	11.8 (8.8)	13.8 (9.7)	12.6 (9.2)	16.8 (11.97)
SAT	73.7 (13.3)	68.9 (16.5)	73.7(10.1)	74.0 (14.4)

Note: **PPI** = McGill Pain Questionnaire present pain intensity, **MPQ-PRI** = McGill Pain Questionnaire pain rating index, **RMDQ** = Roland Morris Disability Questionnaire, **IMIQ** = Implicit Model of Illness Questionnaire (R= responsibility, S = Severity, C = Control, CH = Change), **SAI GLOB** = Schema Assessment Inventory Global Score, **CSQ** = Coping Strategies Questionnaire (CAT = Catastrophizing, CTL = ability to control pain, DEC = ability to decrease pain, EFFICACY = average rating of CTL and DEC combined), **PILL** = Pennebaker Limbid Languidness Questionnaire, **SAT** = Job Satisfaction.

participants, on average, were satisfied with their employment. At the six-month period, job satisfaction was negatively correlated with depressive symptoms, r (17) = -.49, p = .02.

At three-months, a steady leveling off of adjustment measures were presented (see Table 5). Interestingly, compared to admission values, men tended to catastrophize more about their condition than women. There was a slight elevation in BDI scores at Time-3, and slight decreases in understanding of the pain condition (SAIGLOB). The reductions in pain levels and depressive symptoms were not large from a clinical perspective.

Six-months following injury, the participants with continuing pain reported levels of pain, somatasizing, and catastrophizing that exceeded admission values. In contrast, gains made at discharge for IMIQ-Control were lost at six-months, and an overall steady decline in the understanding of their condition (SAIGLOB) was reported by patients at this time. The BDI indicated greater variability (i.e., larger standard deviation) at six-months, compared to previous time periods. At six-months, participants with continuing pain reported the first indication of a decline in the extent to which they felt their condition was changeable.

In summary, as hypothesized, the descriptive evaluation of the course of the pain complaint revealed an overall decline in self-reported disability and depressive symptoms from admission to discharge to three-months. An increasing focus on somatization, compared to admission levels as measured by the PILL, across all time periods also was observed. At the six-month time period, both catastrophizing and pain increased above admission levels, for those individuals with continuing pain. There was an expected increase at discharge regarding self-efficacy, however, this leveled off at the six-month period.

Taking into consideration that correlation values increase as sample sizes decrease, significance values were examined. As seen in Table 4, the MPQ-PRI, and the BDI were significantly correlated at six-months. Pain and disability, and disability and the BDI, were more significantly correlated at discharge.

Treatment discharge status. The extent to which subjects demonstrated an improvement in functioning from admission to discharge was examined. A series of one-tailed pairwise t-tests were performed and corrected for Type I and Type II error with alpha set to .004 (.05/12 = .004). The choice for this correction was obtained by balancing the number of tests performed with Type II error. One-tailed tests were performed based on the a priori assumption that treatment would not result in iatrogenic effects. Hence, scores on dependent variables were expected to remain constant or indicate relative improvement at discharge. As shown in Table 6, six variables of interest demonstrated a statistically significant improvement at discharge compared to admission values. The SAI Global score did not reach significance in the analyses.

A significant improvement in disability status, pain ratings, depressive symptoms, responsibility, and catastrophizing thoughts about the pain experience at program completion was demonstrated. Clients reported a significant increase in their ability to control their pain symptoms (i.e., self-efficacy). There was a tendency toward an increase in feelings of control over their condition as measured by the IMIQ C-scale, however, this failed to reach significance after correcting for multiple comparisons ($p < .006$).

Change scores. In the current design, change scores were calculated from pre- and post-treatment scores, subjected to paired t-tests, and then corrected for multiple comparisons. The variables chosen were related to the hypotheses proposed. The significant change scores, which reflect gains following treatment, were used to classify groups in a logistic regression analysis (cf., Dozois et al., 1995). This approach is comparable to a split plot design with one within and one repeated measures factor. While an alternative strategy would be to use pre-treatment scores as covariates and conduct multiple ANOVAs, this approach would not reflect the more interesting question of group classification that is related to clinical intervention markers.

Concomitants of chronic pain development. Table 6 lists variables that were significantly changed from program admission to program completion. The MPQ-PRI, BDI, RMDQ, Self-Efficacy, CSQ-Catastrophizing, IMIQ-Responsibility were chosen for further analysis because they were both statistically significant and clinically meaningful.

Table 6

Pairwise Tests of Mean Differences Between Admission and Discharge Variables

Variable	Admission	Discharge	df	Significance
RMDQ	15.9 (4.6)	7.7 (6.1)	32	p < .0001
IMIQ-R	3.3 (.93)	4.5 (1.3)	33	p < .0001
IMIQ-C	4.5 (1.3)	5.3 (1.5)	33	p < .006
IMIQ-S	7.1 (.86)	7.0 (.91)	-	ns
CSQ EFFICACY	2.9 (1.1)	3.9 (1.2)	27	p < .0001
CSQ CAT	9.4 (8.3)	6.4 (7.1)	32	p < .004
CSQ COGCOP	13.4 (4.7)	12.6 (6.3)	-	ns
CSQ DIVERT	13.05 (7.3)	11.5 (7.5)	-	ns
MPQ-PRI	25.9 (13.5)	20.1 (17.6)	33	p < .004
BDI	9.4 (7.3)	7.0 (6.3)	33	p < .004
PILL	12.1 (9.1)	12.0 (9.5)	-	ns
SAI	5.2 (1.6)	4.7 (2.4)	-	ns (n=17)

Note. All tests are one-tailed. Bonferroni adjustment (.05/12) = .004.
RMDQ = Roland Morris Disability Questionnaire, **IMIQ** = Implicit Model of Illness Questionnaire (R= Responsibility, S=Severity, C=Control), CSQ = Coping Strategies Questionnaire (CAT= Catastrophizing, COGCOP= Active Cognitive Coping Factor, DIVERT= Diverting Attention Factor, EFFICACY = ability to decrease and control pain, **MPQ-PRI**= McGill Pain Questionnaire Total Pain Rating Index, **BDI**=Beck Depression Inventory total score, **PILL** Pennebaker Limbid Languidness Questionnaire, **SAI** = Schema Assessment Inventory Global Score.

Admission Variables

Pain, disability and affect. A direct logistic regression analysis of the MPQ-PRI, BDI, and RMDQ using admission scores significantly classified acute and chronic pain groups where, $\chi 2$ (N=27, 3) =11.97, p = .007. These variables combined correctly classified 89% of cases. Examination of group means indicated that chronic pain patients presented with greater self-reported depressive symptoms (M =9.1, SD= 6.3) and pain (M = 27.2, SD= 13.0) at admission compared to acute patients (M =4.3, SD=5.1; M = 15.3, SD=11.0), respectively. The difference among self-reported disability levels at admission were negligible between groups. A summary of this regression analysis and those that follow appear in Table 7.

Implicit models of illness The IMIQ-S, -R, and -C scales were subjected to a direct logistic regression analysis to predict group membership (Acute vs. Chronic pain). This relationship was not significant, where, $\chi 2$ (N=27, 3) =2.23, p = >.05.

Coping strategies. Four coping factors were prepared for analysis using a direct logistic regression and MANOVA. Three factors comprising CSQ subscales were determined from previous work (cf. Lawson et al., 1990; Rosenstiel & Keefe, 1983). These factors were: 1) Cognitive Coping (ignoring sensations, coping self-statements, reinterpreting sensations), 2) Diverting Attention (diverting attention, praying or hoping), and 3) Self-Efficacy (control over pain, ability to decrease pain). In addition, the CSQ Catastrophizing subscale was examined. Due to the small number of injured workers whose pain resolved, these factors were analyzed in separate families in order to observe relationships in the data. Contrary to the hypothesis, acute pain patients presented with higher levels of catastrophizing than chronic pain patients. Examination of group means indicated that women presented with higher levels of catastrophizing than men (see Table 8). Given the obtained sample size, decomposition into males and females was not possible. Hence, analyses proceeded using cognitive coping, diverting attention, and self-efficacy dimensions to measure their association with group membership for descriptive purposes.

Table 7

Logistic Regression Analyses of Acute and Chronic Pain Classification

Predictor Variables	Statistic	Significance	Effect Size
Admission Scores			
MPQTOT	χ2 (N =27, 3) = 11.97	p = .0075	**.36**
RMDQ			
BDI			
Admission Scores			
IMIQ-R	χ2 (N =27, 3) = 2.23	p = .52	**.08**
IMIQ-S			
IMIQ-C			
Change Scores			
CSQ Active Coping	χ2 (N =21, 2) =6.83	p = .033	**.36**
CSQ Diverting Attention			
Change Scores			
RMDQ	χ2 (N =22, 2) =6.83	p = .033	**.64**
BDI			

Note. * Small effect size = .10, medium = .25, large = .40 (Cohen, 1992). **BDI** = Beck Depression Inventory total score, **RMDQ** = Roland Morris Disability Questionnaire, **MPQ-PRI**= McGill Pain Questionnaire Pain Rating Index. **CSQ** = Coping Strategies Questionnaire (Active Cognitive Coping Factor; Diverting Attention Factor). **IMIQ-R** (Responsibility), **-S** (Seriousness), **-C** (Control).

Table 8

Group Means And Standard Deviations of Variables Across Time

Measures	Admission			Discharge		
	Acute	Chronic	Dropouts	Acute	Chronic	Dropouts
	n = 7	n = 20	n = 12	n = 7	n = 20	n = 10
MPQ-PRI	15.3 (10.96)	27.2 (13.0)	28.2 (16.3)	10.0 (20.20)	25.2 (14.4)	18.0 (21.5)
RMDQ	16.1 (5.27)	15.1 (5.40)	17.1 (4.72)	2.2 (2.60)	9.2 (6.00)	7.8 (6.70)
BDI	4.3 (5.10)	9.1 (6.30)	11.6 (8.06)	5.4 (7.40)	7.7 (6.70)	6.4 (5.40)
male	2.6 (2.3)	9.3 (7.1)	8.5 (6.3)	0.0 (0.0)	8.0 (7.7)	6.2 (4.1)
female	8.5 (9.1)	8.4 (3.1)	16.4 (8.8)	13.5 (7.1)	6.7 (2.1)	8.0 (7.1)
IMIQ-R	3.9 (1.03)	3.6 (1.07)	2.9 (.88)	5.1 (.88)	4.3 (1.50)	4.3 (1.4)
IMIQ-S	7.1 (1.80)	6.9 (.85)	7.1 (.83)	7.1 (.31)	7.1 (1.00)	6.9 (1.10)
IMIQ-C	5.1 (1.68)	4.8 (1.20)	3.7 (1.05)	5.8 (.79)	5.2 (1.6)	5.3 (1.8)
CAT	8.6 (7.8)	7.0 (7.4)	10.3 (9.02)	5.8 (9.6)	5.2 (4.4)	7.1 (9.2)
male	6.2 (8.2)	6.5 (7.4)	8.8 (8.3)	0.0 (0.0)	4.1 (2.9)	2.8 (2.7)
female	14.5 (.71)	8.6 (8.1)	12.0 (11.5)	11.5 (12.0)	8.5 (7.1)	12.2 (13.4)
DIVERT	17.6 (7.4)	12.05 (6.6)	12.17 (6.7)	8.4 (9.52)	10.9 (7.5)	12.1 (5.9)
COGCOP	14.4 (4.8)	14.33 (4.4)	11.92 (5.3)	5.3 (5.45)	13.9 (5.9)	12.5 (6.2)
EFFICACY	3.8 (1.03)	3.08 (1.19)	2.6 (.97)	4.5 (1.5)	3.7 (.96)	4.3 (1.6)
PILL	6.1 (8.05)	11.4 (7.7)	15.09 (10.13)	8.0 (8.16)	11.2 (8.3)	14.8 (11.7)

Measures	3-Months		6-Months	
	Acute	Chronic	Acute	Chronic
	n = 7	n = 20	n = 5	n =13
MPQ-PRI	2.9 (5.15)	20.6 (12.9)	2.8 (2.7)	19.39 (15.5)
RMDQ	0.4 (1.13)	7.1 (5.1)	1.2 (2.7)	4.5 (4.9)
BDI	2.4 (3.90)	8.1 (6.7)	0.5 (1.0)	7.1 (8.3)
male	0.8 (1.3)	8.9 (7.5)	0.66 (1.2)	8.6 (9.4)
female	6.5 (6.3)	5.8 (3.4)	0.0 (0.0)	3.8 (4.5)
IMIQ-R	4.9 (.93)	4.1 (1.4)	5.5 (1.2)	4.7 (1.4)
IMIQ-S	6.0 (2.17)	7.2 (1.71)	6.9 (.46)	6.8 (.96)
IMIQ-C	5.8 (1.8)	4.7 (1.2)	6.4 (1.2)	4.9 (1.6)
CAT	1.3 (3.27)	4.6 (4.3)	2.3 (3.9)	7.5 (7.9)
male	1.6 (3.6)	5.0 (4.5)	4.0 (5.6)	7.0 (8.5)
female	0.0 (0.0)	3.6 (4.3)	0.5 (.71)	8.5 (7.2)
DIVERT	5.5 (5.1)	8.7 (7.85)	4.4 (5.93)	9.9 (9.0)
COGCOP	5.1 (2.2)	14.3 (7.14)	10.17 (7.2)	11.72 (7.5)
EFFICACY	4.4 (1.2)	3.3 (1.05)	4.0 (1.3)	3.3 (1.4)
PILL	2.1 (2.8)	12.6 (9.2)	5.4 (8.96)	12.8 (11.2)

Consistent with hypotheses, there was a main effect for Time on the 2 X 2 (Time by Group) MANOVA on the catastrophizing dimension F (1,22)= 11.25, $p = .003$. As shown in Table 8, there was an overall reduction in catastrophizing across groups at three-months. Contrary to hypotheses, there were no significant group or interaction effects for self-efficacy.

Discharge Change Scores

A logistic regression analysis was conducted to examine coping treatment change score variables as predictors of chronic pain. The CSQ Coping and Diverting Attention factor change scores were subjected to a direct logistic regression. This analysis resulted in significantly distinguishing groups, $\chi 2$ (N = 21, 2)= 6.83, $p = .03$, whereby, these variables correctly classified 90% of cases. Examination of group means indicated that at discharge, acute pain patients presented with a dramatic decline in the use of cognitive coping strategies (mean difference = 7.9; SD = 8.4) and diverting attention strategies (mean difference = 7.6; SD = 8.3) compared to chronic patients (mean difference = .29; SD = 3.6, and 1.5; SD = 3.7, respectively).

Disability and depressive affect. The role of depressive affect and disability ratings was examined to determine if changes in these variables following treatment were predictive of chronic pain classification (cf. Roland & Morris, 1983). A direct logistic regression of treatment change scores resulted in a significant classification of groups, $\chi 2$ (N = 22, 2)= 6.83, $p = .03$. Changes in self-reported disability as measured by the RMDQ and the BDI correctly classified 91% of cases. Examination of group means indicated that acute pain patients displayed the greatest reduction in self-reported disability (mean change = 12.8; SD = 5.0) compared to those patients whose pain continued (mean change = 6.24; SD = 5.4) at discharge. Acute patients displayed a slight increase at discharge from treatment compared to their already low BDI scores at admission (mean change = -.6; SD =6.2) compared to those individuals who later presented with chronic pain (mean change 1.5; SD = 4.5). Data presented in Table 8 serve to elaborate these differences.

Discussion

Taken together, these results suggest that following introduction of an acute health threat, self-reported disability levels, coping strategies, and emotional appraisal appear related to the development of chronic pain. Results were more clearly definable in terms of post-treatment functioning as predictive of continuing pain. These findings are in keeping with data reported recently by Hadjistavropoulos and Craig (1994), in their cross-sectional analysis of acute and incongruent chronic pain patients. Collapsing across all groups, there is a systematic improvement in functioning following treatment. However, when individuals whose pain resolves are compared to individuals who continue to have pain, a number of differences emerge. Results indicate that those individuals who go on to develop a chronic condition a) present initially with significantly more depressive symptoms and pain than those patients whose pain resolves, and b) patients who later develop a chronic condition present with fewer gains following acute care treatment compared to those whose pain eventually resolves. As noted by Melzack (1988), Sternbach (1984), and others (e.g., De Gagné, 1995; Romano and Turner, 1985), it is generally suggested that as pain persists, there is an increased likelihood of the development of depression and further functional impairment.

Interestingly, in the current study, physiotherapist ratings of the severity of the injury at admission were not significantly related to the development of chronic pain. This lack of association between physical examination and outcome has been previously reported (e.g., Gallagher et al., 1989; Lacroix, 1990; Murphy, Sperr, & Sperr, 1986). As pain persisted, it was shown that somatic focus increased alongside reports of pain and catastrophizing. Depressive symptoms were shown to be synchronous with the continued presentation of pain and somatic focus, as has been suggested by Romano and Turner (1985).

In this study, only individuals with a first injury were selected in order to control for previously learned symptom management strategies regarding the presentation of low back pain. It was expected that the changes in responses would allow for an examination of coping responses over time for individuals faced with a new health threat. Hence, the specific influence of previously learned responses to an acute low back injury would not play a role in adjustment. The data suggest that injured workers who eventually develop chronic pain do not perform as well during their acute stages of treatment compared to those individuals whose pain eventually resolves. Individuals whose pain does not resolve also do not demonstrate improvements in the areas of somatic appraisal, depressive symptoms, and coping strategies. These findings support and extend those of Roland and Morris (1983), Hadjistavropoulos and Craig (1994), Romano and Turner (1985), Philips and Grant (1991), and of cluster analytic studies (Klapow et al., 1993; Sanders and Brena, 1993), suggesting that temporal aspects of chronic pain development may be related to 1) a synchronicity between pain and depression, 2) increased somatic focus and maladaptive coping strategies, and 3) self-reported disability and depressive symptoms. These characteristics may be markers of future functioning during the acute presentation of a low back injury. Similarly, minimal changes in key variables following treatment also may be markers for appropriate psychological intervention. These variables include depressive affect, self-reported disability, somatic focus, reported pain, and coping strategies such as diverting attention and active cognitive coping.

Initially, it may appear circular to find that individuals whose pain does not resolve have greater somatic focus, self-reported disability, and depressive symptoms. However, there are important aspects of these data that indicate the unique contribution of these variables independent of group classification. To elucidate, this study was prospective and subjects who later develop chronic pain presented with higher levels of the aforementioned dimensions at the initial acute evaluation. Patient evaluations were made at that time, not after the patient had been defined as chronic or acute. Hence, these data extend beyond previous cross sectional studies that look at potential predictors of chronic pain after the fact, and out of synchrony with the development of the disorder. Furthermore, the patients who continued to present with pain at three-months reported higher levels of depressive symptoms during the initial acute phase of their injury compared to those whose pain resolved at three-months. This is important to denote because the levels of affect initially reported by these individuals are not traditionally regarded as clinically significant. However, these levels were predictive of chronic pain development. Hence, the opportunity to follow these individuals prospectively to a point, whereby, they were classified as experiencing chronic pain, revealed significant developmental concomitants of chronic pain that only have been alluded to in previous cross sectional studies (e.g., Hadjistavropoulos & Craig, 1994; Jensen et al., 1991; Romano & Turner, 1985).

These results also suggest that elevated levels of somatic focus and maladaptive cognitions occur along the temporal gradient from injury onset to six-months. Depressive symptoms remain relatively stable across the six-month gradient. These findings support the notion that the implicit coping demands of ongoing pain and lack of resolution are associated with psychological challenges. The length of time pain is experienced appears to be related to psychological coping variables, which are associated with illness impact and disability. This is indicated by the continuance of depressive symptoms and increases in reported pain and catastrophizing over time. Hence, the actions patients take in response to their illness presentation may be related to their appraisal of the threat; self-reported disability, pain levels, personal responsibility for wellness, depressive symptoms, and perceived self-efficacy over symptoms.

Implicit Illness Models

Contrary to the initial hypothesis, there was no clear delineation of the role of implicit models of illness as measured by either the SAI or IMIQ to predict the development of chronic pain. Despite the fact that the data were in the hypothesized directions, the effect was not significant. There are a number of reasons why this hypothesis may be not have been supported. It may be that the impact of these implicit indicators of coping may take longer to establish along the temporal gradient. For example, it may be that the continual re-appraisal of the threat and subsequent emotional coping may eventually lead to the expression of lack of control, lowered experience of personal responsibility and increased self-reported severity of condition that is often evidenced in rehabilitation programs for patients with longstanding chronic pain (e.g., Härkäpä, 1991; Hinkley, & Jaremko, 1994; Toomey et al., 1993). Hence, this effect may take longer to generate than could be measured in the current study. At this point, the length of time to develop these schemata, or implicit models is not clearly understood other than coping may be continually re-evaluated against an existing pain schema (cf., Edwards & Pearce, 1994), or continue to be evaluated over time as posited by Leventhal (1980). Furthermore, the effect size associated with the IMIQ predicting chronic pain status was extremely small. This may indicate that this measure may not posses properties to adequately predict adjustment early on in the disability process or that the small sample was inadequate to assess the small associated effect. Regarding the significance of small effects, this study proposed that larger effects would be more clinically meaningful. It follows that the IMIQ and the SAI were not sensitive enough to test the hypotheses proposed in the study.

Relatedly, differences between the samples recruited in other studies, compared to the current study, also may have affected the results. For example, Lacroix et al., (1990) recruited injured workers within three to six-months of their first back injury. In some cases, the patients assessed by Lacroix et al. (1990) presented two years following their injury. Compared to the acute and chronic patients observed in the current study, different patient samples may have contributed to the differences observed regarding the impact of implicit models of illness. It may be that the longer time period observed by Lacroix et al. (1990) enabled the authors to delineate the role of patients' understanding of their condition and related outcome, while the current study was unable to demonstrate this point.

Hinkley and Jaremko (1994) posited that longer pain duration was related to decreased coping and increased psychological disturbance. Their sample comprised 635 orthopedic patients, grouped into one of five different pain-duration categories. Patients with pain duration from 9 to 12-months exhibited the greatest degree of psychological disturbance. The absence of significant findings regarding personal responsibility and control dimensions in the current study may be because patients were not followed beyond six-months. In this regard, Cassileth et al., (1984) reported the existence of a three-month adjustment period following diagnosis, at which time they posited psychological adjustment and renegotiation of self-concept to begin taking place. In summary, it may be that a longer period of time may be necessary to examine the extent to which personal responsibility and control variables impact significantly on adjustment to injury.

Cognitive Strategies

The construct of catastrophizing has been well documented in the literature to date (e.g., Jensen & Karoly, 1991; Rosenstiel & Keefe, 1983, Sullivan and D'Eon, 1990). Interestingly, differences in catastrophizing between men and women also have been noted, primarily in relation to the recency of the threat (Unruh, 1996). The current data captured this gender difference, indicating that females catastrophize more at injury onset than males and also use more active coping strategies. However, due to sample size limitations, when the groups were divided by gender, a test for the statistical interaction of this difference was not possible. Gender differences were not present at the chronic stage, and in fact, it appeared that men catastrophized more than women. Future studies may want to examine gender differences in catastrophizing over time in this population. Such an approach may facilitate our understanding of clinical presentations across gender, and could affect the cognitive treatment focus with men and women. Finally, research on the gender invariance across the factor structure of the CSQ in pain populations would be informative. This may reveal that normative groups for men and women are needed. A confirmatory factor analysis of the CSQ testing for factorial invariance across gender in a pain population has yet to appear in the literature.

Rosenstiel and Keefe (1983) have reported that higher levels of active cognitive coping and diverting attention have been associated with impaired physical functioning. This hypothesis was tested in the current data and applied to treatment change scores to determine if minimal changes at discharge from treatment were related to further development of chronic pain status. The significant prediction of group membership based on cognitive coping and diverting attention change scores, suggests that a minimal decline in the use of coping self-statements and diverting attention at discharge may signal the potential for future dysfunction. This is noteworthy in light of admission coping self-statement reports that were equally balanced among acute and chronic pain patients. This finding is in agreement with reports by Rosenstiel and Keefe (1983) who noted that higher CSQ scores in areas such as cognitive coping and diverting attention, and praying and hoping, were correlated with higher levels of functional impairment and higher pain levels. As predicted, there was an overall reduction in catastrophizing cognitions when comparing admission with three-month scores. Interaction among groups or a main effect for self-efficacy failed to reach significance. These insignificant findings may be due to at least three factors. The sample size used for this analysis may have been too small to demonstrate an effect. As previously discussed regarding the characteristics of schema development, it also may be that the course of time examined was not long enough to determine changes. Alternatively, this variable may not in fact be important in the development of chronic pain.

While coping and adjustment have been studied in low back patients before, this investigation presented the first attempt to compare and contrast the development of acute and chronic pain, in a prospective manner, in a population that had not previously been injured. It was originally hypothesized that illness schemata would serve to elucidate differences in adjustment and coping, and be predictive of the development of chronic pain. While some measures failed to demonstrate significant differences in initial coping or beliefs about patients' condition, significant differences in terms of how the groups interpreted self-reported disability, somatic focus, depressive symptoms and reported pain levels did emerge. This is consistent with the hypothesis that as pain persists it may be more difficult for chronic pain patients to understand, conceptualize, and predict the course of their symptom presentation other than it appears to be getting worse and uncontrollable. It is possible that this is related to the interpretation of the illness process itself. Without a readily identifiable disease process, it may be that the chronic pain patient does not have easy access to a prototypic explanation, or illness schema for their ongoing presentation of symptoms. The notion of uncertainty and ambiguity, as noted by Markus and Wurf (1987), may be inherently stressful and exacerbate the interpretation of the hopelessness of their situation. This was evidenced in these data by increases in catastrophizing and pain levels at six-months. In contrast, acute pain patients present with decreases in these domains at discharge from treatment, which appear to be representative of a resolution of their symptoms at three-months.

In the absence of previous experience to interpret or understand their illness, (e.g., a previous back injury) chronic pain patients may find it more difficult to take personal responsibility for the management of their symptoms. Hence, less diverting attention, less active coping, greater self-reported disability and depressive symptoms may be manifest during the continuing experience of back pain. The predominant psychologic model of treating chronic pain leads health care professional into a course of action that often targets these domains, such as personal responsibility, active coping, reducing somatic focus, and in some cases, treatment for depressive symptoms (cf., Turk & Meichenbaum, 1988; Turk, Meichenbaum & Genest, 1983).

According to Leventhal's model of coping with a health threat, the re-appraisal process of the threat suggests that patients are actively coping with the demands of continued pain. As suggested by the current data, as the pain condition continues, there is a re-emergence of an acute state of appraisal when the problem becomes unremitting and longer term. Hence, a return to more acute appraisal of the threat is seen, which includes catastrophizing, somatic focus, and increased reports of pain levels. One interpretation may be that during the course of ongoing demands of work and pain, the pain may indeed be increased due to the ongoing repetitive strain of returning to pre-injury employment. According to gate control theory (Melzack & Wall, 1965), ongoing pain forces interpretation of the threat. In time, it may be that the individual becomes more frustrated thus, pain levels are increased, resulting in increased catastrophic thoughts that would include cognitions such as "it's terrible and I feel it is never going to get better" or " it is awful and I feel it overwhelms me," as measured by the CSQ (Rosenstiel & Keefe, 1983). Conversely, there may be an increased demand for the use of active coping or attention diversion strategies. This was evidenced in the data by the significant contribution of the coping and diverting attention factors to predict chronic pain.

Pain, Disability, And Affect

It appears that at injury onset, pain and depressive features may be predictive of future functioning, while other responses to the injury are relatively constant across groups. However, at discharge, differences in coping, affective distress, self-reported disability and pain are predictive of future functioning. Roland and Morris (1983) noted that self-reported disability four weeks after injury onset may be associated with poor outcome. In this regard, high disability scores reported by low back pain patients at presentation to primary care have been related poor outcome (Roland & Morris, 1983). In addition, the role of depressive affect also has been linked to chronic pain (De Gagné & Korol, 1996; Romano & Turner, 1985). These relationships also are present in the current data as predictive of continuing pain.

From a descriptive viewpoint, those individuals who continue to present with pain across the four time points, generally show a similar pattern as described by Philips and Grant's (1991a) motor vehicle accident patients. What is noteworthy is that somatic focus continues to rise across each time period, and pain intensity, and catastrophizing increase to above admission levels. This is interesting in light of the absence of further injury, that people perceive their pain as greater than when they were initially injured. This would suggest that ongoing pain is perceived as more severe compared to the initial injury time point. Examining correlations across time points, it appears that disability, depressive symptoms, and somatic focus become more highly correlated across each time period as pain levels persist. This would be in keeping with Romano and Turner's (1985) contention that self-reported disability or impairment is synchronous with the development of increases in depressive symptoms. Geisser et al. (1993) also reported that somatic focus was related to depressive affect. De Gagné (1995) specified a causal model of this relationship, whereby pain, increased functional interference, and social support were related to the expression of depressive features.

Adjustment To Injury

Jensen and Karoly (1992) posited that beliefs about personal competencies and capacities may be important components of adjustment to prolonged pain. Results of their study of 118 chronic pain patients revealed that after controlling for pain severity, those patients who regarded themselves as disabled demonstrated lower levels of psychological well-being as measured by the CES-D. The authors suggested that future research should determine whether beliefs assessed prior to treatment were related to subsequent functioning. The current study established this link in an acute low back pain population by assessing the extent to which psychological variables close to injury onset were related to adjustment.

In their review of the return to work literature, Fishbain et al. (1993) reported that the goals associated with the multidisciplinary treatment of chronic pain patients have included improvement in strength and functional status, to restore occupational functioning, and reduce psychological impairment (cf., Aronoff, Evans, & Enders, 1982; Chapman, Brena, & Bradford, 1981; Roy, 1984). Fishbain et al. (1993) also reported a number of psychosocial variables that were found to be predictive of return to work that included depressive symptoms, self-efficacy and ability to control pain, and perceived disability. These studies noted the importance of coping strategies as primary indicators of adjusting to injury. The relevance of these findings to the current sample of acute low back patients who were followed over time is clear as regards the impact of coping variables. In the current study, it appears that the impact or presence of these important predictor variables was substantively reduced upon completion of the treatment program. Thus, reductions in risk factors such as catastrophizing, ability to control pain, taking personal responsibility for wellness, self-reported pain and disability, may at this level serve as a buffer regarding the development of chronic pain or further adjustment difficulties that have previously been noted in the literature (e.g., Jensen & Karoly, 1992; Jensen et al., 1991; Klenerman et al., 1995; Rosenstiel & Keefe, 1983). Such a position may be tested using causal modeling to establish the impact of these variables on future functioning.

Taken together, the results of the current study suggest that adjustment may be related to a person's appraisal of a health threat and his or her perceived ability to overcome the threat (cf. Lau & Hartman, 1983; Leventhal et al, 1980, 1984; Turk, Rudy & Salovey, 1986). An emphasis on objective evaluation and emotional coping with the experience regarding the amelioration of symptoms is fundamental in this approach. A more passive stance would speak to externalization of the agent of change and possibly reinforce more passive means of coping such as praying and hoping, or diverting attention from the pain (Lawson et al., 1990). In this regard, reinforcing personal control and responsibility may speak to the efficacy of active approaches to symptom management which is in keeping with cognitive-behavioral approaches to pain management (cf., Turk, Meichenbaum, & Genest, 1983).

As noted by previous researchers (e.g., Jensen & Karoly, 1992; Phillips and Grant, 1991), one way of determining the impact, or severity of the pain report is to evaluate the effects of pain on various physical and psychological dimensions of the person's experience. In this regard, the current results indicate that present pain intensity levels and self-reported disability become increasingly correlated at discharge. This relationship also was reported by other researchers tracking the evolution of acute pain, whereby, patients' self-reported disability was associated with pain levels, and pain ratings were associated with psychological functioning and activity levels (e.g., Phillips & Grant, 1991). This suggests that as pain progresses, self-reported disability also increases, subsequently setting the stage for possible maladaptive functioning. Although this implies causality, such a relationship was not tested in the current study. Reification of a causal relationship with maladaptive functioning could be tested using longitudinal causal modeling.

The presence of emotions (mood) may activate thoughts based on an acute schema, in part, because chronicity implies lack of control over outcomes and the acceptance of permanent negative changes in the self (Leventhal et al., 1984). This may be a suitable explanation of the current data as evidenced by the BDI's significant contribution to the classification of patients with chronic pain. This is especially relevant in the context that most illnesses follow an acute pattern (Leventhal, et al., 1984). Although not all representations are expected to fall within the model of acute illness, this mold is powerful, as was demonstrated in the current data. Leventhal et al. (1984) posited that patients conceptualize their illness in acute terms with the expectation of symptom resolution after a finite period of time (i.e., they get better). Such an expectation is regarded as highly resistant to change. It follows, in the context of the current data, that in the absence of change, somatic focus is again turned inward. This focus serves to address the nature of the persistent pain, which in the acute stage is regarded as a warning signal, signifying tissue damage. Herein lies the utility and clinical application of gate control theory (Melzack & Wall, 1965), whereby, these signals are interpreted and related thoughts, emotions and behaviors are enacted. Unfortunately, in the chronic context, reactions such as catastrophizing, somatic focus, and loss of control, do not serve a protective function. Highly resistant schema, such as interpreting pain as a warning signal or tissue damage, is the premise upon which many cognitive-behavioral interventions for chronic pain reside (cf., Turk, Meichenbaum & Genest, 1983). Based on the current data, it appears that studying conditions that sustain one experience over another (cf., Leventhal et al., 1984), such as depressive symptoms, self-reported pain and disability, and cognitive coping, are appropriate goals of research and clinical intervention.

Taken together, results suggest that acute low back pain patients report decreased levels of feeling overwhelmed by their symptoms at discharge as measured by catastrophizing, increased levels of taking responsibility for their injury, and greater reports of self-efficacy. This suggests the appearance of an overall increase in the quality of life of these participants post-program, as well as demonstrates the existence of quantifiable increases in self-reported levels of improvement in disability ratings.

Clinical Application of Adjustment Variables

Behavioral based treatments for chronic pain generally reflect one of two theoretical approaches (Turner & Clancy, 1988). The operant-conditioning approach developed by Fordyce (1976) attempts to decrease pain behaviors and increase well behaviors. This is achieved by modifying concomitant social and environmental cues. As reported by Keefe and Gil, (1988) a number of behavioral observation techniques are available to record specific pain behavioral patterns such as; 1) motor pain behaviors (e.g., body posturing, facial expressions); 2) activity level; and 3) medication intake. These approaches have evolved beyond Fordyce's (1976) self-monitoring approach that employed a daily activity diary. In this regard, the cognitive-behavioral approach has been outlined by Turk, Meichenbaum and Genest (1983). This latter approach is designed to help patients learn to live more effectively in their environment despite the presence of varying levels of discomfort (Turk & Meichenbaum, 1988). This type of remediation emphasizes patient acquisition of cognitive and behavioral skills for coping with pain (Turner & Clancy, 1988).

Cognitive and behavioral interventions typically focus on cognitive, physical, and behavioral components of the pain experience to optimize client functioning. In relationship to the current findings, coping strategies, affect, self-reported disability, and pain levels were predictive of the further development of chronic pain. In keeping with current theoretical paradigms, application of cognitive and behavioral techniques to the current marker variables closer to injury onset may serve to enhance a return to previous levels of functioning.

Limitations

Despite significant results, meaningful effect sizes, and the apparent representativeness of the sample, one must be mindful of the limits pertaining to sample size. Hence, more extensive longitudinal replication, with a larger sample would serve to extend the findings of this research. Generalization of clinical research findings across settings and populations is problematic for at least two reasons: 1) the extent to which heterogeneous groups of patients are treated across settings makes comparisons across sites less than optimal, and 2) differing treatment philosophies may be represented across treating institutions. In the context of the present sample, results can only be compared to other centres that assess low back pain patients very close to acute onset.

Although significant demographic differences did not emerge in the data screening, this may have been due to limited power related to the sample size. In this regard, a visual inspection of means revealed that those individuals who later developed chronic pain generally reported higher income and education levels than acute pain patients. This may be an artifact of the sample obtained in Ottawa. However, given the literature, such a presentation would work against the proposed hypotheses, rather than in favor of finding differences regarding acute and chronic pain patients. In this regard, the literature has identified that lower educational status and lower income has typically been linked to poor outcome (e.g., Lacroix, 1990). If this were a significant covariate in the current sample, those individuals with higher education and income would be expected to perform better than those occupying a different social status. This was not the case, hence, may be an artifact in the current data set, and not a confound leading to the prediction of chronic pain.

Type of treatment programs being offered or geographical concomitants are relevant in the current study and should be taken into consideration when comparing these data to other institutional settings. Several contextual variables also are worthy of mention in the sample data reported. For example, there were no litigation variables involved in this study, hence, this variable was not relevant to the current results as has been noted in other studies (cf., Fishbain et al., 1993; Philips & Grant, 1991). Furthermore, the sample comprised individuals who were not receiving a WCB claim, and rival hypotheses regarding chronic pain development and compensation do not apply. Although the current sample is unique, from the point of view of tracking individuals very near injury onset, not all participants in the clinic chose to participate. Analyses of available data does not suggest the presence of implicit differences among these individuals regarding injury onset. Participants and non-participants may have differed in other ways that could not be determined because data were not available for individuals who declined to be approached for the study. Hence, this sample is considered limited as regards the external validity of the findings and the generalizability of the results should be mindfully interpreted.

The difficulty of obtaining a larger sample also presented as a limitation in the study. The presence of strong effect sizes, cross study comparisons, and normality of the distribution would seem to offer support for these data given this limitation. The fact that these results are consistent with previous prospective or cross-sectional studies is encouraging (e.g., Dozois et al., 1995; Hadjistravropoulos & Craig, 1994; Jensen & Karoly, 1992; Jensen et al. 1994; Phillips and Grant, 1991). Furthermore, a conservative analytic approach was taken despite the fact that assumptions of normality, linearity, and homogeneity of variance-covariance matrices held for the data. This speaks to the strength and representativeness of the data despite the small sample obtained.

Conclusion

In conclusion, these data suggest that in the context of a brief rehabilitation protocol, psychological variables were delineated that may be predictive of the development of chronic pain. Admission variables previously reported in the literature to be associated with chronic pain status were noted to be significantly reduced at discharge from the clinic (e.g., coping strategies, depressive symptoms, pain, self-reported disability status). This may speak to the importance of interventions that play a key role in mediating the ongoing presentation of self-reported pain and disability.

Most noteworthy was the strength of association among discharge variables measuring depressive symptoms, diverting attention, active coping, disability, and pain. The evolution or natural unfolding of pain in the current data set appears to resemble and extend the findings presented by Philips and Grant (1991). That is, participants with continuing pain demonstrated a decrease in cognitive coping alongside steady increases in somatic focus and self-reported pain levels. In keeping with Leventhal et al's (1984) model of coping with health threats, it may be that relying on an acute model of emotion appraisal at the six-month time point requires a re-evaluation of the initial event and current health status of the individual. This process may necessitate a renegotiation of the self, and perceived symptoms, to initiate more adaptive cognitions and adjustment to the limits imposed by the initial injury.

As noted by Rogers & Shoemaker (1971), communication and change are not synonymous, although communication is a necessary condition in order to invoke change. Change, then, occurs at the macro (societal) level as well as at the micro (individual) level. To date cognitive-behavioral efforts have focused on changing the perception of the source (the adaptation message) so that the receiver will perceive the message as credible. Communication, however, is only an intermediate process in a person's decision to change. In their review of over 1500 articles on social change, Rogers and Shoemaker (1971) noted that in order for diffusion of an innovation to occur, and invoke change, four elements are crucial to this process: 1) the innovation must be presented: 2) in turn it is communicated through multiple channels to the receiver of the innovation: 3) this process occurs over time, not over a single trial; and 4) members of the social system should influence the behavior of individual members of the social system. It is the concept of time which distinguishes the diffusion process from other communication research. The concepts of time and ongoing pain speak to adjustment issues in chronic pain in the current data, and notably in the data presented by Hinkly and Jaremko (1994) that speak to the necessity of early intervention to decrease the potential for the further development of chronic pain.

Future Research

There is a paucity of controlled longitudinal studies in the literature. Further controlled research with appropriate follow-up components will potentially serve to delineate good fits between the presenting problems and suitable cost-effective treatment alternatives. It follows that such interventions would impact on reducing health care expenditures, and concomitant physical, emotional, and financial burdens of workers and society at large.

From a social learning perspective, clinical interventions generally provide clients with opportunities to assess their own risk of acquiring a disease, or injury (Kelly & St. Lawrence, 1990). Further, interventions provide persons with; 1) the opportunity to question and acquire knowledge; 2) to practice self-regulatory skills; 3) receive feedback about their performance (reinforcement); 4) engage in problem solving (e.g., learn assertiveness); and 5) to generate a more accurate perception of peer norms. Utilizing these aspects of intervention embedded in a diffusionary theme, one possible clinical strategy could be designed within a prospective cohort evaluation study, with interventions aimed at early injury onset.

Another possible future direction would be to implement a longitudinal causal model based on Leventhal et al's (1984) work and the constructs presented herein to determine, longitudinally, causal features of future function such as depressive affect, somatic focus, personal responsibility, and catastrophizing. In the current data set, the IMIQ and SAI were not useful to delineate group membership. It is recommended that further use of the IMIQ be entertained with these limitations in mind. Such a model may include the variables reported in this study, ultimately predicting depression status or level of functional impairment and employment status.

Future research also may speak to the further delineation and impact of these variables through early intervention at the time of injury onset and appropriate follow-up procedures. For example, reinforcement of active repertoires of symptom management strategies closer to injury onset may enhance adjustment to injury and discourage the further development of maladaptive coping. Emphasis on gate control theory is clearly implied. As applied to the current study, a component of this design could include cognitive behavioral interventions to one of two groups. One group would receive usual care as described in this study. Subsequent to appropriate matching procedures, another group of recently injured low back pain patients would receive immediate cognitive behavioral interventions based on their scores on questionnaires designed to measure coping strategies, understanding of their illness process, depressive symptoms, etc. Hence, the intervention strategy would focus on marker variables presented in this study to enhance adjustment to the injury process. Optimal comparison of groups would take place over a one year period to examine patients' adjustment to the injury process and outcome utilization of active self-management strategies reinforced during treatment.

Summary

These results, taken together, suggest that the expectation of future functioning cannot be delineated by the objective severity of the presenting injury. The mediating role of affect, perceived disability, self-reported pain intensity, and coping strategies should continue to be considered in the treatment and management of acute low back injury. This study was unique regarding its attempt to track the evolution of chronic pain status in the absence of previous injuries. While there were some suggestive trends, acute and chronic pain patients did not significantly differ on many admission variables. Results were more clearly definable in terms of post-treatment functioning as predictive of chronicity. This would suggest, as Roland and Morris (1983) have posited, that four-weeks following injury, subsequent to treatment, group differences and identifiable markers of maladaptive future functioning will be more apparent. Cassileth et al's (1984) three-month criteria of adjustment to a health threat also may be relevant in the evolution of chronic pain status. This suggests that at onset, these variables may be marked for early intervention to ensure more productive functioning following treatment and potentiate greater adaptation to the initial injury for those at risk of developing chronic conditions.

It is apparent at the six-month juncture, that chronic pain patients begin to exhibit a decline in significant areas of interpreting their experience (e.g., catastrophizing, somatic focus, pain complaints). In fact, this may lead to reports of learned helplessness in this population. Given the continued presentation of their pain over time, these patients are repeatedly exposed to unpleasant aversive symptoms outside of their control. Consequently, these patients learn to expect aversive consequences, (e.g., catastrophize), increase their symptom focus, increase their reports of pain, and perceive a general lack of control over their symptoms and report greater disability (helplessness). Over time, such appraisals may become conditioned cognitive responses to aversive stimuli. With repeated exposure, the development of maladaptive cognitions may result in the development of cognitive schema to process physical stimuli. In this regard, Edwards and Pearce (1994) noted that persons with pain will endorse more pain related words in a word identification task. In the context of the current data, it appears that ongoing pain sets the stage for reification of an acute pain schema and related coping. Based on the observed data, prospective research that focuses on the role of depressive affect, coping strategies, and self-reported pain levels and disability may serve to provide more important information about the developmental characteristics of chronic pain.

References

Anastasi, A. (1982). Psychological testing(5th ed.). N.Y.: Macmillan Publishing.

Aronoff, G. M. (1982). A review of follow-up studies of multidisciplinary pain units. Pain, 16, 1-11.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. Psychology Review, 84, 191-215.

Barber, T. (1959). Towards a theory of pain. Psychological Bulletin, 56, 430-455.

Bergner, M., Bobbitt, R.A., Carter, W.B., & Gilson, B.S. (1981). The Sickness Impact Profile: Development and final revision of a health status measure. Medical Care, 19, 787-805.

Block, A.R., Kremer, E.F., & Gaylor, M. (1980). Behavioural treatment of chronic pain: variables effecting treatment efficacy. Pain, 8, 367-375.

Blumer, D., & Heilbronn, N. (1982). Chronic pain as a variant of depressive disease: the pain-prone disorder. Journal of Nervous and Mental Disease, 170, 381-406.

Bonica, J.J. (1990a). Definitions and taxonomy of pain. In J. J. Bonica (Ed). The Management of Pain (2nd ed., pp. 18-27). U.S.: Lea & Febiger.

Bonica, J.J. (1990b). General considerations of chronic pain. In J. J. Bonica (Ed). The Management of Pain (2nd ed., pp. 180-196). U.S.: Lea & Febiger.

Boyd, J.H. & Weissman, M.M. (1982). Epidemiology. In E.S. Paykel (Ed.), Handbook of affective disorders (pp. 109-125). New York: Guilford Press.

Brown, G.K. (1990). A causal analysis of chronic pain and depression. Journal of Abnormal Psychology, 99, 127-137.

Cassileth, B.R., Lusk, E. J., Strouse, T.B., Miller, D.S., Brown, L.L., Cross, P.A., Tenaglia, A.N. (1984). Psychosocial status in chronic illness: A comparative analysis of six diagnostic groups. New England Journal of Medicine, 311, 506-511.

Chapman, C. R. (1978). Pain: The perception of noxious events. In R. A. Sternbach (Ed.)., The Psychology of Pain. N.Y.: Pergamon Press. pp. 169-202.

Chapman, S.L., Evans, W.O., Enders, P.L. (1981). Treatment outcome in a chronic pain rehabilitation program. Pain, 11, 255-268.

Cohen, J. (1992). A power primer. Psychological Bulletin, 112, 155-159.

De Gagné, T. (1995). Depressive Affect: Testing for Causal Structure in Chronic Pain Patients. Presented at the Canadian Pain Society, Annual Meeting. Ottawa, Ontario, May, 1995.

De Gagné, T., & Korol, C. (1996). Factorial Validity and Invariance of the Beck Depression Inventory Across Gender: A Second Order CFA model. Presented at the 8th World Congress on Pain. Vancouver, Canada, August, 1996.

De Gagné, T., Mikail, S., D'Eon, J. (1995). Confirmatory factor analysis of a 4-factor model of chronic pain evaluation. Pain, 60, 195-202.

Deutsch, F., Kroll, J., Weible, A., Letourneau, L., & Goss, R. (1988). Spontaneous trait generation: A new method for identifying self-schemas. Journal of Personality, 56, 327-354.

Doxey, N.C.S., Dzioba, R.B., Mitson, G.L., Lacroix, M.J. (1988). Predictors of outcome in back surgery candidates. Journal of clinical Psychology, 44, 611-622.

Dozois, D.J., Dobson, K.S., Wong, M., Hughes, D., Long, A. (1995). Factors associated with rehabilitation outcome in patients with low back pain (LBP): Prediction of employment outcome at 9-month follow-up. Rehabilitation Psychology, 40, 243-259.

Dworkin, R., & Gitlin, M. (1991). Clinical aspects of depression in chronic pain patients. Clinical Journal of Pain, 7, 79-94.

Edwards, L.C., & Pearce, S.A. (1994). Word completion in chronic pain: Evidence for schematic representation of pain? Journal of Abnormal Psychology, 103, 379-382.

Eimer, Bruce, (1989). Psychotherapy for chronic pain. A cognitive approach. Arthur Freeman, Karen Simon, Larry Beutler, & Hal Arkowitz (Eds)., Comprehensive handbook of cognitive therapy. N.Y. : Plenum Press. (pp. 449-465).

Ellis. A. (1962). Reason and emotion in psychotherapy. Lyle Stuart, N.Y.

Fishbain, D.A., Rosomoff, H.L., Goldberg, M., Cutler, R., Abdel-Moty, E., Khalil, R.M., Rosomoff, R.S. (1993). The prediction of return to the workplace after multidisciplinary pain center treatment. The Clinical Journal of Pain, 9, 3-15.

Fiske, S., & Linville, P. (1980). What does the schema concept buy us? Personality and Social Psychology Bulletin, 6, 543-557.

Fordyce, W.E.(1976). Behavioural methods for chronic pain and illness. U.S.: Mosby.

Frymoyer, J.W., & Cats-Baril, W. (1987). Predictors of low back pain disability. Clinical Orthopaedics and Related Research,221, 89-98.

Gallagher, R.M., Raugh, V., Haugh, L.D., Milhous, R., Callas, P.W., Langelier, R., et al. (1989). Determinants of return-to-work among low back pain patients. Pain, 39, 55-67.

Geisser, M.E., Gaskin, J.E., Robinson, M.E., & Greene, A.F. (1993). The relationship of depression and somatic focus to experimental and clinical pain in chronic pain patients. Psychology and Health, 8, 405-415.

Gorsuch, R. (1983). Factor analysis. Hillsdale, N.J.: Erlbaum.

Gorsuch, R. (1990). Common factor analysis versus component analysis: Some well and little known facts. Multivariate Behavioral Research,25, 33-39.

Hadjistravropoulos, H., & Craig, K.D. (1994). Acute and chronic low back pain: Cognitive, affective and behavioural dimensions. Journal of Consulting and Clinical Psychology, 62, 341-349.

Hanson, R.W., & Gerber, K.E. (1990). Coping with chronic pain. N.Y.: Guilford Press.

Harris, R.J. (1985). A primer of multivariate statistics(2nd ed.). U.S.: Academic Press.

Hendler, N. (1984). Depression caused by chronic pain. Journal of Clinical Psychiatry, 45, 30-38.

Hinkley, B., & Jaremko, M. (1994). Effects of pain duration on psychosocial adjustment in orthopedic patients: The importance of early diagnosis and treatment of pain. Journal of Pain and Symptom Management, 9, 175-185.

Huck, S. W., & McLean, R.A. (1975). Using a repeated measures ANOVA to analyze the data from a pretest-posttest design: A potentially confusing task. Psychological Bulletin, 82, 511-518.

Iggo, A. (1987). Physiology of pain. In Graham D. Burrows, Diana Elton, & Gordon V. Stanley (Eds)., Handbook of chronic pain management. N.Y. : Elsevier. (pp. 7-18).

International Association for the Study of Pain. (1986). Classification of chronic pain. Pain, 3, (suppl.), S1-S225.

Jamieson, J. (1994). Correlates of reactivity: Problems with regression based methods. International Journal of Psychophysiology, 17, 73-78

Jensen, M, Turner, J., Romano, J., and Karoly, P. (1991). Coping with chronic pain: A critical review of the literature. Pain, 4, 249-283.

Jensen, M, Turner, J., Romano, J., and Lawler, K. (1994). Relationship of pain-specific beliefs to chronic pain adjustment. Pain, 57, 301-309.

Jensen, M., & Karoly, P. (1992). Comparative self-evaluation and depressive affect among chronic pain patients: an examination of selective evaluation theory. Cognitive Therapy and Research, 16, 297-308.

Jensen, M., & Karoly, P. (1991). Control beliefs, coping efforts, and adjustment to chronic pain. Journal of Consulting and Clinical Psychology, 59, 431-438.

Jensen, M., Nygren, A., Gamberale, F., Goldie, I, & Westerholm, P. (1994). Coping with long-term musculoskeletal pain and its consequences: Is gender a factor? Pain, 57, 167-172.

Jensen, M., Turner, & J., Romano, J. (1991). Self-efficacy and outcome expectancies: Relationship to chronic pain. Pain, 44, 263-269.

Jensen, M., Turner, & J., Romano, J. (1992). Chronic pain coping measures: individual vs. composite scores. Pain, 51, 273-280.

Jensen, M.P., Strom, S.E., Turner, J.A., Romano, J.M. (1992). Validity of the Sickness Impact Profile Roland scale as a measure of dysfunction in chronic pain patients. Pain, 50, 157-162.

Keefe, F. (1982). Behavioral assessment and treatment of chronic pain: Current status and future directions. Journal of clinical and consulting psychology, 50, 896-911.

Keefe, F.J., Dunsmore, J., & Burnett, R. (1992). Behavioural and cognitive-behavioral approaches to chronic pain: Recent advances and future directions. Journal of Consulting and Clinical Psychology, 60, 528-536.

Kelly, J., & St. Lawrence, J. (1990). The impact of community-based groups to help persons reduce HIV infection risk behavior. AIDS Care, 2, 25-36.

Kerns, R.D., & Jacob, M.C. (1992). Assessment of the psychosocial context in the experience of pain. In D.C. Turk and R. Melzack (Eds.), Handbook of pain assessment (pp. 235-256). N.Y.: Guilford Press.

Kidder, J.H., & Judd, C.M. (1986). Research methods in social relations(5th ed.). N.Y.: Holt, Rinehart and Winston.

Klapow, J., Slater, M., Patterson, T., Doctor, J., Atkinson, J., & Garfin, S. (1993). An empirical evaluation of multidimensional clinical outcome in chronic low back pain patients. Pain, 55, 107-118.

Kleneman, L., Slade, P., Stanley, I., Pennie, B., Reilly, J., et al. (1995). The prediction of chronicity in acute attack of low back pain in a general practice setting. Spine, 20, 478-484.

Lacroix, J.M. (1991). Assessing illness schemata in patient populations. In J.A. Skelton and R.J. Croyle (Eds.), The mental representation of health and illness: Models and applications (pp. 103-219). N.Y.: Springer-Verlag.

Lacroix, J.M., Clarke, M.A., Bock, J.C., & Doxey, N.C.S. (1986). Physiological changes after biofeedback and relaxation training for multiple-pain tension-headache patients. Perceptual and Motor Skills, 63, 139-153.

Lacroix, J.M., Martin, B., Avendano, M., & Goldstein, R. (1991). Symptom schemata in chronic respiratory patients. Health Psychology, 10, 268-273.

Lacroix, J.M., Powell, J., Lloyd, G.J., Doxey, N., Mitson, B.L., & Aldam, C.F. (1990). Low-Back pain: Factors of value in predicting outcome. Spine, 15, 495-499.

Lau, R.R., & Hartman, K.A. (1983). Common sense representations of common illnesses. Health Psychology, 2, 167-185.

Lau, R.R., Bernard, T.M. & Hartman, K.A. (1989). Further explorations of common-sense representations of common illnesses. Health Psychology, 8, 195-219.

Lawson, K., Reesor, K., Keefe, F., & Turner, J. (1990). Dimensions of pain related cognitive coping: Cross validation of the factor structure of the Coping Strategy Questionnaire. Pain, 43, 195-204.

Lazarus, R., & Folkman, S. (1984). Stress, appraisal, and coping. Springer, N.Y.

Leventhal, H. & Nerenz, D.R. (1983). A model for stress research with some implications for the control of stress disorders. In D. Meichenbaum and M. Jarenko (Eds.), Stress reduction and prevention (pp. 5-38). N.Y.: Plenum Press.

Leventhal, H., Meyer, D., & Nerenz, D. (1980). The common sense representation of illness danger. In S. Rachman (Ed.), Contributions to medical psychology (Vol. 2, pp. 7-30). N.Y.: Pergamon Press.

Leventhal, H., Zimmerman, R., & Gutmann, M. (1984). Compliance: A self-regulation perspective. In W.D. Gentry (Ed.), Handbook of behavioral medicine (pp. 369-436). N.Y.: Guilford Press.

Lonetto, L., Kumuchy, G., & Robinson, J. (1986). Back to normal. Doubleday Canada Ltd., Toronto, Canada.

Lowe, N.K., Walker, S. N., & MacCallum, R.C. (1991). Confirming the theoretical structure of the McGill Pain Questionnaire in acute clinical pain. Pain, 46, (1991) 53-60.

Markus, H. (1977). Self-schemata and processing information about the self. Journal of Personality and Social Psychology, 35, 63-79.

Markus, H. (1990). Unresolved issues of self-representation. Cognitive Therapy and Research, 14, 241-253.

Markus, H., & Wurf, (1987). The dynamic self-concept: A social psychological perspective. Annual Review of Psychology, 38, 299-337.

Mechanic, D. & Volkar, E.H. (1961). Stress, illness behavior, and the sick role. American Sociological Review, 26, 51-58.

Mechanic, D. (1962). The concept of illness behavior. Journal of Chronic Diseases, 15, 189-194.

Melzack, R. & Wall, P.D. (1965). Pain mechanisms: A new theory. Science, 50, 971-979.

Melzack, R. & Wall, P.D. (1982). The challenge of pain. Great Britain: Penguin Books.

Melzack, R. (1975). The McGill Pain Questionnaire: Major properties and scoring methods. Pain, 1, 277-299.

Mikail, S., D'Eon, J., De Gagné, T. (1996). Validation of the pain beliefs and perceptions inventory, Pain Research and Management, 1, 31-38.

Murphy, J.K., Sperr, E.V., & Sperr, S.J. (1986). Chronic pain: An investigation of assessment instruments. Journal of Psychosomatic Research, 30, 289-296.

Norusis, Marija. (1990). SPSS advanced statistics user's guide. Chicago: SPSS Inc.

Pennebaker, J. (1984). Accuracy of symptom perception. In A. Baum, S. Taylor, & J. Singer (Eds.), Handbook of psychology and health: Vol IV. Social psychological aspects of health (pp. 189-217). Hillsdale, H.J: Lawrence Erlbaum Associates.

Pennebaker, J.W. (1982). The psychology of physical symptoms. N.Y.: Springer-Verlag.

Philips, H.C. (1989). Thoughts provoked by pain. Behavior Research Therapy, 27, 469-473.

Philips, H.C., & Grant, L. (1991). The evolution of chronic back pain problems: A longitudinal study. Behavior Research Therapy, 29, 435-441.

Philips, H.C., Grant, L., & Berkowitz, J. (1991). The prevention of chronic pain and disability: A preliminary investigation. Behavior Research Therapy, 29, 443-450.

Reesor, K.A., Craig, K.D. (1988). Medically incongruent chronic back pain: physical limitations, suffering, and ineffective coping. Pain, 32, 35-45.

Rogers, E., & Shoemaker, F. (1971). Communication of innovations (2nd ed.). N.Y.: Free Press.

Roland M., & Morris, R. (1983). A study of the natural history of low-back pain. Part I. Development of a reliable and sensitive measure of disability in low-back pain. Spine, 8, 141-144.

Romano, J., & Turner, J. (1985). Chronic pain and depression: Does the evidence support a relationship? Psychological Bulletin, 97, 18-34.

Rosenstiel, A., & Keefe, F. (1983). The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and adjustment. Pain, 17, 33-43.

Roy, R. (1984). Pain clinics: Reassessment of objectives and outcomes. Archives of Physical and Medical Rehabilitation, 65, 448-451.

Rudy, T., Kerns, R., & Turk, D. (1988). Chronic pain and depression: Toward a cognitive mediation model. Pain, 35, 129-140.

Rybstein-Blinchik, E. (1979). Effects of different cognitive strategies on the chronic pain experience. Journal of Behavioural Medicine, 2, 93-102.

Safran, J.D., Segal, Z.V., Hill, C., Whiffen, V. (1990). Refining strategies for research on self-representations in emotional disorders. Cognitive Therapy and Research, 14, 143-160.

Sanders, S., and Brena S. (1993). Empirically derived chronic pain patient subgroups: the utility of multidimensional clustering to identify differential treatment effects. Pain, 54, 51-56.

Schober, R. & Lacroix, J.M. (1991). Lay illness models in the enlightenment and the 20th century: Some historical lessons. In J.A. Skelton and R.J. Croyle (Eds.), The mental representation of health and illness: Models and applications (pp. 10-31). N.Y.: Springer-Verlag.

Schrout, P.E., & Fleiss, J.L. (1979). Intraclass correlations: Uses in assessing rater reliability. Psychological Bulletin, 86, 420-428.

Spitzer, W.O. (1987). Scientific approach to the assessment and management of activity-related disorders: A monograph for clinicians. Report of the Quebec task force on spinal disorders. Spine, 12, (suppl.), S1-S53.

Sternbach, R.A., (1986). Survey of pain in the United States: The Nuprin report. The clinical Journal of Pain, 2, 49-53.

Sternbach, R.A., Wolf, S.R., Murphy, R.W., & Akeson, W.H. (1973). Aspects of chronic low back pain. Psychosomatics, 14, 226-229.

Strong, J., Ashton, R., & Stewart, A. (1994). Chronic low back pain: Towards an integrated psychosocial assessment model. Journal of Consulting and Clinical Psychology, 69, 1058-1063

Strong, J., Ashton, R., Cramond, T., & Chant, D. (1990). Pain intensity, attitude and function in back pain patients. Australian Occupational Therapy Journal 37, 179-183.

Sullivan, M., & D'Eon. (1990). Relation between catastrophizing and depression in chronic pain patients. Journal of Abnormal Psychology, 99, 260-263.

Sullivan, M., Reesor, K., Mikail, S., & Fisher, R. (1992). The treatment of depression in chronic low back pain: Review and recommendations. Pain, 50, 5-13.

Tabachnik, B.G., & Fidell, L.S. (1989). Using multivariate statistics(2nd ed.). N.Y.: Harper & Row.

Tabachnik, B.G., & Fidell, L.S. (1996). Using multivariate statistics(3rd ed.). N.Y.: Harper & Row.

Toomey, T., Mann, J., Abashian, S., & Thompson-Pope, S. (1991). Relationship between perceived self-control of pain, pain description and functioning. Pain, 45, 129-133.

Toomey, T., Mann, J., Abashian, S., Carnrike, C., Hernandez, J. (1993). Pain locus of control scores in chronic pain patients and medical clinic patients with and without pain. The Clinical Journal of Pain, 9, 242-247.

Turk, D., & Meichenbaum, D. (1988). A cognitive-behavioral approach to pain management. In P. Wall & D. Melzack (Eds.), Textbook of pain (2nd ed., pp. 1001-1009). New York: Churchill Livingston.

Turk, D.C., & Melzack, R. (1992). The measurement of pain and the assessment of people experiencing pain. In D.C. Turk and R. Melzack (Eds.), Handbook of pain assessment (pp. 3-12). N.Y: Guilford Press.

Turk, D.C., Meichenbaum, D., & Genest, M. (1983). Pain and behavioral medicine. A cognitive-behavioral perspective.N.Y: Guilford Press.

Turk, D.C., Rudy, T.E., & Salovey, P. (1986). Implicit models of illness. Journal of Behavioural Medicine,9, 453-473.

Turner, J., & Romano, J. (1984). Self-report screening measures for depression in chronic pain patients. Journal of Clinical Psychology, 40, 18-34.

Unruh, A. (1996). Gender variations in clinical pain experience. Pain, 65, 123-167.

Von Korff, M. (1992). Epidemiological and survey methods: Chronic pain assessment. In D.C. Turk and R. Melzack (Eds.), Handbook of pain assessment (pp. 391-408). N.Y.: Guilford Press.

Von Korff, M., Le Resche, L., & Dworkin, S. (1993). First onset of common pain symptoms: A prospective study of depression as a risk factor. Pain, 55, 251-258.

Waddel, G. (1982). An approach to backache. British Journal of Hospital Medicine, 22, 187-219.

Wade, J.B., Dougherty, L., Hart, R.P., & Cook, D.B. (1992). Patterns of normal personality structure among chronic pain patients. Pain, 48, 37-43

Wall, P. (1989). The dorsal horn. In P. Wall & D. Melzack (Eds.), Textbook of pain (2nd ed., pp. 102-111). New York: Churchill Livingston.

Ward, N. (1990). Pain and depression. In J.J. Bonica (Ed.), The management of pain (2nd ed., pp. 310-319). U.S.: Lea & Febiger.

Ward, N.G., Bloom, V.L., Dworkin, S., Fawcett, J., Narasimhachari, N., & Friedel, R.O. (1982). Psychological markers in coexisting pain and depression: Towards a unified theory. Journal of Clinical Psychiatry, 43, 32-39.

Weisenberg, M. (1987). Cognition and pain. In Graham D. Burrows, Diana Elton, & Gordon V. Stanley (Eds)., Handbook of chronic pain management. N.Y. : Elsevier. (pp. 77-84).

Weiss, D. J., Dawis, R.V., England, G.W., & Lofquist, L.H. (1967). Manual for the Minnesota Satisfaction Questionnaire St. Paul, MN.: Work Adjustment Project, Industrial Relations Centre, University of Minnesota.

Williams, D.A. , & Thorn, B.E. (1989). An empirical assessment of pain beliefs. Pain, 36, 351-358.

World Health Organization. (1980). International classification of impairments, disabilities, and handicaps: A manual of classification relating to the consequences of disease. USA: Author.

Appendix A

Demographic Information Form / Consent Form

INJURED WORKERS' REHABILITATION CLINIC

INFORMED CONSENT FORM

Measuring Adjustment to Pain

We are currently evaluating how people interpret their reactions to injury and pain. The goals of the evaluation are: 1) to examine the possible factors involved in the adjustment to pain; 2) to track your successes from your initial entry at the Injured Workers' Rehabilitation Clinic to when you are discharged; and 3) to monitor your progress for a time after you leave the Injured Workers' Rehabilitation Clinic.

In order to answer these questions, we are asking each person who receives physiotherapy for their back pain at the Injured Workers' Rehabilitation Clinic to complete some questionnaires related to their pain experience at four different points in time: 1) before starting physiotherapy sessions; 2) after you are discharged; 3) about two months after you are discharged; 4) and finally, about three months after that. At each time point you will be asked to fill out some questionnaires that are related to your pain experience.

You may take the questionnaires home to fill out. It will take you about 45 minutes to complete these questionnaires. The questionnaires ask about a number of topics, such as functional abilities, activity levels, the strategies you use to cope with pain, and how you are doing at work. Your responses will help us find out how people adjust to their pain and identify areas that we need to improve. Your participation in this project is entirely voluntary and you can withdraw from the study at any time you choose.

If you have any questions about your participation in this project, you may contact Dr. Joyce D'Eon, Co-investigator (737-7350, ext. 564) or Dr. Gaétan Tardif, Chair, Research Review Committee (737-7350. ext. 306).

If you decide to take part, please read and sign the declaration below.

I agree to participate in this study with the understanding that information will be collected and used for research purposes only and will be treated as confidential. I have been informed about the purpose of this study and realize that I am under no obligation to participate and may withdraw at any time. Refusal to participate or withdraw from the study will in no way affect my present and/or future treatment at the Injured Workers' Rehabilitation Clinic or The Rehabilitation Centre.

Name: ____________________ ______________________
Date: ____________________ Signature

Witness: ____________________ ______________________
Date: ____________________ Signature

A clinic of The Rehabilitation Centre
1800 BANK • SUITE 204 • OTTAWA • ONTARIO • K1V 0W3 • (613) 731-9822

PERSONAL INFORMATION FORM

S#_______

1. Name__________________________ Male_____ Female_______
2. Date of Birth_____________________
   Year Month Day
3. Today's Date_____________________
4. Marital Status (circle one)
   1. single
   2. married or common-law
   3. widowed
   4. divorced (number of previous marriages___)
5. Highest level education obtained
   1. less than 9 years
   2. some high school
   3. completed high school
   4. some college or university
   5. completed an undergraduate diploma
   6. graduate work or professional school
6. <u>Before</u> your injury, what was your employment status?

   (circle one)

   1. Working full-time
   2. Working part-time
   3. Modified Work (due to injury)
   4. Unemployed and looking for work
   5. Full-time student
   6. Part-time student
   7. On temporary disability leave
   8. On permanent disability
   9. Working in the home (homemaker/parenting)

7. Which of the following categories best describes your current employment status?

   (circle one)

   1. Working full-time
   2. Working part-time
   3. Modified Work (due to injury)
   4. Unemployed and looking for work
   5. Full-time student
   6. Part-time student
   7. On temporary disability leave
   8. On permanent disability
   9. Working in the home (homemaker/parenting)

8. In your most recent job, what is/was your title?

   ____

9. Are you currently receiving (expecting) Workers' Compensation benefits?
   1. = Yes
   2. = No
10. Are you receiving compensation, pension, or insurance benefits from any other source?
    1. = Yes
    2. = No

PLEASE TURN OVER > > >

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11. Are you involved in any legal action around your medical problems or pension status?
1. = Yes
2. = No

If yes, please describe. ______________________________

____

12. Within the following broad categories, what is your total family income for a year? **(circle one)**

1 = under $10,000
2 = $10,000 - $20,000
3 = $21,000 - $30,000
4 = $31,000 - $40,000
5 = $41,000 - $50,000
6 = over $50,000

13. What medications are you currently taking? Please list all the medications that you have taken **over the past two weeks**, along with the average daily does of each. Include non-prescription medication, such as Tylenol or Aspirin.

TYPE OF MEDICATION	DOSE	NUMBER OF PILLS A DAY

14. What is your native language? **(circle one)**
1 = English
2 = French
3 = Other (please specify) ______________

15. In what country were you born?______________________

16. To which ethnic or cultural group do you feel you belong?
(please specify)______________

THE FOLLOWING QUESTIONS ASK ABOUT YOUR PAIN.

17. When did your pain first start? Month_____ Year_____

18. How did your pain begin?
1 = Motor vehicle accident
2 = Accident at work
3 = Other injury
4 = Pain just began
5 = Other (please specify)

19. Have you ever had surgery for your current pain?
1 = Yes
2 = No
If yes, please specify dates.

Thank you for taking the time to fill this out.
Please proceed to the next section.

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Appendix B

Coping Strategies Questionnaire

Date: ______________________ S#:____________

COPING STRATEGY QUESTIONNAIRE

Individuals who experience pain have developed a number of ways to cope or deal with their pain. These include saying things to themselves when they experience pain, or engaging in different activities. Below are a list of things that patients have reported doing when they feel pain. For each activity, please indicate, using the scale below, how much you engage in that activity when you feel pain, where 0 indicates you never do that when you are experiencing pain, a 3 indicates that you sometimes do that when you experience pain, and a 6 indicates you always do it when you experience pain. Remember you can use any point along the scale.

0	1	2	3	4	5	6
Never do			Sometimes do that			Always do that

WHEN I FEEL PAIN...

_____1. I try to feel distant from the pain almost as if the pain was in somebody else's body.
_____2. I leave the house and do something, such as going to the movies or shopping.
_____3. I try to think of something pleasant.
_____4. I don't think of it as pain but rather as a dull or warm feeling.
_____5. It is terrible and I feel it is never going to get better.
_____6. I tell myself to be brave and carry on despite the pain.
_____7. I read.
_____8. I tell myself that I can overcome the pain.
_____9. I count numbers in my head or run a song through my mind.
_____10. I just think of it as some other sensation, such as numbness.
_____11. It is awful and I feel it overwhelms me.
_____12. I play mental games with myself to keep my mind of the pain.
_____13. I feel my life isn't worth living.
_____14. I know someday someone will be here to help me and it will go away for awhile.
_____15. I pray to God it won't last long.
_____16. I try not to think of it as my body, but rather as something separate from me
_____17. I don't think about the pain.
_____18. I try to think years ahead-what everything will be like after I've gotten rid of the pain.
_____19. I tell myself it doesn't hurt.
_____20. I tell myself I can't let the pain stand in the way of what I have to do.
_____21. I don't pay any attention to it
_____22. I have faith in doctors that someday there will be a cure for my pain.
_____23. No matter how bad it gets, I know I can handle it.
_____24. I pretend it is not there.
_____25. I worry all the time about whether it will end.
_____26. I replay in my mind pleasant experiences in the past.

[please turn page over]

WHEN I FEEL PAIN...

_____27. I think of people I enjoy doing things with.
_____28. I pray for the pain to stop.
_____29. I imagine that the pain is outside of my body.
_____30. I just go on as if nothing happened.
_____31. I see it as a challenge and don't let it bother me.
_____32. Although it hurts, I just keep going.
_____33. I feel I can't stand it any more.
_____34. I try to be around other people.
_____35. I ignore it.
_____36. I rely on my faith in God.
_____37. I feel like I can't go on.
_____38. I think of things I enjoy doing.
_____39. I do anything to get my mind off the pain.
_____40. I do something I enjoy, such as watching TV or listening to music.
_____41. I pretend it is not a part of me.
_____42. I do something active, like household chores or projects.

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. Remember, you can circle any number along the scale.

0	1	2	3	4	5	6
No control			Some control			Complete control

Based on all the things you do to cope or deal with pain, on an average day, how much are you able to decrease it? Please circle the appropriate number. Remember, you can circle any number along the scale.

0	1	2	3	4	5	6
Can't decrease it at all			Can decrease it somewhat			Can decrease it completely

Appendix C

Schema Assessment Inventory

SYMPTOM ASSESSMENT INVENTORY

Name:____________________
Date:__________

For us to understand better the nature of your pain experience, and the factors that add to your experience, please fill out this questionnaire as carefully as possible. When you finish, we will have a chance to go over your answers together.

1. In the space provided below, please list all the symptoms you are currently experiencing. You also may look at the attached symptom checklist if you like.

SYMPTOM LIST

1.________________________
2.________________________
3.________________________
4.________________________
5.________________________
6.________________________
7.________________________
8.________________________
9.________________________
10.________________________

2. Based on the symptoms you described in your SYMPTOM LIST, now group the symptoms together that you think are related to each other. For example, if you think that having the sniffles and having a scratchy throat, are related to each other, then you would group them together like this:

GROUP1 sniffles, scratchy throat

If you thought that having a cough was related to **GROUP 1** but not related to a sore toe, you would group them separately like this:

GROUP 1 sniffles, cough
GROUP 2 sore toe

Now please group your symptoms together on the space provided below. It is okay if some of your symptoms cannot be grouped with other symptoms.

SYMPTOM GROUPS

GROUP 1________________
GROUP 2________________
GROUP 3________________
GROUP 4________________
GROUP 5________________
GROUP 6________________

(please turn page over)

SYMPTOM ASSESSMENT INVENTORY

3. What is your understanding of the cause of each group of symptoms?

CAUSE

GROUP 1________________

GROUP 2________________

GROUP 3________________

GROUP 4________________

GROUP 5________________

GROUP 6________________

4. If you are aware of the causes, describe how you obtained this information (doctors, friends, books, etc.)

____

____

5. To the best of your knowledge, what are the prospects of recovering from your pain? (For example, fully recover? Length of time to recover?)

____

____

SAI
Therapist's Form

Client:__________

Date:___D___M___Y Intake ( ) Discharge (pink) Time 3 ( ) Follow-up ( )

Rater: **(circle one)** Isabelle, Mo, Pat, Rhonda

Severity of Medical Condition

On strictly organic criteria, how severe is the patient's medical condition?

1 2 3 4 5 6 7

where 1 = numerous major physical findings (in excess of no.2)
2 = very advanced, multilevel degenerative disc disease (DDD), 2 or more discs protruding
3 = advanced DDD, acute/chronic nerve irritation, herniated disc
4 = some DDD, moderate mechanical pain, facet pain
5 = major soft tissue injury, bad sprain/strain
6 = minor soft tissue injury, pulled muscle
7 = some spasm, tender points

Functional Prognosis

On the basis of physical findings only, what is the prognosis for the patient's condition?

1	**2**	**3**	**4**	**5**	**6**	**7**
Extreme	Very poor	Poor	Moderate	Good	Very good	Excellent

where 1 = unable to work, receives permanent total disability pension
2 = sedentary, part-time work, receives permanent partial (PP) disability pension
3 = permanent modified work, numerous restrictions, receives PP
4 = permanent modified work, few restrictions, PP disability pension
5 = should eventually resume regular work after temporary modified work
6 = discharged to regular duties, little difficulty expected
7 = discharged to regular duties, no difficulty expected

(INTAKE ONLY)

How many weeks do you anticipate the patient will be in therapy at IWRC?
(circle one)

1 2 3 4 5 6 7 8 9 10 11 12 more than 12

What do you expect the patient's time of healing to be, given the type of injury they have?

_____ weeks

Inquiry Information

(see patient copy for their symptom groups)

Specify The Expected Symptom Groups

(expected groups are what **you** think should be in the symptom groups)

Specify the way in which the patient **should have** grouped according to the available medical / psychological information.

1.______________________________
2.______________________________
3.______________________________
4.______________________________
5.______________________________
6.______________________________

PATIENT VS. THERAPIST
Symptom Partitioning

In comparing the **patient's** and **therapist's** groups, to what extent does the **patient's** division of symptoms into groups agree with **yours?**

1	2	3	4	5	6	7
No differentiation at all; purely arbitrary						Perfect differentiation; fully in keeping with the medical/psychological evidence

SAI
Therapist's Form

GROUPING CONTENT ACCURACY

Compare the symptoms in the client's groups with the therapist's groups. Do the symptoms in the client's groups **agree** with the medical (or psychological) evidence? In terms of percent agreement:

1	2	3	4	5	6	7
<14%	14-28%	28-42%	42-56%	56-72%	72-86%	>86%
None	Very poor	Poor	Moderate	Good	Very good	Perfect

Cluster 1						
1	2	3	4	5	6	7
Cluster 2						
1	2	3	4	5	6	7
Cluster 3						
1	2	3	4	5	6	7
Cluster 4						
1	2	3	4	5	6	7
Cluster 5						
1	2	3	4	5	6	7
Cluster 6						
1	2	3	4	5	6	7

CAUSES

Look at each of the client's groups. Based on the client's answer to **QUESTION 3** (Cause), does the **client** understand the cause of the symptoms based on the

medical evidence? i.e., Saying that symptoms are due to lifting a box **IS NOT** an understanding of what is causing the symptom; that is only what led to the trauma. Good answers are muscle strain, disc, etc.

1	2	3	4	5	6	7
None	Very poor	Poor	Moderate	Good	Very good	Perfect
=			=			
Not at all, no understanding of medical/psychological conceptions whatsoever			Some approximation of understanding for some symptoms (half) but very poor for others			

Cluster 1						
1	2	3	4	5	6	7
Cluster 2						
1	2	3	4	5	6	7
Cluster 3						
1	2	3	4	5	6	7
Cluster 4						
1	2	3	4	5	6	7
Cluster 5						
1	2	3	4	5	6	7
Cluster 6						
1	2	3	4	5	6	7

Appendix D

Pennebaker Limbid Languidness Questionnaire

S#________

SYMPTOM CHECKLIST

On the following pages several common symptoms or bodily sensations are listed. Most people have experienced most of them at one time or another. We are currently interested in finding out how common these symptoms are among individuals. All information will be kept confidential.

Please respond by circling the appropriate letter for each question.

(A) Have never or almost never experienced the symptom
(B) Less than 3 or 4 times per year
(C) Every month or so
(D) Every week or so
(E) More than once every week

For example, if your eyes tend to water once every week or so, you would circle D next to #1.

1.	Eyes water	A	B	C	D	E
2.	Itching or painful eyes	A	B	C	D	E
3.	Ringing in ears	A	B	C	D	E
4.	Temporary deafness or hard of hearing	A	B	C	D	E
5.	Lump in throat	A	B	C	D	E
6.	Choking sensations	A	B	C	D	E
7.	Sneezing spells	A	B	C	D	E
8.	Running nose	A	B	C	D	E
9.	Congested nose	A	B	C	D	E
10.	Bleeding nose	A	B	C	D	E
11.	Asthma or wheezing	A	B	C	D	E
12.	Coughing	A	B	C	D	E
13.	Out of breath	A	B	C	D	E
14.	Swollen ankles	A	B	C	D	E
15.	Chest pains	A	B	C	D	E
16.	Racing heart	A	B	C	D	E
17.	Cold hands or feet even in hot weather	A	B	C	D	E
18.	Leg cramps	A	B	C	D	E
19.	Insomnia	A	B	C	D	E
20.	Toothaches	A	B	C	D	E
21.	Upset stomach	A	B	C	D	E
22.	Indigestion	A	B	C	D	E
23.	Heartburn	A	B	C	D	E

[please turn page over]

Please respond by circling the appropriate letter for each question.

(A) Have never or almost never experienced the symptom
(B) Less than 3 or 4 times per year
(C) Every month or so
(D) Every week or so
(E) More than once every week

24.	Severe pains or cramps in stomach	A	B	C	D	E
25.	Diarrhea	A	B	C	D	E
26.	Constipation	A	B	C	D	E
27.	Hemorrhoids	A	B	C	D	E
28.	Swollen joints	A	B	C	D	E
29.	Stiff muscles	A	B	C	D	E
30.	Back pains	A	B	C	D	E
31.	Sensitive or tender skin	A	B	C	D	E
32.	Face flushes	A	B	C	D	E
33.	Severe itching	A	B	C	D	E
34.	Skin breaks out in rash	A	B	C	D	E
35.	Acne or pimples on face	A	B	C	D	E
36.	Acne or pimples other than face	A	B	C	D	E
37.	Boils	A	B	C	D	E
38.	Sweat even in cold weather	A	B	C	D	E
39.	Strong reactions to insect bites	A	B	C	D	E
40.	Headaches	A	B	C	D	E
41.	Sensations of pressure in head	A	B	C	D	E
42.	Hot flashes	A	B	C	D	E
43.	Chills	A	B	C	D	E
44.	Dizziness	A	B	C	D	E
45.	Feel faint	A	B	C	D	E
46.	Numbness or tingling in any part of body	A	B	C	D	E
47.	Twitching of eyelid	A	B	C	D	E
48.	Twitching other than eyelid	A	B	C	D	E
49.	Hands tremble or shake	A	B	C	D	E
50.	Stiff joints	A	B	C	D	E
51.	Sore muscles	A	B	C	D	E
52.	Sore throat	A	B	C	D	E
53.	Sunburn	A	B	C	D	E
54.	Nausea	A	B	C	D	E

Appendix E

Implicit Models of Illness Questionnaire

IMIQ

We would like you to respond to a number of statements about your pain condition. Please indicate your level of agreement or disagreement with each statement by placing an "X" in the most appropriate blank.

1. This condition is controllable.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

2. This condition requires medical attention.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

3. This condition is chronic (long lasting) rather than acute (short-lived).

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

4. This condition is disabling.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

5. This condition is caused by changes in weather.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

6. This condition is painful.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

7. The symptoms of this condition are similar to the common cold.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

8. This condition is permanent rather than temporary.

Strongly Strongly

Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

9. This condition is cured by reduced stress.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

10. This condition is caused by stress or nerves.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

11. This condition goes away on its own.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

12. This condition is caused by one's own behavior.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

13. This condition is cured by proper eating habits.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

14. This condition is controllable by the individual.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

15. The presence of this condition relates to something the individual did.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

16. This condition is contagious.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

17. This condition is caused by germs or virus.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

18. This condition is caused by lack of rest.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

19. This condition is serious.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

20. This condition often comes back.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

21. This condition is changeable.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

22. This condition is caused by a poor diet.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

23. This condition changes over time.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

24. This condition is cured by physical exercise.

Strongly Strongly
Agree _____:_____:_____:_____:_____:_____:_____:_____:_____:Disagree

Appendix G

McGill Pain Questionnaire

Date: ____________________ S#:______

McGill Pain Questionnaire

Some of the words below describe your <u>present</u> or your usual pain. Read each group and underline which of the words - if any - describe your pain. Underline only <u>one</u> word in each group, if you find one that best describes your pain. If none of the words in a group apply to you, do not underline any - move on to consider the next group.

1. Flickering, Quivering, Pulsing, Throbbing, Beating, Pounding
2. Jumping, Flashing, Shooting
3. Pricking, Boring, Drilling, Stabbing, Lancinating
4. Sharp, Cutting, Lacerating
5. Pinching, Pressing, Gnawing, Cramping, Crushing
6. Tugging, Pulling, Wrenching
7. Hot, Burning, Scalding, Searing
8. Tingling, Itchy, Smarting, Stinging
9. Dull, Sore, Hurting, Aching, Heavy
10. Tender, Taut, Rasping, Splitting
11. Tiring, Exhausting
12. Sickening, Suffocating
13. Fearful, Frightening, Terrifying
14. Punishing, Gruelling, Cruel, Vicious, Killing
15. Wretched, Blinding
16. Annoying, Troublesome, Miserable, Intense, Unbearable
17. Spreading, Radiating, Penetrating, Piercing
18. Tight, Numb, Drawing, Squeezing, Tearing
19. Cool, Cold, Freezing
20. Nagging, Nauseating, Agonizing, Dreadful, Torturing

Appendix H

Roland Morris Disability Questionnaire

S: ______

RMDQ

When you are in pain, you may find it difficult to do some of the things you normally do. These are some sentences that people have used to describe themselves when they have pain. When you read them, you may find that some stand out because they describe you. How well do each of the following statements describe the way that you have been <u>over the past two weeks?</u>

		True	**False**
1.	I stay at home most of the time because of pain.	______	______
2.	I change position frequently to try and get comfortable.	______	______
3.	I walk more slowly than usual because of pain.	______	______
4.	Because of pain, I am not doing any of the jobs that I usually do around the house.	______	______
5.	Because of pain, I use a handrail to get upstairs.	______	______
6.	Because of pain, I lie down to rest more often.	______	______
7.	Because of pain, I have to hold onto something to get out of an easy chair.	______	______
8.	Because of pain, I try to get other people to do things for me.	______	______
9.	I get dressed more slowly than usual because of pain.	______	______
10.	I only stand up for short periods of time because of pain.	______	______
11.	Because of pain, I try not to bend or kneel down.	______	______
12.	I find it difficult to get out of a chair because of pain.	______	______
13.	I have pain almost all the time	______	______
14.	I find it difficult to turn over in bed because of pain.	______	______
15.	My appetite is not very good because of pain.	______	______
16.	I have trouble putting on my socks (or stockings) because of pain.	______	______
17.	I only walk short distances because of pain.	______	______
18.	I sleep less well because of pain.	______	______

<u>**PLEASE TURN OVER**</u> >>>>

		True	**False**
19.	Because of pain, I get dressed with help from someone else.	_____	_____
20.	I sit down for most of the day because of pain.	_____	_____
21.	I avoid heavy jobs around the house because of pain.	_____	_____
22.	Because of pain, I am more irritable and bad tempered with people than usual.	_____	_____
23.	Because of pain, I go upstairs more slowly than usual.	_____	_____
24.	I stay in bed most of the time because of pain.	_____	_____

Appendix I

Minnesota Satisfaction Questionnaire

Name ______________________________

SATISFACTION QUESTIONNAIRE

This questionnaire gives you a chance to tell how you feel about your present job. On the basis of your answers and those of thousands of other people throughout the nation, we hope to get a better understanding of the things people like and dislike about their jobs.

Ask yourself: How satisfied am I with this aspect of my job?

VS= I am very satisfied with this aspect of my job
S= I am satisfied with this aspect of my job
N= I can't decide whether I am satisfied or not
DS= I am dissatisfied with this aspect of my job
VDS= I am very dissatisfied with this aspect of my job

	VDS	DS	N	S	VS
1. Being able to keep busy all the time					
2. The chance to work alone on the job					
3. The chance to do different things from time to time					
4. The chance to be "somebody" in the community					
5. The way my boss handles his/her workers					
6. The competence of my supervisor in making decisions					
7. Being able to do things that don't go against my conscience					
8. The way my job provides for steady employment					
9. The chance to do things for other people					
10. The chance to tell people what to do					
11. The chance to do something that makes use of my abilities					
12. The way company policies are put into practice					
13. My pay and the amount of work I do					
14. The chances for advancement on this job					
15. The freedom to use my own judgment					
16. The chance to try my own methods of doing the job					
17. The working conditions					
18. The way my co-workers get along with each other					
19. The praise I get for doing a good job					
20. The feeling of accomplishment I get from the job					