University of Alberta

Developing and testing a model of peoples' beliefs

about chronic pain and recovery

by

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Dedication

I dedicate this achievement firstly, to my husband, Michael. His unconditional love and support nurtures me to the core and his unwavering belief in my ability gives me strength. He supports me, joins me in my triumphs and when I falter he stands strong with me. I feel truly lucky to share this achievement and my life with this wonderful, kind, thoughtful man!

I dedicate it to my parents, Marj and Gil Bouchard, whose continuous unconditional love, support, long-term commitment and dedication allowed me to dream, set my academic aspirations high and helped me realize my dreams. I am forever thankful for their love, helping me believe anything was possible and their support in making anything possible! My parents allowed me to strive and thrive.

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Abstract

Examination of peoples' beliefs about chronic pain and recovery has resulted in identification of a plethora of beliefs and development of many alternative complementary and competing models. The current study is designed to amalgamate this vast literature into a comprehensive model which identifies the relevant beliefs, organizes these beliefs into a comprehensive model and tests the plausibility of that model as a whole. The current study is designed to provide clarity through coalescing the literature in a comprehensive, testable manner. A thorough literature review led to the identification of 16 relevant belief variables: 7 Pain beliefs (Organic, Psychological Influence, Pain as a Mystery, Pain Permanence, Pain Constancy, Self-Blame, and External Pain Responsibility), 1 Internal Pain Control belief, 2 Goal-Pursuit beliefs (Assimilative and Accommodative), 2 'maladaptive' beliefs (Worry and Catastrophizing), 2 'adaptive' beliefs (Acceptance and Pain Self-Efficacy) and 2 Fear-Avoidance beliefs (Physical Activity-Related and Work-Related). Based on the respective literature for each variable these beliefs were organized into three models: The Theoretical Model, The Less Restricted Theoretical Model and The Parsimonious Theoretical Model. Through dual-sampling from the community and a rehabilitation facility 139 self-referred participants completed a 14-item Demographic Survey and questionnaires validated with a chronic pain sample for each variable: The Chronic Pain Acceptance Questionnaire (CPAQ; McCracken, Vowles & Eccleston, 2004), Chronic Pain Intrusion and Accommodation Scale (CPIAS; Jacob, Kerns, Rosenberg & Haythornthwaite, 1993), Fear-Avoidance Beliefs Questionnaire (FABQ; Waddell, Newton, Henderson, Somerville & Main, 1993), Pain Beliefs and Perceptions

Inventory (PBPI; Williams & Thorn, 1989), Pain Beliefs Questionnaire (PBQ; Edwards, Pearce, Turner-Stokes & Jones, 1992), Pain Catastrophizing Scale (PCS; Sullivan, Bishop & Pivik, 1995), Pain Locus of Control Questionnaire (PLC; Main & Waddell, 1991), and the Pain Self-Efficacy Questionnaire (PSEQ; Nicholas, 1989). The results supported the independence of all 16 variables and the Less Restricted Theoretical Model ($\chi^2 = 60.961$, $p = 0.0992$, $df = 48$; RMSEA = 0.0430 [0.0 – 0.0740]; SRMR = 0.0311). The specific features of this passing model along with the research/clinical implications and the limitations are discussed.

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Pain—one of the most pressing issues of our time—*John J. Bonica (1974)*

Chapter 1: Introduction

Chronic pain is described as a benign, intractable, aversive sensation that persists beyond the normal healing time for injury or disease, which typically ranges from 3 (International Association for the Study of Pain [IASP], 2003) to 6 months (King, 2000). It is a global concern estimated to affect from 10.1% (Statistics Canada, 2002) to 45% of the population (Birse & Lander, 1998) with 11% reported as a conservative pooled global prevalence estimate for severe chronic pain. In Canada this equates to at minimum 3.2 million adults reporting severe chronic pain (Statistics Canada, 2002).

This conservative prevalence rate may underestimate "the global toll of [chronic pain]" due to paucity of prevalence studies in under-developed countries and the documented exponential increase in incidence (IASP, 2003, p. 4). However, the findings of a four-year study in the United Kingdom suggest exponential increasing trends in prevalence rates which is related to low recovery rates rather than increased incidence of chronic pain (Elliott, Smith, Hannaford, Smith & Chambers, 2002). For example, Elliott et al. (2002) found the average annual incidence of chronic pain was 8.3% and the average annual recovery rate was only 5.4%. This high prevalence rate coupled with the low recovery rate provides a strong impetus for improving understanding in this area of global concern.

Further compelling the importance of chronic pain research are the far-reaching negative emotional and physical effects. Specifically, chronic pain is associated with depression (Chiu et al., 2005), inactivity (Williams et al., 1996), impairment in occupational, recreational, social and self-care activities (Breivik, Collett, Ventafridda, Cohen & Gallacher, 2006) and disability (Arnoff & Feldman, 2000). People with chronic pain tend to perceive themselves as having greater functional impairment than typical patients with cancer, diabetes, congestive heart failure, myocardial infarction or hypertension (Faneule et al., 1999). These physical, mental, financial, and interpersonal losses "culminate in loss of self-worth, future and hope" (Walker, Sofaer & Holloway, 2006, p. 199). As a result, people with chronic pain have double the risk of death by suicide (Tang & Crane, 2006).

As well, chronic pain is one of the leading causes of long-term disability and has been dubbed a "disability epidemic" (Arnoff & Feldman, 2000, p. 157). In 2002, Statistics Canada reported over 2.4 million adult Canadians indicating chronic pain related activity limitations making this "the most common form of disability among working-age adults" (Statistics Canada, 2002, p. 21). Therefore, chronic pain is "not only a major medical problem but also a major economic problem" (Van Tulder, Koes & Bouter, 1995, p. 233; see also Birse, 1994; Loeser, 1999). The economic costs are incurred indirectly and directly. Indirectly through prevention costs, production losses and related costs to society due to morbidity and mortality, including work absenteeism, disablement and decreased earnings (Birse, 1994; Loeser, 1999; Van Tulder et al., 1995). For example, it is estimated that chronic pain is responsible for

nearly half a million lost work days per year in the United States (United States Bureau of the Census, 1996).

On the other hand, the direct economic costs are incurred through the medical costs of diagnosis, treatment, continuing care, and rehabilitation (Birse, 1994; Loeser, 1999; Van Tulder et al., 1995). For example, the experience of chronic pain leads to frequent use of the healthcare system and medication, despite lack of improvement (Blyth, March, Brnabic & Cousins, 2004; Breivik et al., 2006; Elliott, Smith, Penny, Smith & Chambers, 1999; Linton, 1999) and continued suffering with the negative effects of chronic pain (Crook, Weir & Tunks, 1989; Elliott et al., 1999). Van Tulder et al. (1995) estimated "the indirect costs constituted 93% of the total costs of back pain [and] the direct medical costs contributed only 7%" (p. 233). The total of these direct and indirect costs together is staggering. In the United States this equates to over US$150 billion per year being spent on health care, disability and on related costs (United States Bureau of the Census, 1996) and approximately 1.7% of the gross national product in the Netherlands (Van Tulder et al., 1995). An epidemiological study in Canada suggests global similarities in the prevalence and impact of chronic pain (Birse, 1994; Birse & Lander 1998). Therefore these estimates may act as benchmark for the costs in Canada as well.

In order to decrease the prevalence, decrease the negative effects associated with the physical, emotional, and economic costs of chronic pain and increase the rate of recovery "we must change concepts of pain and disability" (Loeser, 1999, p. 957):

> A historical review (Allan and Waddell 1989) suggested that low back pain has affected man throughout recorded history but that chronic disability due to

> simple backache is a relatively recent and peculiarly Western epidemic (Waddell, 1987b). The increase in low back disability appears to depend more on society's and medicine's understanding and management of low back pain than on any change in the biological disorder. (Waddell, Newton, Henderson, Somerville & Main, 1993, p. 158)

One area that has been particularly fruitful in expanding this understanding is the examination of people's beliefs about chronic pain and recovery.

Beliefs differ from schemas, attributions, appraisals and attitudes. Overall, these constructs are broader, more general and more stable than beliefs. All of these psychological processes are theorized to contribute to the formation of specific thoughts and to underlie affective reactions but in unique ways. Specifically, a schema is "a stable cognitive pattern" used to organize and facilitate information processing of circumstances (Beck, Rush, Shaw & Emery, 1979, p. 12; Sullivan et al., 2001). An attribution is the inference of meaning to one's circumstances (Smith, Haynes, Lazarus & Pope, 1993). Appraisals are judgements regarding the degree of personal threat within these circumstances (Lazarus & Folkman, 1984; Smith et al., 1993). Attitudes reflect ones' stance towards circumstances (Lazarus & Folkman, 1984; Strong et al., 1992; Williams & Thorn, 1989). Finally, a belief encompasses the personal meaning of these circumstances (Fishbein & Ajzen 1975; Jones, Ravey & Steedman, 2005; Strong, Ashton & Chant, 1992).

In comparison with these other cognitive experiences, beliefs have a high degree of personal relevance. Therefore, in the context of chronic pain, pain beliefs are peoples' "conceptualization of what pain is and what pain means for them" (William

& Thorn, 1989, p. 351). For example, peoples' beliefs about chronic pain and recovery reflect their conceptualization about the presence of chronic pain in their life and the personal meaning of what recovery is for them (Lazarus & Folkman, 1984; Strong et al., 1992; Williams & Thorn, 1989).

It is understood that what people believe about their chronic pain and about their recovery are "not... mere artifacts of the chronic pain experience that will disappear once the correct diagnosis and treatment is found" (DeGood & Shuttty, 1992, p. 215); Rather, beliefs are "active and critical ingredient[s]" for recovery (Burns, Kubilus, Bruehl, Harden & Lofland, 2003, p. 81). In fact, research has found psychological factors, such as beliefs, are more influential to recovery than the actual physicality of the injury or diagnosis causing the chronic pain (King, 2000; Nicholson, 2000) and changes in beliefs precede improvements in physical functionality (which is generally used as a measure of recovery) (Burns, Glenn, Bruehl, Harden & Lofland, 2003; Burns, Kubilus, et al., 2003).

However, the study of beliefs has been fraught with difficulty. One problem in particular is the lack of a clear differentiation between the different psychological processes outlined above (i.e., schema, attributions, appraisals, etc.) Second, each program of research utilizes idiosyncratic or discipline-specific nomenclature, promotes alternative psychological processes/beliefs as important and proposes many causal models, both competing and complementary, to explain the interrelation of peoples' beliefs about their chronic pain and their recovery. Evidence of this fracturing in the chronic pain belief literature is found in the sheer number of models that have been put forth to explain the process of belief change in chronic pain: such as, the

Fear-Avoidance Model (Lethem, Slade, Troup & Bentley, 1983; see also Cook, Brawer & Vowles, 2006; Linton, Melin & Gotestam, 1984; Vlaeyen, Kole-Snijders, Boeren & Van Eek, 1995), Philips Model of Chronic Pain (Philips, 1987, 1989), Model of Control (Walker, Akinsanya, Davis & Marcer, 1989, 1990), Dual-Process Model of Coping (Brandtstädter, 1992; Schmitz, Saile & Nigles, 1996), Pain Self-Efficacy Model (Bandura, 1992), Biopsychosocial Model of Low Back Pain and Disability (Waddell et al., 1993), Motivational Model for Pain Self-Management (Heapy et al., 2005; Jensen, Nielson & Kerns, 2003), Cognitive-Behavioral Model of Fear of Movement/(Re)Injury (Vlaeyen, Kole-Snijders, Boern et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, Ruesink & Heuts, 1995), Stress-Response Model (Reitsma & Meijler, 1997), Stage of Change Model (Kerns, Rosenberg, Jamison, Caudill & Haythornthwaite, 1997; see also Glenn & Burns, 2003; Kerns & Rosenberg, 2000), Self-Regulatory Model (Leventhal et al., 1997; see also Hobro, Weinman & Hankins, 2004), Disability Model (Arnstein, Caudill, Mandle, Norris & Beasley, 1999), Depression Model (Arnstein et al., 1999), Self-Pain Enmeshment Model (Pincus & Morley, 2001; see also Morley, Davies & Barton, 2005), Schema Activation Model (Sullivan et al., 2001), Appraisal Model (Sullivan et al., 2001), Attentional Model (Sullivan et al., 2001), Diathesis-Stress Model of Chronic Pain (Turk, 2002), Motivational Model of Self-Management (Jensen, Nielson, et al., 2003), the Anxiety Sensitivity Model (Asmundson & Taylor, 1996; Norton & Asmundson, 2004) and the Return-To-Work Model (Schultz et al., 2004). As well, several qualitative studies have outlined models from their respective disciplines' perspectives (Bullington, Nordemar, Nordemar & Sjostrom-Flanagan, 2003; Gustafsson, Ekholm & Ohman, 2004; Knish &

Calder, 1999; Miles, Curran, Pearce & Allan, 2005; Risdon, Eccleston, Crombez & McCracken, 2003; Smith, 2001; Walker, Holloway & Sofaer, 1999).

Each of these models/theories from methodologically varied studies focus on a different subset of beliefs amongst other selected variables, utilize similar terminology in an idiosyncratic manner, and highlight unique aspects of the experience of chronic pain and recovery from various vantage points (i.e., people with chronic pain, clinicians working with people with chronic pain, literature reviews/analyses and empirical analyses). Overall, the theories and analyses fit within the rubric of cognitive-behavioural theories in that they "posit that patients' beliefs about their pain play a crucial role in their adjustment (Jensen et al., 1991b)" (Jensen, Turner, Romano & Lawler, 1994, p. 301). As well they "emphasize the role of individuals' beliefs and the personal meaning they attributed to various aspects of the experience of pain" and recovery (Novy, Nelson, Francis & Turk, 1995, p. 243). They also recognize the multidimensionality of the pain experience (the physical, behavioural, affective and cognitive dimensions) in a manner that can be translated into psychological interventions to facilitate treatment and recovery from the negative effects of chronic pain (Novy et al., 1995).

However, this thorough and comprehensive explanation of the chronic pain experience leaves the reader with a sense of incoherence, instability and uncertainty about how to best elucidate the relevant beliefs regarding chronic pain and recovery. There is no consensus regarding the nomenclature, relevancy and no overarching theory that summarizes the interaction of peoples' beliefs about chronic pain and recovery in a manner that integrates the research findings. Novy et al. appeal that:

> Theory development [is needed] to explicate the interrelations among facets of chronic pain and the subsequent empirical testing of explicitly specified models [which] will lead to sharpened understanding of chronic pain as well as the identification of the salient facets of the chronic pain experience. Furthermore, such developments can yield an empirically compelling framework in which to organize numerous individual differences associated with chronic pain. Such a framework also has assessment, treatment, and research implications....Such results would help identify, justify, and ultimately enable the provision of the most relevant and necessary constituent services to tackle the costly burden of chronic pain treatment. (p. 244-245)

Purpose

The purpose of this research is to bring order to the existing literature on peoples' beliefs about their chronic pain and their recovery through uniting the chronic pain belief literature into a consensual nomenclature that is then utilized to develop and test a model of peoples' beliefs about chronic pain and recovery. Chapter 2 presents a thorough review of the chronic pain belief literature to identify and operationally define the relevant beliefs into a consensual nomenclature. The focus of the literature review is on studies self-identifying variables as beliefs to ensure inclusively. Then the literature regarding models of peoples' beliefs, in relation to the identified beliefs, is reviewed to develop a Theoretical model of peoples' beliefs about chronic pain and recovery and two alternative models that test the assumptions of the Theoretical Model (i.e., The Parsimonious Theoretical Model and the Less Restricted Theoretical Model). Chapter 2 closes with a visual representation of these models.

Chapter 3 presents a thorough review of Structural Equation Modeling (SEM), the analysis method to test the models. Chapter 4 describes the research procedures and provides the descriptive statistics regarding the sample utilized. Chapter 5 presents the results. Finally, Chapter 6 consists of a discussion and interpretation of the findings, conclusions, clinical implications, limitations of the study, and suggestions for future research.

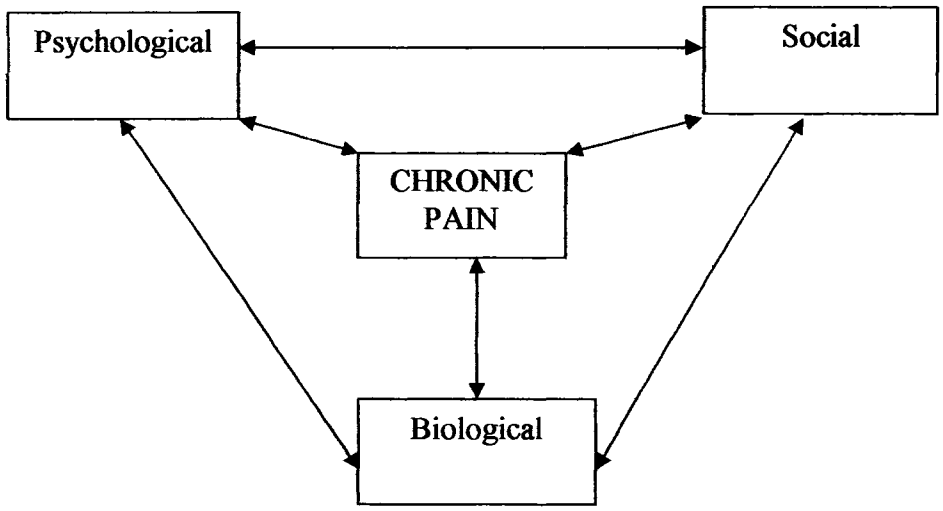
Clinical Relevance

This research is designed to add to the literature base through reducing ambiguity and clarifying peoples' beliefs about chronic pain and recovery. It is proposed this model will help "guide and focus cognitive approaches and allow for evaluation of cognitive changes as a result of treatment" (Philips, 1989, p. 469). This research is designed to facilitate the development of more effective treatment interventions through illuminating specific beliefs to target for change and challenge in treatment (Jensen, Keefe, Lefebvre, Romano & Turner, 2003). It will aid in determining some of the types of thinking patterns people engage in while honouring each person's idiosyncratic experience of chronic pain and of recovery. Thus, it is proposed the resulting model will facilitate decisions regarding the timely, most effective and most efficient utilization of health care services to help reduce the medical, economic and personal costs of chronic pain through informing treatment to facilitate recovery from the negative effects of chronic pain.

Chapter 2: Literature Review

Conceptualization of chronic pain has evolved from a purely physical or purely psychological phenomenon to a complex manifestation of both "physiological and psychological processes" (Novy et al., 1995, p. 241). The main catalyst for this evolution was the reductionistic nature of the single dimension conceptualizations (Birse, 1994; Novy et al., 1995). For example, mind-body dualism, historically one of the primary single dimension conceptualizations of chronic pain, hinges on the assumption that "the solution to a pain problem is to treat the underlying medical condition whereupon pain will resolve" (Nicholson, 2000, p. 58). However, the underlying medical condition is important but it only accounts for a portion of the phenomenon: "Chronic pain is not simply acute pain that last for a long time" (Breen, 2002, p. 52) and not every person with similar injuries develop persistent pain after the acute healing time (King, 2000). Criticism indicates this single dimension perspective is "dually deficient in proposing a mind-body dichotomy and in omitting the role of environmental factors" (Loeser, 1999, p. 959). These single dimension conceptualizations, such as the medical model and the psychological model, do not capture the complex interaction between the multiple factors influencing the experience of pain (Gonzales, Martelli & Baker, 2000; Novy et al., 1995). In the literature the complexity is proposed to encompass influences of the body, mind and environment and is reflected in the Biopsychosocial Model (see Figure 1) (Birket-Smith, 2001; Nicholson, 2000; Novy et al., 1995).

Figure 1. The Biopsychosocial Model.



Adapted from "Somatization and chronic pain," by M. Birket-Smith, 2001, *Acta Anaesthesiologica Scandinavica, 49*, p. 1118. Copyright 2001 by Acta Anaesthesiologica Scandinavica.

The Biopsychosocial Model includes the interaction between the biological and the psychosocial. As Allen and Carlson (2003) noted:

> Psychosocial is the term used to describe those characteristics of individual functioning that are influenced by psychological factors such as an individual's self-perceptions, attitudes, values and emotions and by social factors such as interactions with people, role performance, social conduct and responses to the environment, and the interplay between those internal psychological and external social factors (p. 186)

As presented this model elegantly facilitates the integration of the subjective experience of chronic pain into clinical understanding but the elemental domains of the biological, social and psychological still require further explication to facilitate their use to develop better treatment (Novy et al., 1995). The current study is designed to meet this need. The focus of this research is to clarify the psychological component of the model through creating a consensual nomenclature from the literature to develop and test a model of peoples' beliefs about chronic pain and recovery.

This chapter presents a literature review of chronic pain beliefs and models to create a consensual nomenclature through identifying and operationally defining the relevant beliefs. This is followed with an integration of the research findings and the salient chronic pain belief models for each identified belief to develop a comprehensive model of peoples' beliefs about chronic pain and recovery. The chapter closes with a visual representation of the Theoretical Model to be tested.

Consensual Nomenclature

Models regarding peoples' beliefs about chronic pain and recovery have consensual agreement on the importance of pain beliefs (Boersma & Linton, 2006; Lackner & Quigley, 2005; Lethem et al., 1983; Linton, Buer, Vlaeyen & Hellsing, 2000; Philips, 1987) and the related concept of pain-related fear/anxiety (Lethem et al., 1983; Van den Hout, Vlaeyen, Heuts, Sillen & Willen, 2001; Vlaeyen, Kole-Snijders, Boern et al.,1995; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995; see also Cook et al., 2006; Swinkels-Meewisse, Roelofs, Oostendorp, Verbeek & Vlaeyen, 2006). From the pain belief literature two lines of research expand. One line focuses on the interaction between pain beliefs and 'maladaptive' beliefs such as Worry (De

Vlieger, Crombez & Eccleston, 2006; Lackner & Quigley, 2005; see also Aldrich, Eccleston & Crombez, 2000; Eccleston, Crombez, Aldrich & Stannard, 2001) and Catastrophizing (De Vlieger et al., 2006; Lackner & Quigley, 2005; Sullivan, 2004; Sullivan, Bishop & Pivik, 1995; Sullivan, Lynch & Clark, 2005; Sullivan et al., 2001; Vlaeyen, Kole-Snijders, Boern et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel et al., 1995; see also Cook et al., 2006; Osman et al., 2000; Swinkels-Meewisse et al., 2006; Van Damme, Crombez, Bijttebier, Goubert & Van Houdenhove, 2002; Van den Hout et al., 2001) while another focuses on the interaction between pain beliefs and 'adaptive' beliefs such as Acceptance (Geiser, 1992; McCracken, 1998, 1999; McCracken & Eccleston, 2006; McCracken, Vowles & Eccleston, 2004) and Pain Self-Efficacy (Arnstein, 2000; Arnstein et al., 1999; Bandura, 1977; Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987; Nicholas, 1989, 2007). Two other areas discussed within both the 'adaptive' and 'maladaptive' belief literature are Goal Pursuit beliefs (i.e., Accommodative and Assimilative beliefs) (Haythornthwaite, Menefee, Heinberg & Clark, 1998; Schmitz et al., 1996) and Pain Control beliefs (i.e., locus of control) (Haythornthwaite et al., 1998; Schmitz et al., 1996; Toomey, Mann, Abashian & Thompson-Pope, 1991).

However, within the extant literature, operationalization of these six categories of beliefs (pain beliefs, pain-related fear/anxiety, 'adaptive', 'maladaptive', control and goal pursuit) is idiosyncratic, lacks consistency and cohesiveness. Development of a consensual nomenclature and operationalization is necessary in order to integrate the respective findings for each identified category of beliefs into a comprehensive model.

The following section focuses on expanding and operationalizing the identified beliefs.

Pain Beliefs

Goubert, Crombez and De Bourdeaudhuij (2004) found that pain beliefs are not unique to a chronic pain population but rather are widespread even for individuals that are pain-free (see also Linton et al., 2000). Pain beliefs may even be present "*before* the episode of pain" and interact with the experience of pain to produce negative effects (Linton et al., 2000, p. 1057). Linton et al. (2000) were not able to determine the a priori or post hoc presence of pain beliefs in relation to pain onset, but they did support "the potential role that pain beliefs may have, even very early on, in the development of a...pain problem" (p. 1057; see also Jensen & Karoly, 1992). The conclusion is "pain beliefs might be necessary ... [but they] are not sufficient in the development of chronic disability" (Boersma & Linton, 2006, p. 164).

In line with the Biopsychosocial Model understanding the role of pain beliefs in how people make sense of their pain provides a partial picture and may prove crucial to informing treatment to attenuate the negative effects of chronic pain. Pain beliefs provide the story of pain:

> Implicit within our understanding of pain is the need for it to make sense (Radley, 1994). And to make sense of pain means finding a story that 'works' (Stainton & Rogers, 1991). As the process of 'hunting a cause' takes hold, patients ... develop their different stories to explain how, given the failure of pain to act as a symptom, such a situation of prolonged and distributed

> suffering has arisen (Garro, 1992, 1994)." (Eccleston, Williams & Rogers, 1997, p. 700).

Qualitative studies illuminate some important thematic consistencies within the stories of chronic pain sufferers. Specifically, in his unpublished doctoral dissertation, Williams (1988) informally asked chronic pain sufferers to describe their beliefs about their pain. The responses revealed seven common content areas: (1) beliefs about pain permanence, (2) beliefs about the cause of pain, (3) beliefs about the mysteriousness of pain, (4) beliefs about the impact of pain, (5) beliefs about personal control, (6) beliefs about blame, and (7) beliefs about pain constancy. Through factor analysis these seven content areas were reduced to three factors: Self-Blame beliefs, perceptions of Pain as a Mystery, and beliefs regarding the temporal stability of pain (Williams & Thorn, 1989). Through subsequent analysis these three factors were then expanded into four; Beliefs about the temporal stability of pain were separated into Pain Permanence beliefs and Pain Constancy beliefs (Williams, Robinson & Geisser, 1994). Early studies examining these four factors referred to Pain Permanence as acceptance (Herda, Siegeris & Basler, 1994; Strong et al., 1992):

> But given that this scale is correlated with many constructs typically associated with negative outcome and suffering, the content of this scale is not accurately reflected in a label that could be misconstrued as a positive or helpful belief. (Williams et al., 1994, p. 72)

In a separate line of research examining the stories of people with chronic pain Eccleston et al. (1997) focused on beliefs regarding the cause of pain. They too found the theme of blame but added beliefs about responsibility and identity protection in

regards to advocacy for the legitimacy of the chronic pain. The concept of identity protection is conceptually similar to beliefs about the causal source of the pain and personal control beliefs.

Adding another dimension to the gestalt of pain sufferers stories, Goubert et al. (2004) identified six factors underlying people's beliefs about pain: Pain means harm, belief in a medical cure for pain, belief that limited physical activity is the best treatment for pain, the belief that caution regarding activity is necessary to take care of pain, belief in lack of self-control regarding pain and the belief that pain medication is necessary to treat pain. The results of their analysis reiterate the causal beliefs of organicity and personal control but add activity related pain-beliefs and beliefs about appropriate treatment.

Therefore, the relevant areas for examination appear to be beliefs related to organic causation, responsibility, blame, the perception of pain as a mystery, pain endurance (i.e., Pain Permanence and Pain Constancy) and activity-related pain beliefs. However, coalescing the findings into a consensual nomenclature is difficult given these studies employed alternate methodologies and differential operationalization. Therefore, these conceptual areas are not empirically based but rather based on subjective analysis within the framework provided in the aforementioned pain belief studies. It is proposed that each belief is unique and will contribute uniquely within the Theoretical Model of peoples' beliefs about chronic pain and recovery presented later in this chapter. In the following sections the literature regarding each identified belief area is examined and each section concludes with an operational definition for the pain belief identified. However, although

activity-related beliefs about the perceived consequences of pain were identified as pain beliefs they are not reviewed in this section because these beliefs appear to align more appropriately with the pain-related fear/anxiety beliefs which are expanded upon in a later section.

Organic Causation. Organic causation beliefs are "beliefs about the importance of organic factors in the experience of pain and the logical sequelae of this position" (Edwards, Pearce, Turner-Stokes & Jones, 1992, p. 271). People who hold organic beliefs about their pain believe that "pain is the result of damage to the tissues of the body" and "experiencing pain is a sign that something is wrong with the body" (Walsh & Radcliffe, 2002, p. 24). The origin of organic beliefs has been linked with Western culture (Aldrich et al., 2000; Kulrich & Baker, 1998; Philips, 1987; Williams et al., 1994). Philips (1987) noted the focus on organicity in people with chronic pain "is firmly founded on the traditional medical model (Rachman and Philips, 1975; Philips, 1988).... [where] pain is regarded as a sensory experience that reflects the type and extent of tissue damage or disturbance" (p. 469).

But, organic beliefs are more common for people with chronic pain then for the general population (Edwards et al., 1992). Therefore, it is likely there is an interaction effect where culture and the experience of chronic pain influence the impact of organic causation beliefs. Specifically, Aldrich et al. (2000) hypothesized organic beliefs are part of peoples' interpretation of pain which influences both perception of threat and problem-solving attempts to mitigate the negative effects of chronic pain, such as suffering (see also Lackner & Quigley, 2005).

In other words, in acute pain situations an organic focus may be normal and adaptive (Eccleston et al., 1997; Knish & Calder, 1999; Lethem et al., 1983; McCracken, 1998; Williams et al., 1994). These beliefs may mobilize the person to seek a cure and comply with treatment, given that implicit in these beliefs are specific implications for appropriate treatment (Edwards et al., 1992; Goubert et al., 2004; Kulrich & Baker, 1998; Walsh & Radcliffe, 2002). For example, people holding organic causation beliefs tend to endorse statements indicating "the belief that other people with power (usually doctors) and chance or fate control health status" (Edwards et al., 1992, p. 271; see also Walsh & Radcliffe, 2002). In turn, people who believe pain is controlled by powerful others, such as a doctor or family member may be more likely to comply with treatment (Gibson & Helme, 2000; Schwartz, De Good & Shutty, 1985; Walsh & Radcliffe, 2002).

Alternatively, an organic focus in the context of chronic pain:

> May [make the person] seek out multiple concurrent treatments without benefit... [and]...the chronic disability and recurrent course of chronic pain may reinforce this somatic focus, with the patient's worry and somatic concern reinforced by multiple unsuccessful intervention strategies aimed at pain relief. (Kulrich & Baker, 1998, p. 304)

Specifically, people with chronic pain in the medical process sustain long periods of time waiting, report feeling "insignificant" (p. 623), experience frustration with the lack of a firm diagnosis, feel as though the doctors and health professionals are failing them and feel devalidated with the implication that it was "all in their minds" (p. 624).

Paradoxically, despite the lack of benefit found in the medical services, the ongoing belief that there must be a medical cure for pain is the best predictor of continued medical utilization and dysfunction among individuals with chronic pain (Reitsma & Meijler, 1997), accounting for 8% of the variance in professional service utilization (Jensen & Karoly, 1992). Notably, Jensen and Karoly's (1992) findings regarding the strength and directness of the influence of organic beliefs on disability were not replicated in a subsequent study (Jensen et al., 1994). But the findings do support an association between decreases in organic beliefs and decreased disability. Further, subsequent research suggests changes in organic beliefs do not reflect "a general responsiveness of some patients" and therefore fit within the rubric of beliefs rather than an attitudinal stance (Walsh & Radcliffe, 2002, p. 30).

Therefore, although the directness and magnitude of the association between organic beliefs with dysfunction is less clear (Jensen et al., 1994), the literature maintains organic beliefs are of critical importance in the interpretation of chronic pain. The inclusion of organic beliefs about causation represents an acknowledgement that people's beliefs about the organicity may significantly influence their beliefs about chronic pain and recovery. Specifically, organic causation beliefs have implications regarding who is responsible for treatment and pain management (Walsh & Radcliffe, 2002). Organic Causation beliefs fit under the rubric of 'I am not responsible for my pain management/care'.

Responsibility. On the other hand, under the rubric of 'I am responsible for my pain management/care' are Responsibility beliefs. These are referred to as Psychological Influence beliefs in the literature. These beliefs are related to, but

independent from, beliefs regarding the organicity of the pain (Eccleston et al., 1997). Usage of the term psychological does not refer to the opposite of organic beliefs or 'psychogenic' pain but rather refer to an independent, alternative way of viewing pain that recognizes the influence of personal psychological factors (Edwards et al., 1992; see also Walsh & Radcliffe, 2002). Psychological influence beliefs reflect the "belief that psychological factors may play a role" in the management and treatment of chronic pain and are associated with beliefs about personal "control over their own health and well-being" (Walsh & Radcliffe, 2002, p. 271). As a result, a person could hold organic beliefs and psychological beliefs at the same time without any contradiction (Walsh & Radcliffe, 2002).

Similar to organic beliefs, it is difficult to determine the a priori or post hoc presence of psychological beliefs as dispositional risk-factors or situational reactions to the threat of chronic pain. However, psychological beliefs appear to be more present in a non-chronic pain sample than a chronic pain sample (Edwards et al., 1992) and are not associated with subsequent disability (Walsh & Radcliffe, 2002). Therefore, they may only partially capture the concept of Responsibility indicated in the pain belief literature. The portion missing, based on the chronic pain sufferers stories of responsibility, are beliefs that others are "at least in part responsible for their back pain" and there is nothing sufferers can do until the legitimacy of their claim is accepted (Walker et al., 1999, p. 625). Further, in the chronic pain sufferers' stories there seems to be a common thread of "battling for benefits", trying to "establish a legitimate claim" and feeling as though their lives are "controlled by other people" (Walker et al., 1999, p. 624, 625).

The negative valence of this aspect suggests the pain belief of Responsibility identified in the literature may extend beyond beliefs about personal psychological influence on chronic pain to entail beliefs about the role of others in the cause and treatment of the pain (such as doctors, medication, significant others, etc.) (Main & Waddell, 1991). Meaning responsibility beliefs in the context of chronic pain encompass psychological beliefs, as aforementioned, as well the belief that others are responsible for the pain and treatment. This additional facet aligns with external control (i.e., External Pain Responsibility beliefs). External control beliefs are conceptually similar to organic causation beliefs but add a dimension of helplessness and solicitousness regarding treatment. They reflect beliefs that pain was caused by others and others are responsible for treatment (cf., believing pain is organically caused and requires a cure).

In general, the belief others caused the pain is significantly related to greater distress and behavioural disturbance (DeGood & Kiernan, 1996); However, lower ratings of blame toward others were not associated with acceptance of the pain (a construct that will be fully explained in a latter section) (Rankin & Holttum, 2003). Rather, people with External Pain Responsibility beliefs tend to have higher perceived disability (Walsh & Radcliffe, 2002), higher pain ratings and have been labelled by rehabilitation professionals as "symptom magnifiers" (Kulrich & Baker, 1998, p. 304). Further, externalization of responsibility has been found to be a predictor of negative mental health status (Wu, Tang, & Kwok, 2004). However, as noted in relation to organic beliefs, people who believe that pain is controlled by powerful others, such as

a doctor or family member, may also be more likely to comply with treatment (Gibson & Helme, 2000).

Therefore, operationally, the two dimensions of responsibility are Psychological Influence beliefs and external pain control beliefs (i.e., External Pain Responsibility beliefs) with each construct aligning with an alternative literature base. Despite the similarity of these constructs to each other and to Organic Causation beliefs, they all are uniquely related to disability, pain ratings and treatment participation and therefore, are all included to ensure that their respective unique influence can be examined.

Blame. As noted above, implicit within the rubric of responsibility is the attribution of blame:

> To be in chronic pain is to enter into a relationship with powerful others where one is automatically positioned as dependent, as less morally visible or responsible (Brody, 1987; Frank, 1991), and as a subject within discourses of blame (Hilbert, 1984).... [This results in a] positioning of sufferer as blameworthy, where chronic pain is due to carelessness and an irresponsible approach to medicine. (Eccleston et al., 1997, p. 706)

Williams et al. (1994) refer to this experience as Self-Blame and define it as peoples' belief that they are the appropriate target of blame for their pain experience.

The concept of Self-Blame has not been thoroughly studied and as a result there is a lack of clarity regarding the presence and impact of these beliefs, even amongst the authors utilizing the term (Williams, 1988; Williams & Thorn, 1989; Williams et al., 1994; Eccleston et al., 1997). For example, Williams and Thorn (1989)

found people with chronic pain "rarely blamed themselves for their pain" (p. 357). Further Eccleston et al. (1997) noted people with chronic pain do not endorse feelings of guilt in relation to carelessness or irresponsibility. However, disappointment with self was demonstrated and Philips' (1989) noted the presence of beliefs such as, "I feel guilty about having pain episodes", "I am angry with myself for being in pain", and "I am disappointed with myself for having another bout of pain" (p. 471).

Based on these studies Self-Blame is expected to exert a negative influence. But research findings suggest the impact of Self-Blame beliefs is equivocal or perhaps even helpful to recovery, when present. As evidence of the equivocal nature of Self-Blame one study found viewing pain as their fault was not associated with physical or mental health (Dysvik, Lindstrom, Eikeland & Natvig, 2004) whereas in another study Self-Blame was associated with improved physical functioning (Williams et al., 1994). Further complicating our understanding of the impact of Self-Blame beliefs, Herda et al. (1994) found Self-Blame only had one correlation in their study: "People who blamed themselves for their pain reported to be less frequently in pain" (p.89).

Illuminating the underlying process influencing these unexpected findings, research with severe accident victims found Self-Blame beliefs are associated with increases in peoples' sense of personal control over chaotic negative events (Bulman & Wortman, 1977). This may explain Williams et al. (1994) and Herda et al.'s (1994) findings associating Self-Blame with improved physical functioning and less pain. However, Bulman and Wortman's study did not mention the chronic pain experience of their participants and therefore it is uncertain how these results would generalize to people with chronic pain. But, combining these results suggests within Self-Blame is

the perception there is something the person can do; therefore, Self-Blame may be related to personal control beliefs.

This may also account for the findings of Williams et al. (1994) that Self-Blame beliefs are associated with depressive symptoms (i.e., helplessness). But, of note, self-depreciation in the form of negative self-evaluation (i.e., failure, guilt, punishment, self-dislike and Self-Blame) was not supported as part of depression for people with chronic pain (Morley, Williams & Black, 2002). The implication is the negative connotation Philips (1989) and Eccleston et al. (1997) identified for Self-Blame may not have direct negative consequences but rather may be mediated through control beliefs.

In light of the aforementioned section regarding causation and responsibility, the reasons for the equivocal finding regarding the presence of Self-Blame beliefs and the counterintuitive findings when it is found may be two-fold: (1) current research focusing on Self-Blame beliefs compacts causation and responsibility for pain management into one term, and/or (2) Self-Blame may be related to blame regarding causation in another way. Therefore examining both causation and responsibility in this study will facilitate differentiation of the effect of Self-Blame.

Pain as a Mystery. Beliefs that pain is a mystery are also related to causation beliefs but reflect less specificity (Williams et al., 1994). They encompass:

> The belief that pain is a mysterious, aversive event that is poorly understood. Endorsement of this belief differs from the culturally shared belief that pain serves a useful warning function. Endorsement of this belief may also

represent a temporary measure of how well patients are proceeding in formulating a new understanding of their pain. (Williams et al., 1994, p. 72)

The primary research program for Mystery beliefs, Williams et al.'s research program, has demonstrated the utility of these beliefs independent from organicity beliefs (Eccleston et al., 1997; Williams, 1988; Williams et al., 1994; Williams & Thorn, 1989). As well, viewing pain as a mysterious, poorly understood experience results in poorer mental health (Dysvik et al., 2004; Herda et al., 1994; Williams & Thorn, 1989), is associated with both depression and anxiety symptoms (Williams et al., 1994), poor response to treatment (William & Thorn, 1989) and greater interference of pain on daily functioning (Strong, Ashton, Cramond & Chant, 1990).

Mystery beliefs are also associated with trait-anxiety and the negative coping strategy of catastrophizing (Herda et al., 1994). Specifically people endorsing both Pain Permanence and Mystery beliefs tend to report high levels of catastrophizing and report feeling less able to control their pain (Williams & Keefe, 1991). In addition, Yong (2006) suggests:

> Belief in the mysterious nature of pain may influence when and how pain is communicated to others (Beese et al., 1999). The cautious reluctance in reporting pain may stem from one or more of the following: the fear of not being believed given the chronicity of their pain problem, the desire to be a 'good' patient, not being a burden on others, and the need to maintain a relationship with significant others (Morley et al., 2000). (p. 400)

As a result of these findings, in conjunction with the identification of Mystery and Organic beliefs in the pain belief literature discussed at the beginning of this

section, Pain as a Mystery beliefs are included in this analysis. The purpose of their inclusion is to test the independence of each belief in examining their respective influence in the model of peoples' beliefs about chronic pain and recovery.

Pain Endurance. Pain Endurance beliefs are:

> ...future-oriented beliefs that pain is and will be an enduring part of life. Endorsement of this belief differs from a common culturally shared belief that pain is time-limited and fixable. (Williams et al., 1994, p. 72)

These beliefs are related to decreased compliance with treatment and the physical and behavioural dimensions of the pain experience such as pain intensity (Herda et al., 1994; Williams et al., 1994; Williams & Thorn, 1989). However, some research has had inconsistent findings in relation to this association. Specifically, Dysvik et al. (2004) noted viewing pain as constant or permanent was not associated with physical or mental health.

Part of the reason for this inconsistency may be related to the factor structure reorganization separating Pain Endurance into two independent components: Pain Constancy beliefs and Pain Permanence beliefs (Herda et al., 1994; Williams et al., 1994). Pain Permanence retains the same content valence as the original Pain Endurance beliefs (William et al., 1994): The belief that "pain will be an enduring part of life" that "will persist into the future" (Williams et al., 1994, p. 74, 76). Pain Permanence is associated with anxiety but not with self-reported pain intensity (Williams et al., 1994). As well, these authors' suggest people may hold these beliefs rigidly and may not see the value to attempt pain control, given pain is viewed as a permanent condition (Williams & Thorn, 1989).

On the other hand, Pain Constancy entails the belief that pain is "a constant experience and pain-free intervals are an exception" (Herda et al., 1994, p. 89). Pain Constancy is associated with greater self-reported pain intensity (Williams et al., 1994), trait-anxiety, catastrophizing cognitions, more frequent pain episodes, and more general physical troubles (Herda et al., 1994). As well, the belief pain is unchanging and stable is associated with poor compliance with both physically and psychologically oriented treatments (Williams & Thorn, 1989).

These two related but independent dimensions reflect pain-specific beliefs about disease identity/symptom level and timeline/duration (Leventhal et al., 1997). As a result the constructs of Pain Constancy and Pain Permanence will be utilized rather than the overarching construct of Pain Endurance. However, similar to many of the beliefs identified thus far, the inconsistencies in the research make attributions regarding the unique impact of these beliefs tenuous. But, the findings reported suggest both represent unique aspects of pain beliefs that are likely related to the intricacies of the situation, thus, they are included in this analysis.

Pain-Related Fear/Anxiety Beliefs

Pain-Related Fear/Anxiety beliefs focus on the consequences of pain and the impact of activity on pain (Goubert et al., 2004; Williams, 1988). Operationalization of fear and anxiety beliefs includes a wide nomenclature: fear of pain (Asmundson, Norton & Allerdings, 1997; Asmundson & Taylor, 1996; De Gier, Peters & Vlaeyen, 2003; Lethem et al., 1983; Pfingsten et al., 2001); pain-related fear (Sieben, Portegijs, Vlaeyen & Knottnerus, 2005); fear of movement/(re)injury (Leeuw et al., 2007; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995); kinesiophobia (Kori, Miller & Todd,

1990); fear-avoidance beliefs (Al-Obaidi, Beattie, Al-Zoabi & Al-Wekeel, 2005; Al-Obaidi, Al-Zoabi, Al-Shuwqie, Al-Zaabie & Nelson, 2003; Boersma & Linton, 2006; Brox, Storheim, Holm, Friis & Reikeras, 2005; Ciccone & Just, 2001; Fritz & George, 2002; Fritz, George & Delitto, 2001; Linton et al., 2000; Pfingsten et al., 2001; Staerkle et al., 2004; Vowles & Gross, 2003; Waddell et al., 1993); fear in general (McCracken, Gross, Aikens & Carnrike, 1996; Vowles & Gross, 2003); pain expectancies (Ciccone & Just, 2001; Crombez et al., 1996); pain-related anxiety (McCracken & Gross, 1993; McCracken et al., 1996; McCracken, Gross, Sorg & Edmands, 1993; McCracken, Zayfert & Gross, 1992; Vlayen, Kole-Snijders, Boeren, et al., 1995; Waddell et al., 1993) and anxiety sensitivity (Asmundson, Kuperos & Norton, 1997; Asmundson, Norton & Norton, 1999; Asmundson & Taylor, 1996; Norton & Asmundson, 2004; McNally & Lorenz, 1987; Peterson & Reiss, 1992; Reiss, 1987, 1991; Reiss & McNally, 1985; Reiss, Peterson, Gursky & McNally, 1986; Zvolensky, Goodie, McNeil, Sperry & Sorrel, 2001).

However, this diversity in terminology does not reflect clarity in understanding the uniqueness of the constructs. For example, some studies utilize the term fear-avoidance beliefs as a synonym for the more general term of pain-related fear (Al-Obaidi et al., 2003), as a synonym for fear of pain (Staerkle et al., 2004; Waddell et al., 1993) or as an all encompassing term that combines fear of pain and fear of injury (Ciccone & Just, 2001; Crombez, Vlaeyen, Heuts & Lysens, 1999). Others utilize the term pain-related anxiety as a synonym for fear of pain (Asmundson et al., 1997) or expectancy as a synonym for beliefs indicating "expectancies explicitly identify the cognitive basis(es) of fear avoidance, namely fear of pain and injury" (Ciccone & Just,

2001, p. 191). On the other hand, there is evidence anticipated pain, such as pain expectancies do "not necessarily predict a subject's ability or beliefs related to performing tasks" (Al-Obaidi et al., 2005, p. 1056; see also Crombez et al., 1999). Each area of study in the fear and anxiety-based nomenclature has propelled our knowledge forward but the inconsistent operationalizations result in imprecision and complexity in the clarifying the role of fear/anxiety beliefs in the context of chronic pain.

Further, synonymous use of anxiety and fear is doubly complex in that there is much debate regarding the independence of these constructs. On one side of the debate, there is evidence that fear-based beliefs are distinct from pain-related anxiety (Crombez et al., 1999; McCracken & Gross, 1998; Zvolensky et al., 2001). However, on the other side of the debate, consistently similar associations with disability suggest colinearity between fear and anxiety beliefs (Al-Obaidi et al., 2003; Asmundson & Taylor, 1996; Ciccone & Just, 2001; Crombez et al., 1999; Fritz et al., 2001; McCracken & Gross, 1993; McCracken et al.,1993; McCracken et al., 1992; Staerkle et al., 2004; Vlayen, Kole-Snijders, Boeren, et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, et al.,, 1995; Vowles & Gross, 2003; Waddell et al., 1993).

Further complicating the situation, there is little consensus and many contradictory theories regarding how fear-based beliefs, such as fear of pain, are associated with chronic pain. In their foundational study regarding fear of pain, Lethem et al. (1983) theorized peoples' level of fear of pain shape their reactions. A high level of fear would result in "exaggerated pain perception" (p. 401). On the other hand, people with a low level of fear are "likely to undertake an increasing range of

physical and social activities, either alone or under supervision; to test the reality of their pain experience at every stage; and to calibrate their pain experience against the nature of the sensory-discriminative stimulus" (p. 404). In other words, a low level of fear facilitates an adaptive response with positive implications for recovery whereas high fear of pain contributes to a maladaptive reaction and has negative repercussions for recovery (Lethem et al., 1983).

In line with this view, De Gier et al. (2003) found lower fear of pain results in higher activity tolerance but this unique influence disappears when controlling for the influence of pain intensity (cf., Al-Obaidi, Nelson, Al-Awadhi & Al-Shuwaie, 2000; Crombez, Vervaet, Lysens, Baeyens & Eelen, 1998; Crombez et al., 1999; Vlaeyen, Kole-Snijders, Boeren et al., 1995). Fear of pain is highly correlated to pain intensity (De Gier et al., 2003) and greater fear of pain is associated with greater expectation and overestimation of pain (Crombez et al., 1996; McCracken et al., 1993). But many people with chronic pain do not overestimate their pain (Rachman, 1994) and may even tend to underestimate expectations for pain (Murphy, Lindsay & Williams, 1997). Contrary to expected, underestimation of pain intensity is also related to negative repercussions (Arntz & Peters, 1995). This suggests pain intensity provides only a partial explanation and perhaps pain-related fear/anxiety may also exert an independent influence. However, there is some evidence questioning the relevance of pain-related fear/anxiety given levels of fear and anxiety sensitivity in chronic pain samples do not differ between people with chronic pain who are "dysfunctional", "interpersonally distressed" and "adaptive copers" (Asmundson et al., 1997) nor do

they differ based on diagnosis (Rose, Klenerman, Atchison & Slade, 1987) (cf., Lethem et al., 1983).

It is proposed these alternative findings may reflect the breadth and all-encompassing nature of operationalization in the nomenclature rather than a reflection of the inappropriateness of the construct. To clarify this debate, given the purpose of this study is to focus on beliefs, examination of the usage of the terms anxiety and fear and the respective findings in the literature may provide some direction, specifically when viewed with the lens of clarifying activity-related pain beliefs, which were identified as the relevant pain belief.

The term fear in the literature maintains the underlying beliefs as an important part of peoples' fear response but usage also simultaneously includes discussion of just the underlying belief structure (Al-Obaidi et al., 2005; Al-Obaidi et al., 2003; Boersma & Linton, 2006; Brox et al., 2005; Ciccone & Just, 2001; Fritz & George, 2002; Fritz et al., 2001; Linton et al., 2000; Pfingsten et al., 2001; Staerkle et al., 2004; Vowles & Gross, 2003; Waddell et al., 1993). As a result, usage of the term fear describes the process but does not necessarily illuminate the specific underlying beliefs due to the inherent inclusiveness in operationalization of fear.

In regards to anxiety, research provides more illumination for the underlying beliefs for the emotions of both fear and anxiety, specifically through examination of anxiety sensitivity (Asmundson et al., 1997; Asmundson et al., 1999; Asmundson & Taylor, 1996; Norton & Asmundson, 2004; McNally & Lorenz, 1987; Peterson & Reiss, 1992; Reiss, 1987, 1991; Reiss & McNally, 1985; Reiss et al., 1986; Zvolensky

et al., 2001). For example, Zinbarg, Barlow and Brown (1997) suggest for people with anxiety disorders:

> Palpitations are feared if persons believe they will lead to cardiac arrest, dizziness or concentration difficulties are feared if individuals believe they will lead to insanity, and trembling or sweating is feared if persons believe it will elicit rejection or ridicule from others. (Asmundson & Taylor, 1996, p. 578)

This view underscores the differentiation of fear and anxiety and highlights the necessity of specificity in the cognitive foundation for these emotions in conjunction with the associated behavioural responses (Fritz & George, 2002; Staerkle et al., 2004; Waddell et al., 1993). Therefore, peoples' *specific* beliefs about pain are important determinants that shape their response to pain. Within a chronic pain population an anxious or fearful response "can be predicted more accurately with higher levels of correspondence between a particular anxiety sensitivity domain and events that closely match that fear" (Zvolensky et al., 2001, p. 683). Suggesting it may not be the presence of fear of pain beliefs but rather the specificity of those beliefs: Pain-specific fear beliefs differentiated the "dysfunctional" group from the other groups (Asmundson et al., 1997, p. 231).

This is in line with the majority of research indicating pain-related anxiety is more predictive of disability, pain severity and pain behaviour than trait anxiety, which is a more generalized condition (McCracken et al., 1996; McCracken et al., 1992). Specifically, activity-related pain anxiety and pain-related fear is more predictive of pain and pain-related disability than more general measures of anxiety and fear of pain in general (McCracken et al., 1996; Vowles & Gross, 2003). For

example, work-related fear of pain was most predictive of work-related disability (Fritz & George, 2002; Fritz et al., 2001; Van Vuuren, Van Heerden, Becker, Zinzen & Meeusen, 2006).

Further emphasizing the importance of specificity in the beliefs, Ochsner et al. (2006) examined the comparative neural correlates of generalized anxiety and pain-related fear/anxiety. They found, in contrast to generalized anxiety, pain-related fear/anxiety was associated with the anterior and posterior cingulated cortices. They concluded that:

> Pain may be a primitive signal for behavioural change.... [Specifically] the posterior cingulate has been associated with evaluating the valence of external and potentially threatening stimuli (Maddock et al., 1997, 2003). Recruitment of the anterior and posterior cingulated cortices may suggest that individuals high in fear of pain closely monitor and evaluate the potential threat value of a painful stimulus. (p. 73-74)

Therefore, they suggest anxiety and fear responses are the result of which appraisal system perceives the pain.

Ochsner et al's (2006) findings imply the appraisal system for people with chronic pain may be skewed (see also Linton et al., 2000). But Asmundson et al. (1997) found no attention bias toward pain-related stimuli when a group with chronic pain was compared to a control group. Further, Asmundson et al. found people with pain and low anxiety sensitivity tended to shift their attention away from pain-related stimuli; whereas those with pain and high anxiety sensitivity did not alter their response based on the relevance or position of the stimuli. These findings contradict

the role of anxiety sensitivity as causing increased saliency in perception of threatening material (Asmundson et al., 1997; Asmundson et al., 1999; Asmundson & Taylor, 1996; McNally & Lorenz, 1987; Norton & Asmundson, 2004; Peterson & Reiss, 1992; Reiss, 1987, 1991; Reiss & McNally, 1985; Reiss et al., 1986; Zvolensky et al., 2001). Further contradicting skewed perception, De Gier et al. (2003) found the presence of an acute threat did not lead to more attentional interference in highly fearful people. However, interestingly, findings suggest avoidance rather than magnification of the perception of threat may actually be related to dysfunction. Specifically, research suggests "efforts to avoid pain through attentional diversion or suppression may prolong pain rather than reduce it, and could contribute to or maintain health anxiety" (Hadjistavropoulos, Craig & Hadjistavropoulos, 1998, p. 151).

Adding even further to the exigent nature of this area of research, there is evidence pain-related anxiety is sensitized with chronic pain (Asmundson et al., 1997; Asmundson & Taylor, 1996; Norton & Asmundson, 2004; Zvolensky et al., 2001). This anxiety sensitivity has been described as a "dispositional trait" (Asmundson et al., 1999, p. 109) in accordance with research regarding anxiety sensitivity with anxiety disorders (McNally & Lorenz, 1987; Peterson & Reiss, 1992; Reiss, 1987; 1991; Reiss & McNally, 1985; Reiss et al., 1986). It is also described as "an individual difference dimension that increases risk for pain responding, particularly in anxiety-provoking contexts" (Zvolensky et al., 2001, p. 684). But the neural correlates do not fully support this dispositional attribution given the threat appraisal system does not appear to be suspect and the contradicting finding in neural activation between pain-

related anxiety and generalized anxiety (Ochsner et al., 2006). Thus, this sensitivity may not be a reflection of "a dispositional tendency to fear symptoms" in general (Asmundson et al., 1999, p. 109).

Genetic research provides further evidence against anxiety sensitivity as a dispositional trait citing that the pain-perception nervous system in humans, as a whole, has followed a positive evolution (Yang et al., 2005, p. 31). Sensitization "of [the] human sensory system, especially the sensory pathway of detecting pain stimuli... could be a refined self-protection mechanism developed during human evolution" (Yang et al., 2005, p. 35). In accordance with Asmundson et al.'s (1999) position, the propensity to perceive pain is sensitized (Yang et al., 2005). But in contrast to Asmundson et al.'s (1999) position, this sensitization is due to an evolutionary trend in *all* humans (individual differences) not reflective of a maladaptive dispositional trait (Yang et al., 2005). Further, in support of the individual difference argument, the neural correlates findings suggest interpretation of the threat of pain symptoms reflects a different process than with generalized anxiety (Ochsner et al., 2006).

Therefore, research suggests anxiety and fear based beliefs are general terms that encompass the emotional and behavioural response to a cognitive interpretation of an event, or threat. It is proposed this sensitized propensity and vigilant awareness in the monitoring of the threat of pain are interpreted through the lens of peoples' pain beliefs. Thereby, the negative effects of pain in conjunction with this sensitized biological preparedness to perceive pain may be mitigated if the cognitive perception of pain as a threat could be reduced. However, given "fears may be quite idiosyncratic

in different patients" (De Gier et al., 2003, p. 128) specificity in examination of these beliefs is necessary. Given the focus of this research is on people's beliefs about pain and recovery and the aforementioned relevancy of specific activity-related pain beliefs it is proposed Physical Activity-Related Fear-Avoidance beliefs and Work-Related Fear-Avoidance beliefs are the relevant constructs.

Work-Related Fear-Avoidance Beliefs. These beliefs represent beliefs about how work influences pain (Waddell et al., 1993). Work-Related Fear-Avoidance beliefs are associated with work loss and disability with activities of daily living, accounting for 26% and 23% of the variance respectively after controlling for pain severity (Waddell et al., 1993). In a subsequent study, Ciccone and Just (2001) found Work-Related Fear-Avoidance beliefs accounted for a smaller portion of the variance in disability in a chronic pain and acute pain samples, 12% and 10% respectively. Despite the differences in magnitude, evidence suggests these beliefs are significantly related to and helpful in predicting self-reported disability (Buer & Linton, 2002; Crombez et al., 1999; Fritz et al., 2001; Pfingsten et al., 2001; Vowles & Gross, 2003). These beliefs also predict help seeking behaviours (McCracken et al., 1996).

Activity-Related Fear-Avoidance Beliefs. Similarly, Activity-Related Fear-Avoidance beliefs represent beliefs about how physical activity influences pain (Waddell et al., 1993). Activity-Related Fear-Avoidance beliefs explain a lesser portion of the variance of disability in activities of daily living, 9% (Waddell et al., 1993). But, even this reduced magnitude of effect supports a significant relationship with and helpfulness in predicting self-reported disability (Buer & Linton, 2002; Crombez et al., 1999; Pfingsten et al., 2001). These beliefs are generally studied in

work-related injury populations and less frequently in the general population. Thus, it is not clear if the magnitude of effect is a reflection of a population characteristic or a result of the work-focused samples in studies.

Examining the effects of both Physical Activity-Related and Work-Related Fear-Avoidance beliefs on peoples' beliefs about chronic pain and recovery will help establish the utility of both and ensure the impact of specificity in these beliefs is recognized, which was identified as an important component of peoples' beliefs.

'Maladaptive' Beliefs

The 'maladaptive' beliefs identified are Worry and Catastrophizing. However, the 'maladaptive' nature of these beliefs was brought into question; "Given the extensive distress and disruption associated with chronic pain, the absence [rather than the presence] of worry would be considered abnormal" (Aldrich et al., 2000, p. 463). De Vlieger et al. (2006) add Worry and Catastrophizing can be considered a "normal response to threat that occurs in stressful situations" (p. 460) and "may be the natural by-product...of repeated attempts to solve the insoluble problem of chronic pain" (De Vlieger et al., 2006, p. 143). Supporting the normalcy of the response Catastrophizing is not as prevalent in the general population as it is in chronic pain populations, which suggests the experience of chronic pain may influence expression of these 'maladaptive' beliefs (Linton et al., 2000). In other words, in the context of chronic pain these 'maladaptive' beliefs may not reflect a pre-existing psychopathology, a deficit in problem-solving or a negativistic general stance toward the world, as in anxiety based disorders (Aldrich et al., 2000; Linton et al., 2000). If they were pre-

existing pathology or a general stance toward the world they should be just as prevalent in the general population as in a chronic pain population, as risk factors.

As well, Worry in the context of chronic pain "is not a form of a Generalized Anxiety Disorder" and does not arise from a "general disposition to worry" (Eccleston et al., 2001, p. 316). In fact, De Vlieger et al. (2006) found the level of generalized Worry and problem-solving competence is normal and does not differ between people with chronic pain and a non-clinical sample: Even after "12 years of failing to solve the problem of chronic pain, general problem solving confidence and attempts to solve [other] problems appear to be unaffected" (DeVlieger et al., 2006, p. 143; see also Van den Hout et al., 2001). But, repeated failure to control pain was associated with negative emotions such as anger and increased physiological responding such as heart rate, but not increased pain levels (Janssen, Spinhoven & Arntz, 2004). Therefore, although "worry does not appear to be psychopathologically relevant" it is influential on peoples' experience of chronic pain (DeVlieger et al., 2006, p. 142).

In other words, there are negative effects of Worry and Catastrophizing. Specifically, generalized Worry has been linked to the affective dimensions of pain, such as suffering which entailed perceptions of pain as uncontrollable and unpredictable (Lackner & Quigley, 2005). Catastrophizing has been linked to heightened pain experience (Haythornthwaite, Clark, Pappagallo & Raja, 2003; Haythornthwaite, Lawrence, & Fauerbach, 2001; Jensen et al., 2002; Sullivan et al., 1995; Sullivan, Rouse, Bishop & Johnston, 1997; Sullivan, Stanish, Sullivan, Tripp, 2002; Turner, Jensen, Warms & Cardenas, 2002). Therefore, it is proposed

'maladaptive' beliefs, such as Worry and Catastrophizing, have negative repercussions for people but they can be framed as part of the normal reaction to chronic pain.

Worry. According to Aldrich et al. (2000) worrying is defined as a "purposeful activity" (p. 460) entailing a "heightened awareness" (p. 459) and "vigilance" to information that is threatening to the self or the body (p. 460). They infer but do not explicitly state that Worry is a "purposeful [cognitive] activity" (p. 460) arising out of attempts to solve the problem of chronic pain. Eccleston et al. (2001) examined the phenomenology of Worry and defined it as "negative and aversive rumination about pain and its consequences" (p. 309). These studies support the view that chronic pain presents a perceived threat to the self and the body and "a normal response to that threat may be chronic worry about pain" (Aldrich et al., 2000; Eccleston et al., 2001, p. 317):

> Engagement in problem solving attempts functions not [only], paradoxically, to solve problems, but as a strategy for managing anxiety. Simply put, if one is vigilant to pain related threat, catastrophizing about the consequences of pain, and worrying about a negative future, then engaging in problem solving, however, ineffective, may be preferable to inaction. (De Vlieger et al., 2006, p. 143)

This characterization of Worry dovetails nicely with Harvey and McGuire's (2000) findings that active suppression of pain-related thoughts facilitated pain management (i.e., management of the negative effects of chronic pain) while "attention to the pain resulted in an enduring increase in pain-related thoughts beyond the period where the instruction to attend was administered" (p. 1123).

Catastrophizing. Catastrophizing in the context of chronic pain is a unitary concept comprised of "negative pain-related cognitions" reflecting magnification, rumination and helplessness (Sullivan et al., 2001, p. 53; see also Osman et al., 1997; Osman et al., 2000; Sullivan et al., 1995). It is defined as "an exaggerated negative 'mental set' brought to bear during actual or anticipated painful experience" (Sullivan et al., 2001, p. 53) and is associated with worse pain, disability and mood (Turner, Mancl & Aaron, 2004). Lamé, Peters, Vlaeyen, Kleef and Patijn (2005) found Catastrophizing about pain is more strongly associated with decreased quality of life (social functioning, vitality, mental health and general health) than pain intensity.

In research Catastrophizing is also referred to as a coping strategy (Rosenstiel & Keefe, 1983) and associated with personality variables such as trait anxiety and external locus of control (Chaves & Brown, 1987). But, Catastrophizing as a coping strategy is not correlated with other forms of coping (Parker, Smarr, Bruesher, et al., 1989; Rosensteil & Keefe, 1983; Turner & Clancy, 1986) and, therefore, may represent the "broader dimension" of an individual's approach to coping rather than a specific coping strategy (Sullivan et al., 2001, p. 60).

Further, Sullivan (2004) adds "catastrophic thinking may have its origins in reality" and may not reflect underlying psychopathology or maladaptive coping (p. 8). For instance, Peterson and Moon (1999) note exposure to traumatic life events may lead to the emergence of catastrophic thinking. Based on these findings the concept of Catastrophizing can be broken down into catastrophic thinking and catastrophic behaviour (Thorn, Ward, Sullivan & Boothby, 2003). Woby, Watson, Roach and Urmston (2005) support this differentiation noting "coping strategy use might only be

related to levels of adjustment via the effect it has on catastrophic thinking and self-efficacy for pain control" (p. 100). Therefore, the focus of our examination is catastrophic thinking.

The constructs of Catastrophizing and Worry are similar. But, Eccleston et al. (2001) differentiate Worry from catastrophic thinking and from pain-related fear. Specifically, Lackner and Quigley (2005) differentiated generalized worry from Catastrophizing in a chronic pain sample, where they shared less than 13% variance. As well, findings with an acute sample suggest catastrophic thinking is independent of pain-related fear (Swinkels-Meewisse et al., 2006). As well,

In the literature three aspects are presented as components of Catastrophizing through use of the Pain Catastrophizing Scale (PCS): Rumination, Magnification and Helplessness. Rumination aligns with Worry, Helplessness aligns with external locus of control beliefs (operationalized as External Pain Responsibility beliefs) and Magnification aligns with Catastrophizing. In support of this differentiation, Sullivan et al. (2001) found that only Magnification is associated with pain intensity.

Adaptive Beliefs

The second line of research expanding from pain beliefs focuses on 'adaptive' beliefs, such as Acceptance and Pain Self-Efficacy. McCracken (1998) summarize:

> ...when usual forms of instrumental control over an aversive event are unattainable the individual may strive for understanding and acceptance of the event. When these are achieved the individual experiences fewer negative emotional consequences (Rothembaum, Weisz & Snyder, 1982).... Those more unaccepting of pain appear to be facing the distress that comes from

> attempting to control an unchangeable aversive experience (Thompson, 1981; Burger, 1988).... (p. 22 & 25)

Acceptance. The construct of Acceptance combines acceptance of pain as the general construct with an element of acceptance of disability (McCracken, 1998). Acceptance is:

> Acknowledging that one has pain, giving up unproductive attempts to control pain, acting as if pain does not necessarily imply disability, and being able to commit one's efforts toward living a satisfying life despite pain.... [While] giving up on attempts to control an aversive experience may at first appear unadaptive.... there are clearly instances when it is helpful to do so (Burger, 1988; Thompson, 1981).... (McCracken, 1998, pp. 22 & 25)

The negative impact of the 'maladaptive' beliefs on the experience of pain (Aldrich et al., 2000; DeVlieger et al., 2006; Lackner & Quigley, 2005) acts as an alternative reaction to Acceptance, which alters attempts to control or avoid the experience of chronic pain (McCracken & Eccleston, 2006). In support of this differentiation, there is support for the independence of Acceptance and Catastrophizing (Viane et al., 2003).

Acceptance does not occur as a result of decreasing levels of pain or as a method to reduce pain but rather as "a way to function regardless of the level of pain present" (McCracken & Eccleston, 2006, p. 28; see also McCracken, 1998; McCracken, Spertus, Janeck, Sinclair & Wetzel, 1999). Acceptance is "the need to focus away from pain to non-pain aspects of life, a recognition that cure of pain is unlikely, and a rejection of any suggestion that 'acceptance' is a sign of personal

failure" (Viane, Crombez, Eccleston, Devulder & De Corte, 2004, p. 283). Findings suggest increased Acceptance leads to improved well-being (McCracken & Eccleston, 2003; McCracken & Eccleston, 2005; McCracken, Vowles & Eccleston, 2005; Viane et al., 2004; Viane et al., 2003) and decreased distress and disability (McCracken & Eccleston, 2003; McCracken & Eccleston, 2006; Viane et al., 2004).

Acceptance reflects "a disengagement from struggling with pain, a realistic approach to pain and pain-related circumstances, an engagement in positive everyday activities" (McCracken & Eccleston, 2003, p. 198). The concept of Acceptance reflects the interaction of pain beliefs and personal control beliefs (much like the 'maladaptive' beliefs mentioned earlier). It reflects a change in the interpretation of the threat of chronic pain and is related to but differentiated from confidence to have a life despite the pain, otherwise known as Pain Self-Efficacy (the other 'adaptive' belief) (McCracken, Carson, Eccleston & Keefe, 2004).

Pain Self-Efficacy. Pain Self-Efficacy beliefs is an offshoot of General Self-Efficacy theory (Arnstein, 2000; Bandura, 1977). This construct refers to peoples' confidence in their ability to perform various activities of daily living given the presence of chronic pain (Altmaier, Russell, Kao, Lehmann & Weinstein, 1993). Based on Bandura's (1977) Self-Efficacy theory four sources of information influence Pain Self-Efficacy, including performance accomplishments, vicarious experiences, verbal persuasion, and physiological states. However, the causal sources are not delineated with this level of clarity in the chronic pain literature despite findings that the concept of Pain Self-Efficacy is useful as an end-product of other beliefs. Despite

Bandura's foundational conceptualization the causal sources of Pain Self-Efficacy are of little focus.

Rather research focuses on the influence of Pain Self-Efficacy on recovery to suggest the main influence of Pain Self-Efficacy is maintenance of gains rather than as a causal agent of the gains (Altmaier et al., 1993). In this examination, Arnstein (2000) found Pain Self-Efficacy mediates the effects of pain intensity on disability (see also Bandura, 1992; Bandura et al., 1987): "A doubting of one own abilities (low self-efficacy), even if pain is mild or moderate in intensity, may be disabling" (Arnstein et al., 1999, p. 488). As well, changes in Pain Self-Efficacy influence pain intensity (Jensen & Harder, 2004). Subsequent research supports this mediation effect and extends it to include clinical and community samples in both urban and rural settings (Arnstein, Wells-Federman & Caudill, 2001). As well, Denison, Åsenlöf and Lindberg (2004) found Pain Self-Efficacy explains the variance in disability better than Catastrophizing, fear of pain, pain intensity or pain duration (see also Asghari & Nicholas, 2001; Buckelew et al., 1994). These results suggest Pain Self-Efficacy influences fear and anxiety beliefs.

Perceived Control Beliefs

Perceived Control beliefs influence the degree of Acceptance. Acceptance "is to relinquish attempts to control aversive thoughts and feelings and to adopt a non-reactive openness (Hayes & Wilson, 1994)" (Viane et al., 2004, p. 286). On the other hand, rigidly attempting to control pain leads to less pain tolerance versus accepting the presence of pain as a part of life (McCracken et al., 2004, p. 4). Acceptance and rigid control are "each for use when it works most effectively in the service of a better

life" (McCracken et al., 2004, p. 6). Based on this conceptualization the control agenda refers to an attempt to control reactive aversive thoughts and feelings. However, in the literature these perceived personal control beliefs are presented as a contextually dependent belief that influences the relative adaptiveness or maladaptiveness of peoples' reactions:

> Perceived control over *effects of pain* on life functioning is more strongly associated with functioning than perceived control over *pain itself* (Tan et al., 2003). Other data show that patients who report greater struggling to control pain also report greater pain, distress, and disability (McCracken et al., under review). (McCracken et al., 2004, p. 5)

Locus of Control. There are two independent dimensions of health locus of control (LOC) beliefs, internal and external (Wu et al., 2004). External health LOC beliefs, but not internal health LOC beliefs, have been found to be a predictor of negative mental health status (Wu et al., 2004). These two dimensions are also present in the pain-related LOC literature (Rotter, 1990; Wallston & Wallston, 1978; Wallston, Wallston, Smith & Dobbins, 1987). As aforementioned, external control beliefs align with External Pain Responsibility, the belief pain is controlled by chance or powerful others, and are described as helplessness beliefs (Asghari & Nicholas, 2001; Main & Waddell, 1991; Philips, 1989). On the other hand, pain-related internal LOC align with increased personal control, the beliefs one has control over behaviour and pain (Bullington et al., 2003).

The pain-related LOC literature demonstrates a similar pattern of results as the health LOC literature:

> Attempts at control in uncontrollable situations can have a significantly negative impact on adjustment to chronic health problems (Christensen et al., 1995; Eitel et al., 1995). In terms of pain it has been demonstrated that when healthy subjects try not to experience experimentally induced pain, they have delayed recovery after pain exposure (Cioffi & Holloway, 1993).... The relationship between acceptance and control of chronic pain may be complex. The target for control attempts may be crucial. Tan et al. (2002) showed that perceived control over effects of pain or life in general are more important correlates of functioning than perceptions of control over pain itself. Adopting a more accepting stance concerning pain may lead chronic pain sufferers to a higher sense of general self-control (Jacob et al., 1993). (McCracken & Eccleston, 2003, p. 201)

However, contrary to the health LOC literature, in addition to the influence of pain-related External LOC beliefs, changes in internal LOC are also related to positive treatment outcomes (Coughlin, Bandura, Fleischer & Guck, 2000; Jensen, Turner, Romano, 2001; Miller, 2000). In fact, people with chronic pain demonstrate greater internal LOC beliefs when compared to other demographics of pain patients (i.e., cancer-related pain) (Arraras, Wright, Jusue, Tejedor & Calvo, 2002). Further clarifying the impact of these beliefs, increased internal control beliefs are not associated with greater pain control or acceptance of pain but rather increased perceived control over the ***negative*** effects of pain on one's life. These increased internal personal control beliefs are associated with decreases in Catastrophizing and perceived disability (Jensen et al., 2001).

Goal Pursuit Beliefs

Lethem et al.'s (1983) Fear Avoidance Model of Exaggerated Pain Perception presents two responses to fear of pain: confrontation or avoidance. Slade, Troup, Lethem and Bentley (1983) expand this notion to explain confrontation and avoidance as two extremes along a continuum of responses to fear of pain. Confrontation as "an adaptive response, in which the individual views pain as a nuisance and has strong motivation to return to normal levels of activity" (Fritz et al., 2001, p. 8) and avoidance as "a maladaptive response causing the patient to avoid certain activities that are anticipated to cause an increase in pain and suffering (Crombez et al., 1998)" (Fritz et al., 2001, p. 8).

McCracken and Eccleston (2006) note "the pain management field may benefit from evolving toward incorporating a less control-oriented and more accommodating view of aversive private experiences in some circumstances" (p. 23). It is proposed this alternative view should incorporate goal-pursuit beliefs, beliefs about the solvability of the chronic pain problem. An important addendum to this conceptualization are findings that problem-solving is not impaired in a chronic pain sample. In fact, De Vlieger et al. (2006) found both the amount of Worry and problem-solving competence is normal and does not differ between people with chronic pain and a non-clinical sample: Even after "12 years of failing to solve the problem of chronic pain, general problem solving confidence and attempts to solve [other] problems appear to be unaffected" (p. 143). However, this conceptualization suggests "the success or failure of attempts at problem-solving will depend upon the

interaction between one's normal approach to solving problems, their ability to solve problems, and a perception of the problem as soluble" (Aldrich et al., 2000, p. 460).

Pairing this conclusion with the literature provides clarity on the effect of goal-pursuit beliefs in the context of chronic pain. Jacob, Kerns, Rosenberg and Haythorthwaite (1993) found people who accommodate to pain demonstrate "fewer depressive symptoms, and fewer pain behaviors [*sic*] reflecting affective distress" (p. 519). Further, Schmitz et al. (1996) found "the ability to flexibly adjust personal goals attenuated the negative impact of the pain experience (pain intensity, pain-related disability) on psychological well-being (depression)" (p. 41). As Viane et al. (2004) note:

> It is...reasonable to assume that acceptance of chronic pain, also implies that pain will sometimes interfere. To preserve a positive life despite the uncontrollable effects of pain is probably best achieved by flexible goal adjustment of personal goals to the current limitations (Brandtstädter & Rothermund, 2002). (p. 287)

Assimilative and Accommodative Beliefs. Goal pursuit is defined as either Assimilative or Accommodative (Jacob et al., 1993; Schmitz et al., 1996). Assimilative and Accommodative coping are reflections of "core beliefs about oneself, aspirations and life-goals" (Viane et al., 2004, p. 287). Assimilative beliefs reflect the belief the situation needs to be altered to maintain the integrity of the goal. Accommodative beliefs reflect the belief goals need to be changed to meet the limitations of the situation. The presence of Assimilative and Accommodative beliefs relates to both situational and personal parameters (Brandtstädter & Renner, 1992): If

a person perceives themselves as capable of changing the environment or views the environment as changeable then Assimilative beliefs will be activated, whereas if the person perceives themselves as unable to change the situation or views the situation as unchangeable then Accommodative beliefs are activated (Brandtstädter, Wentura & Greve, 1993). This suggests LOC and pain beliefs both influences enlistment of the goal-pursuit beliefs.

Summary

The relevant pain beliefs identified are operationalized as Organic Causation beliefs and Responsibility beliefs (i.e., Psychological Influence and External Pain Responsibility beliefs), Self-Blame beliefs, Mystery beliefs, Pain Permanence beliefs and Pain Constancy beliefs. Beliefs about perceived consequences and activity-related beliefs identified as pain beliefs were redefined and operationalized as Physical Activity-Related Fear-Avoidance beliefs and Work-Related Fear-Avoidance beliefs. The 'maladaptive' beliefs identified are Worry and Catastrophizing. The 'adaptive' beliefs identified are Acceptance and Pain Self-Efficacy. The other two relevant belief constructs identified are Internal Pain Control and two Goal-Pursuit beliefs, specifically Accommodative and Assimilative beliefs.

The constructs presented are not solely empirically based but rather they are based on subjective analysis in light of the findings in the literature. It is proposed each belief identified is unique and contributes uniquely to peoples' beliefs about chronic pain and recovery. Inclusion of a variety of beliefs in this study facilitates determination of the relative independence of each belief and allows for a comprehensive thorough examination of the respective impact of each belief within

the context of the other identified beliefs. The next section focuses on creating a model utilizing this empirically informed, subjectively delineated, consensual nomenclature.

Model Development

The sheer number and breadth of models proposed in relation to the cognitive experience of chronic pain is overwhelming and make model building a daunting task. As well, the majority of the studies referenced to create the nomenclature do not facilitate comprehensive model development because they utilize correlational and multiple regression analysis methodologies which provide limited information on the directionality of chains of influence. Further, operationalization of many of the identified beliefs is inconsistent; Researchers rarely compare similar constructs and include only a fraction of the identified beliefs constructs across studies or alternative programs of study. This makes conclusions regarding the relevancy of the constructs and statements regarding directionality of relationships/associations (e.g., beliefs a priori or post hoc to other beliefs) difficult to conclusively identify.

To translate the proposed nomenclature into a model of peoples' beliefs about chronic pain and recovery the chronic pain belief model literature is interpreted and merged according to the operational definitions presented. However, due to the exigent nature of the literature and contradictory findings three testable models are proposed. The primary model is the Theoretical Model. It is most consistent with the literature. Second a Parsimonious Theoretical Model reduces the Theoretical Model to its simplest, linear form. Third a Less Restricted Theoretical Model explores full mediation through the inclusion of more direct relationships than the Theoretical model. This section closes with a visual representation of the models tested.

Integration of Nomenclature with Current Models

The foundational work of Lethem et al. (1983) brought pain beliefs to the forefront with the Fear-Avoidance Model of Exaggerated Pain Perception. The authors hypothesized peoples' beliefs about pain shape their reaction to the presence of pain. The focus was the actual organicity of the pain as the critical determinant in their reaction. This model is the one of the most popular models and has demonstrated validity throughout the last two decades of research. However, Philips (1987) indicated perception, the cognitive component of the experience, is likely the integral ingredient to the expression of chronic pain rather than actual objective organicity. Therefore, the current focus is to expand upon Lethem et al.'s (1983) foundational study and translate it to focus on the cognitive component. Meaning rather than focusing on actual organicity the focus is on Organic beliefs. All seven pain beliefs identified are included in this formulation (Organic Causation, Psychological Influence, Pain as a Mystery, Pain Permanence, Pain Constancy, Self-Blame and External Pain Responsibility). However, in line with Lethem et al.'s Fear-Avoidance Model all are independent variables and proposed to act as the canvas upon which the other identified beliefs operate (Boersma & Linton, 2006; Linton et al., 2000).

Vlaeyen, Kole-Snijders, Rotteveel,et al.'s (1995) revision of the Fear/Avoidance Model of Exaggerated Pain Perception added the influence of Catastrophizing on the development of fear. But, in a subsequent analysis, Vlaeyen, Kole-Snijders, Boern, et al., (1995) reformulated the influence of Catastrophizing to define it as a mediator between pain beliefs and fear/avoidance beliefs rather than as a separate influence. In their reformulated model they specifically focus on the

development of fear of movement/(re)injury and behavioural avoidance. The current study again redefines the physical focus to highlight the cognitive core of physical activity- and work-related beliefs. Klenerman et al. (1995) support this refocus with their findings that pain-related fear is a precursor, and not a consequence, of physical disability (see also Van den Hout et al., 2001). Therefore, in line with the Fear Avoidance Model, the resulting framework for current model development specifies all the pain beliefs and Catastrophizing influence Physical Activity-Related and Work-Related Fear-Avoidance beliefs.

Lackner and Quigley (2005) expand the role of Catastrophizing as a full mediator for the effect of Worry on suffering. They hypothesized "worry influences catastrophizing by inflating negatively skewed beliefs that form the basis for catastrophizing" (p. 952). Suffering in their study was not operationalized as fear-avoidance beliefs but to facilitate current model development it is assumed suffering includes high fear-avoidance beliefs.

Lackner and Quigley (2005) also propose perception of threat and problem-solving strategy influence Worry. This conceptualization maintains the importance of Assimilation and Accommodation beliefs, in accordance with Schmitz et al.'s (1996) Dual-Process Model of Coping. The developing model also maintains Schmitz et al., (1996) contention of full mediation of control beliefs between the pain beliefs and the goal-pursuit beliefs. In other words, to utilize the consensual nomenclature, all of the pain beliefs influence Internal Pain Control beliefs which influence both Goal-Pursuit beliefs (Assimilative and Accommodative beliefs) which in turn both influence Worry. Worry then influences Catastrophizing which then influences both Physical Activity-

Related and Work-Related Fear-Avoidance beliefs. Although not explicitly in reference to this formulation, De Vlieger et al. (2006) note:

> Emerging is a picture of the person with chronic pain verbally ruminating on the potential consequences of multiple threats, catastrophizing about what further adversity pain might lead to, and actively considering various solutions to the problem of pain. (p. 142)

However, a few concerns are of importance in integrating these findings. Specifically, despite the findings presented in the creation of the consensual nomenclature discounting the pathology of Worry (Aldrich et al., 2000; De Vlieger et al., 2006; Eccleston et al., 2001), Lackner and Quigley (2005) operationalized Worry as "a generalized predisposition" (p. 954) and utilized a questionnaire measuring "the extent to which worry is pervasive, uncontrollable, and excessive" (p. 947). It was this operationalization that led to interpretation that "the process of worry lead[s]...to more specific catastrophizing behaviour [which] may intensify more severe pain suffering" (p. 954). As well, it was based on this operationalization Lackner and Quigley concluded the perception of threat may be sensitized for people with chronic pain.

As well, the catastrophizing measure (the Pain Catastrophizing Scale [PCS]) Lackner and Quigley (2005) utilized measures Catastrophizing beliefs which have been differentiated from catastrophizing behaviour (Chibnall & Tait, 2005; D'Eon, Harris & Ellis, 2004; Osman et al., 2000; Osman et al., 1997; Sullivan, 2004; Sullivan et al., 1995; Sullivan et al., 2005; Sullivan et al., 2001; Van Damme et al., 2002). Although this usage is consistent with the purpose of the current study Lackner and Quigley's extension of their conclusions to catastrophizing behaviour may not

completely reflect the operationalization utilized and, therefore, may be slightly premature.

However, despite the operationalization concerns for the variables in Lackner and Quigley's (2005) study, there is external additional support for their conclusion that Catastrophizing fully mediates the relationship between Worry and both Fear-Avoidance beliefs. In support of the generalizability of Lackner and Quigley's findings, Swinkels-Meewisse et al. (2006) found "pain-related fear (resulting in avoidance behaviour [*sic*] is more proxy to actual performance of a moderately feared activity than is pain catastrophizing" (p. 41). As well, Van den Hout et al. (2001) found "pain intensity, pain catastrophizing, pain-related fear, daily stress, and problem-solving are individually related to functional disability, but that this is most convincingly the case for pain intensity and pain catastrophizing" (p. 144). Finally, utilizing Structural Equation Modeling (SEM), Cook et al. (2006) found Catastrophizing directly influenced fear of (re) injury beliefs. But, although Lackner and Quigley's findings are intuitively consistent with current knowledge, the fully mediated effect of Catastrophizing on the relationship between Worry and Fear-Avoidance beliefs requires further testing.

Therefore, the current developing model suggests peoples' beliefs regarding the threat of pain (pain beliefs), solutions (Accommodative or Assimilative) and their perception of problem-solving ability (Internal Pain Control) are determinants of whether the person will engage in Worry and Catastrophizing (Aldrich et al., 2000; DeVlieger et al., 2006; Lackner & Quigley, 2005). In accordance with Schmitz et al.'s (1996) findings that Accommodative beliefs attenuate the negative influence of

Assimilative beliefs, the model depicts these two beliefs as interacting to either attenuate or intensify the subsequent 'adaptive' or 'maladaptive' beliefs.

These hypothesized relationships can result in the negative effects as outlined but, alternatively, peoples' pain beliefs, beliefs about solutions and perception of their problem-solving skill can also lead to Acceptance beliefs thereby increasing their Pain Self-Efficacy beliefs (McCracken, 1998) and decreasing their Fear-Avoidance beliefs. No studies were located explicitly indicating increased Pain Self-Efficacy would decrease the Fear-Avoidance beliefs but this position is supported with Arnstein's (2000) findings that Pain Self-Efficacy mediates the effects of pain intensity on disability (see also Arnstein et al., 1999; Bandura et al., 1987). Specifically:

> Pain and re-injury expectancies appear to be a product of one's confidence to perform functional tasks. Furthermore, these data suggest that "pain catastrophizing," which Turk and Rudy (1992) characterize as of central importance in exacerbating pain problems, also may be a by-product of patients' confidence in their performance capabilities insofar as anticipated outcome of future harm are outcome expectancies (Bandura, 1978). (Lackner, Carosella & Feuerstein, 1996, p. 216)

Also, no study was located focusing on the differential effect of pain beliefs on control beliefs or their respective influence on the goal-pursuit beliefs. Therefore, in accordance with Schmitz et al. (1996), it is proposed the influences of the various pain beliefs on the goal-pursuit beliefs are fully mediated through control beliefs. This acknowledges the literature indicating it is the perception of the problem, perception of your ability to do something and the perception of an available solution that leads to

subsequent reactions (Aldrich et al., 2000; Arnstein, 2000; Arnstein et al., 1999; Bandura et al., 1987; De Vlieger et al., 2006; Lackner & Quigley, 2005; Lethem et al., 1983; Philips, 1987; McCracken, 1998; Schmitz et al., 1996; Van den Hout et al., 2001; Vlaeyen, Kole-Snijders, Boern, et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995).

In addition to the lack of research support for the pain belief relationships, there is some contention regarding the sequence of control beliefs and goal-pursuit beliefs (Schmitz et al., 1996; Haythornthwaite et al., 1998). Further, there is additional contention regarding the interacting influence between Accommodative beliefs and Assimilative beliefs on subsequent 'adaptive' or 'maladaptive' beliefs (Haythornthwaite et al., 1998; Schmitz et al., 1996). In regards to the influence of the goal-pursuit beliefs, Schmitz et al. (1996) indicated Assimilative beliefs lead to increased pain intensity, pain-related disability and depression; compared to Accommodative beliefs which attenuated these negative effects. But, findings suggest "coping flexibility...predicted perceptions of control" regardless of pain severity (Haythornthwaite et al., 1998, p. 33), changes in pain locus of control influenced pain severity, both magnitude and frequency (Toomey et al., 1991) and greater pain complaints were significantly related to physical impairment (Palyo & Beck, 2005). These results suggest goal-pursuit beliefs are a priori to 'adaptive' and 'maladaptive' beliefs.

Summary

These studies together are integrated into a model of peoples' beliefs about chronic pain and recovery. However, due to tenuous or contradictory findings two

other models are also proposed to add testing power to the analysis. The three models present an attempt to coalesce the literature into a comprehensive model. Model 1 is the Theoretical Model (see Figure 2), Model 2 is the Parsimonious Theoretical Model (see Figure 3) and Model 3 is the Less Restricted Theoretical Model (see Figure 4). The intricacies differentiating the models consist of changing the number directness or mediational characteristics of pathways connecting the variables.

Figure 2: The Theoretical Model.

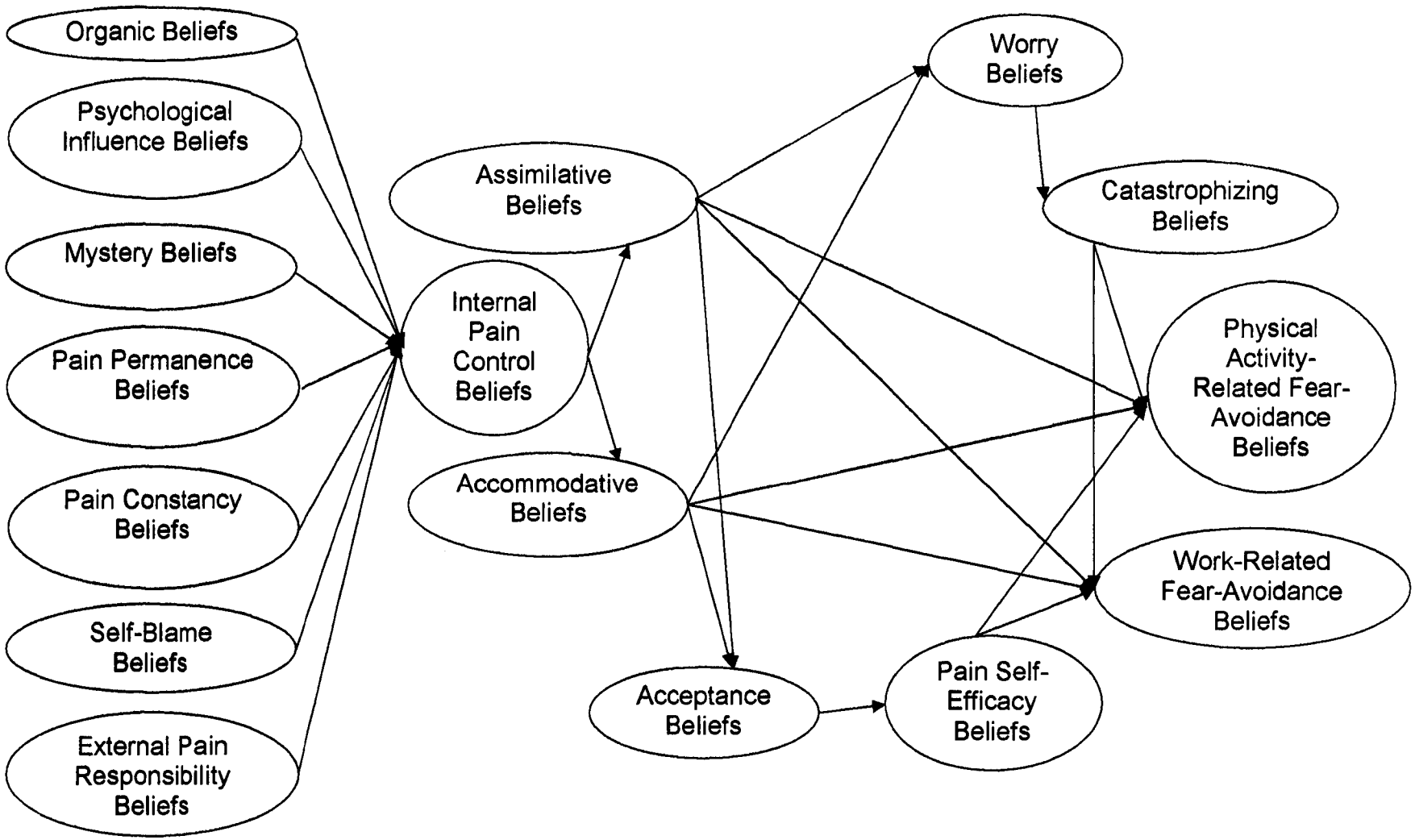


Figure 3. The Parsimonious Theoretical Model.

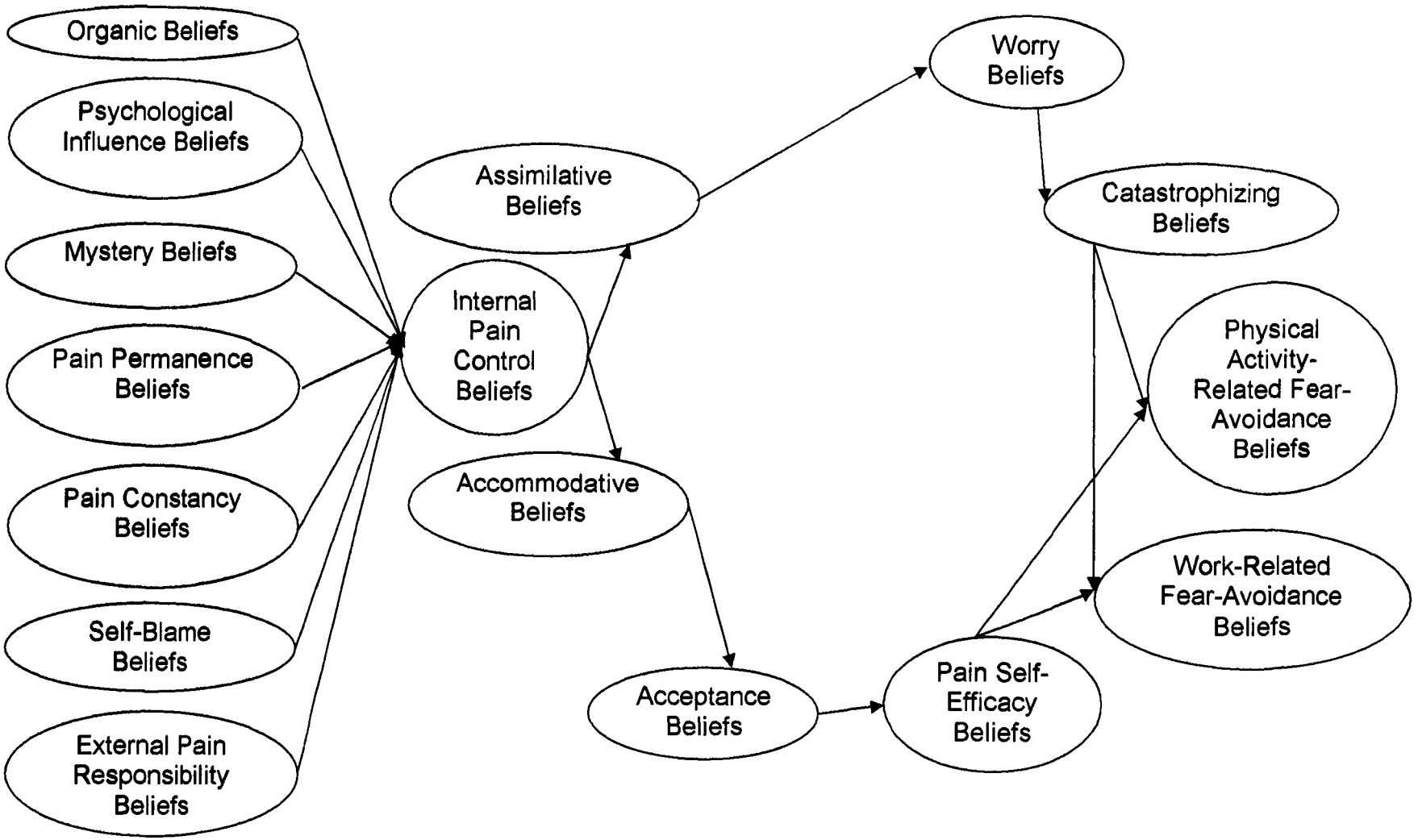
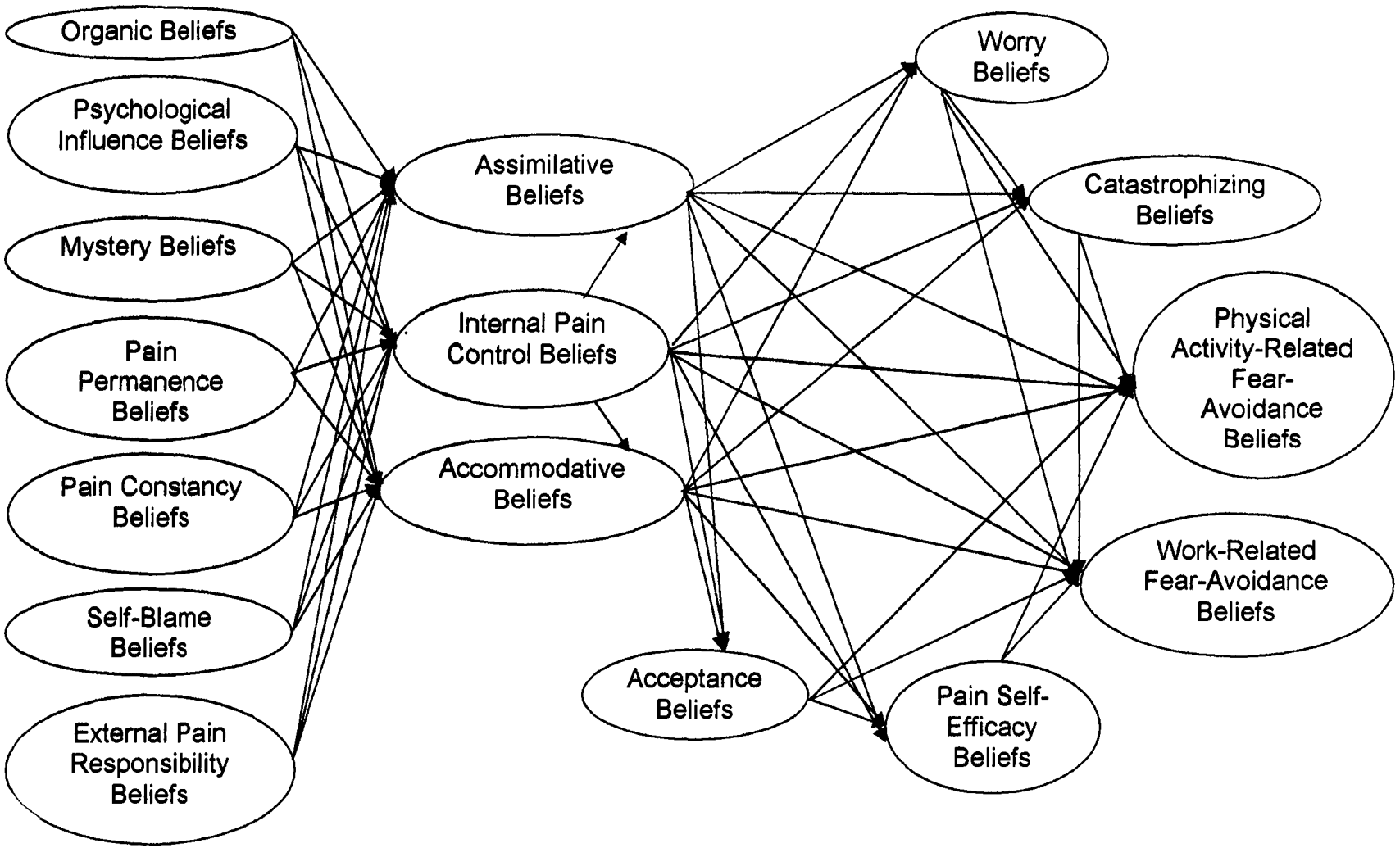


Figure 4. The Less Restricted Theoretical Model.



All three models propose chronic pain is a unique threat and propose peoples' beliefs about chronic pain and recovery may have negative or positive repercussions. Specifically, all three models suggest beliefs that are helpful to prevent further injury and allow for healing in an acute pain experience (i.e., pain means harm, avoid all pain, cure the problem, etc.) may become problematic in the situation of chronic pain because prolonged vigilance (i.e., Worry) to pain can trigger further 'maladaptive' beliefs (i.e., Catastrophizing) and increased Fear-Avoidance beliefs. On the other hand, all three models suggest viewing pain as present but not necessarily harmful to the body and/or possessing an Accommodative view entailing Acceptance of pain and personal pain-related limitations may be helpful to increase Pain Self-Efficacy and reduce Fear-Avoidance beliefs. However, the three models proposed differ in altering the directness and mediational relationships between the identified variables.

The Theoretical Model is most consistent with the literature and is closest to the Fear-Avoidance Model of Exaggerated Pain Perception. The Parsimonious Theoretical Model takes out the contradictory findings and theorizes a linear relationship between the identified beliefs while maintaining the order proposed. The Less Restricted Theoretical Model recognizes the lack of literature exploring the relationships between the identified variables and provides a test of the assumptions of the Theoretical Model. Specifically, to address oversights in the literature the Less Restricted Theoretical Model includes additional direct pathways from the pain beliefs to the goal-pursuit beliefs and direct paths from Internal Pain Control beliefs to the subsequent 'adaptive' and 'maladaptive' beliefs. As well, direct pathways from the goal-pursuit beliefs to the subsequent 'adaptive' and 'maladaptive' beliefs were added.

Direct paths from Worry to the Fear-Avoidance beliefs were added to address the methodological flaws associated with Lackner and Quigley's (2005) study proposing full mediation of Worry between Catastrophizing and the Fear-Avoidance beliefs. Finally, paths were added from Acceptance to both the Fear-Avoidance beliefs in recognition of the absence of literature regarding the applicability of the 'adaptive' beliefs to the Fear-Avoidance beliefs.

Conclusion

The current research represented an exigent challenge in creation of the consensual nomenclature and the integration of this nomenclature with existing models to develop a comprehensive model of peoples' beliefs about chronic pain and recovery. Every effort was made to acknowledge and integrate contradictory findings to clarify and illuminate the underlying beliefs structure. Testing these models has a triple purpose: (1) if the Theoretical Model (or one of the other models) are supported it can provide a framework for further analysis of the interaction of other aspects of the experience of chronic pain, (2) if the Theoretical Model (or the other models) are not supported it can be compared to the other existing models to act as a catalyst for future research, (3) regardless of whether the models are supported or not it will provide evidence regarding the suitability of this synthesis of beliefs into a consensual nomenclature, which future research can expand upon to further our knowledge about peoples' beliefs about chronic pain and recovery.

Chapter 3: Method

The three models developed are tested utilizing Structural Equation Modeling (SEM). SEM is a path analytic statistical approach used to determine how well a sample variance/covariance structure fits a theoretically derived population variance/covariance structure depicted in a path model. In this chapter an overview of SEM methodology, potential problems, potential solutions and points of contention for SEM analysis are reviewed with the aim of providing a clear empirically validated rationale for the procedure utilized.

Structural Equation Modeling

Streiner (2005) indicates SEM cannot establish causality or prove a given model is correct and "no amount of statistical legerdemain can pull cause and effect out of a cross-sectional or cohort study" (p. 116). But SEM can determine how consistent the observed data are with the proposed chain of influence presented in the path model and has been heralded as "an important tool for testing theories with both experimental and nonexperimental data" (Fan & Wang, 1998, p. 702).

Levels of Variables

SEM is similar to regression and factor analysis in that three levels of variables are utilized: the latent level, the manifest or measurement level, and error terms. If SEM were not utilized then the latent variables would be examined through regression and the manifest variables through factor analysis. Both multiple regression and factor analysis include error terms in their analysis but assume the error is zero. The benefit of SEM is that it allows latent and manifest variables to be examined simultaneously

and allows for explicit acknowledgement of the presence of error through the inclusion of error terms in the model.

Latent Variables. The latent variables are the unobservable theoretical constructs labelled either as exogenous or endogenous. Exogenous are the independent variables and endogenous are the dependent variables. No attempt is made to explain the chain of influence affecting the presentation or variation of the exogenous variables. Rather, the causal influence is reflected through saturated correlations between all the exogenous variables. On the other hand, the chain of influence for endogenous variables is explicitly theorized in the model.

Manifest Variables. The second level of variables is the manifest level or measurement level which consists of the measured indicators for the latent variables. Because the model is based on the exogenous variables accounting for the covariance of the endogenous variables it is critical the endogenous variables vary and, therefore, endogenous variables need to be measured on a continuous rather than dichotomous metric.

Error Terms. The ability to model the error associated with each indicator is a desirable feature of SEM, specifically for disciplines frequently utilizing intangible constructs that encompass a broad array of attributes (i.e., beliefs); thereby differentiating SEM from factor analysis and regression where error terms are implied but assumed to be independent and therefore are not explicitly included in their respective analyses. However, no error terms are necessary for the exogenous latent variables because there is no extraneous variation that needs to be accounted for. The chain of influence for the presence and variation of the exogenous variables is not

outlined; to account for their presence and variation the exogenous variables are allowed to freely correlate with each other.

On the other hand, for the endogenous latent level the error terms are assumed to have a mean of zero but the error terms are included and explicitly modeled. This explicit modeling of error allows for recognition that other modalities of influence may be important (i.e., sampling bias, response bias, data entry errors, etc.): Reality rarely conforms to just one modality of influence. Although we may test if A causes B there is always the possibility that both A and C cause B even when C is not measured or explicitly included in the analysis. The inclusion of the error terms allows for this recognition. The endogenous latent variable error terms indicate the model proposed may not be the sole chain of influence for the endogenous variables and there may be other unmodeled factors influencing the presence and variation of the endogenous variables. With this acknowledgment SEM allows for explicit recognition of possible misspecifications and omission of important variables (an important issue that will be addressed later in the Model Identification section) without compromising the validity and accuracy of the model proposed and tested.

For the manifest level, the researcher specifies the error associated with each indicator depending on how much the parent construct differs from the indicator. This ability to specify measurement error facilitates use of multiple measures for a single construct and removes the factor analytic restriction of no correlation between indicators for different constructs. A standard practice in SEM is to use at least two measures for each latent variable to provide an accurate estimation of measurement error (Cook et al., 2006). But if only one indicator is utilized the error variance must

be specified based on external data, such as reliability studies (Schumacker & Lomax, 2004). Obtaining the error variance from reliability studies with other samples ensures the model tested is generalizable to the population of interest and not a result of or reflection of idiosyncratic sample characteristics.

Model Specification

Specification refers to how the model is depicted; Utilizing these three levels of variables the model is specified. The visual diagram of the latent and manifest variables and their respective error terms is generally called the structural model. It includes both the path model of the latent constructs and the measurement model of the indicators (McDonald & Ho, 2002). The structural model implies a series of matrix equations that place constraints (researcher defined restrictions proposing no chain of influence) on the model implied covariance matrix to allow for testing against the sample derived covariance matrix (Hittner & Carpenter, 1994).

Structural Model. In the path model, the latent constructs are depicted with ellipses and connected with arrows depicting the hypothesized flow of dependency (McDonald & Ho, 2002). The exogenous variables appear at the beginning of a model and only have single-headed arrows pointing away from them other than the error term arrows and curved double-headed arrows designating the saturated correlation between the exogenous variables. The endogenous variables have single-headed arrows pointing toward them, indicating the chain of influence is theorized to follow the direction of the arrow. If the latent model presented has arrows flowing in one direction (indicating a linear chain of influence) then the model is called recursive; if

the model contains arrows flowing in both directions (i.e., feedback loops) then it is called non-recursive.

In the measurement model the indicators are depicted with rectangles with the exogenous indicators specified with X and the endogenous indicators with Y. The latent constructs are proposed to cause the variation in the observed indicators, which is depicted with a single-headed arrow flowing from the latent variable to the indicator.

Matrix Equations. The latent and manifest variables along with their respective error terms correspond to the three basic matrix equations, which imply four matrices that summarize the hypothesized model. These equations and accompanying hypothetical model-implied covariance matrices define the model in a manner that enables testing against the variance/covariance matrix of the sample.

The first matrix equation is the Beta-Gamma [$\beta\Gamma$] equation. $\beta\Gamma$ encapsulates all the postulated effects among the latent concepts, with the exogenous variables dubbed Xi (ξ) and the endogenous variables dubbed Eta (η). The $\beta\Gamma$ equation also includes the error variances associated with the endogenous variables, Zeta (ζ). The second equation, Lamda-Y (Λ_y), links the endogenous concepts (η) with its indicators (y) and their respective error variances, Epsilon (ε). Finally the third equation, Lamda-X (Λ_x), links the exogenous indicators (ξ) with its indicators (x) and their respective error variances, Delta (δ).

These three equations imply four theoretically implied covariance matrices (Hayduk, 1987). The $\beta\Gamma$ equation implies a specific covariance structure among the exogenous concepts, called the Phi (Φ) matrix and a specific covariance matrix among

the errors in the latent model called the Psy (Ψ) matrix. The Λ_y matrix implies a covariance matrix among the error terms for the endogenous indicators called the Theta-Epsilon (Θ_ε) matrix. The Λ_x equation implies a covariance matrix among the error terms for the exogenous concepts, which is called the Theta-Delta (Θ_δ) matrix.

Model Identification

The matrix equations compute to create a model-implied covariance matrix that is compared to the covariance matrix of observed sample data. Specification or model identification is the process by which these matrix equations are outlined:

> How many things we have to estimate (such as path coefficients and correlations) in relation to how much information we can derive from the data information in terms of the observed variances of the variables and the covariances among them. (Streiner, 2005, p. 120)

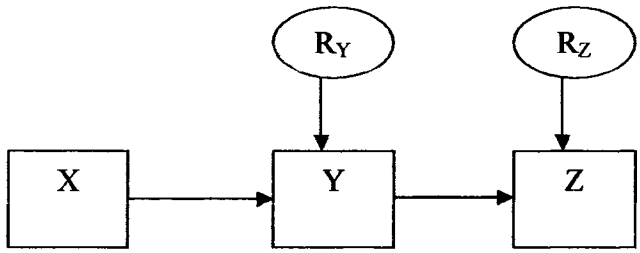
Models can be just identified, underidentified or overidentified. Just identified models have only one solution that is possible from the data available. Underidentified models do not provide enough information to derive a firm solution because there are a multitude of viable possibilities based on the data available. Finally, overidentified models provide more than enough information from which possible solutions can be tested to determine which solution fits the data the best. The most common reason for poor fit is misspecification, which means inclusion of extraneous variables, omission of crucial variables and relations between variables misrepresented (Streiner, 2005). Misspecification is a result of identification problems.

Path Model Identification Problems. For some models there is not just one solution and in fact many equivalent models may explain the covariance in the

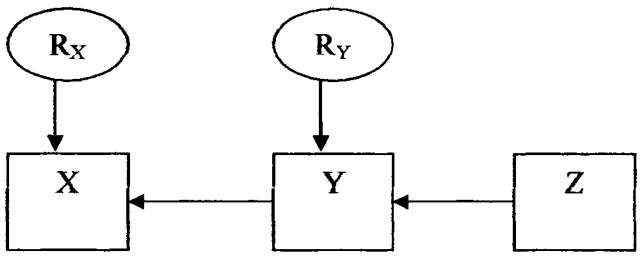
observed data as well as the proposed model (MacCallum & Austin, 2000; Martens, 2005; McDonald & Ho, 2002). To clarify, Figure 5 depicts three equivalent structural models that propose very different causal structures yet "yield the identical covariance matrices, discrepancy function values, and measures of overall fit" (Tomarken & Waller, 2003, p. 580). To facilitate comprehension of this concept traditional SEM diagrammatic procedure (described earlier) will not be utilized in Figure 5. As well, there may be non-equivalent models that may explain the covariance in the observed better than the proposed model or its equivalent forms (Tomarken & Waller, 2003). This is one of the major problems in SEM analysis: There are innumerable equivalent and non-equivalent models that can be developed.

Figure 5. Three equivalent causal models: X, Y, and Z are observed variables and R_x, R_y, and R_z denote error terms representing unmeasured influences on X, Y, and Z.

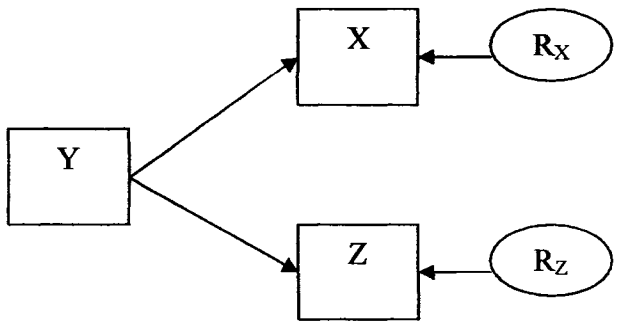
Model 1A.



Model 1B.



Model 1C.



From "Potential Problems with 'Well-Fitting' Models," by A. J. Tomarken and N. G. Waller, 2003, *Journal of Abnormal Psychology, 112*(4), 580. Copyright 2003 by the American Psychological Association.

Path Model Identification Solutions. Testing all possible models is not an efficient remedy for the problem of identification due to the sheer volume of models that could be presented and the reality that "not all equivalent [and non-equivalent] models are equally plausible" (Tomarken & Waller, 2003, p. 582). As well, development of equivalent and non-equivalent models through data driven exploration may result in a model that more accurately reflects the idiosyncrasies of the sample and may have limited generalizability to the population of interest. As Streiner (2005) noted, "path analysis is a model-testing approach, not a model-building one" (p. 121). Therefore, it is necessary to develop the model prior to testing it. This basis allows researchers to create overidentified models that imply restrictions based on their theory and the literature and limit the amount of alternative equivalent and non-equivalent models (Tomarken & Waller, 2003). The aim is to create a theoretically based model that provides more than sufficient information to test the model and determine if it is better than other models in explaining the covariance structure of the variables.

As well, it is recommended that researchers test nested models. A nested model is a model with fewer proposed chains of influence. A nested model allows for a direct comparison between models with the same data set and allows for a determination of which model fits the data the best. The premise driving this procedure is that the more parsimonious the model the better, so all measures of goodness of fit being equal the more parsimonious model should be preferred. However, this assumption is debatable and choice of the 'better' model should be based on theoretical grounds rather than parsimony alone. As a result, Martens (2005)

encourages nested models be developed a priori to reduce confirmation bias and provide viable alternatives should the observed sample's covariance matrix fail to fit the model's hypothesized population covariance matrix.

To provide further methodological rigour Anderson and Gerbing (1988) recommended testing five nested models: a saturated model where every concept is exogenous and allowed to freely correlate (the measurement model), a null model where no relationship is hypothesized between the variables, the theoretical model, "the next most likely" constrained model where fewer relationships are modelled than in the theoretical model and "the next most likely" less constrained model where more relationships are modelled than in the theoretical model (p. 418). Analysis of these five models adds rigour, reduces confirmation bias and introduces theoretical constraints on parsimony-driven acceptance of models. However, this approach is beneficial only when the models fit the data; if the models do not fit then comparison of nested ill-fitting models may not reveal a more appropriate model (McMurtry, 2004).

If nested models are not utilized then Tomarken and Waller (2003) encourage researchers to, at a minimum, acknowledge the possibility of alternative models in their discussion and conduct further research with increasing experimental rigour so as to facilitate discrimination between alternative models.

Measurement Model Problems. Another identification problem is "interpretational confounding" (Anderson & Gerbing, 1988, p. 418). This refers to the separation of the measurement model from the path model: SEM can test both models concurrently but if the manifest variable does not measure the assigned latent variable

then model identification can be suspect. Anderson and Gerbing (1988) note "interpretational confounding is reflected by marked changes in the estimates of the pattern coefficient when alternate structural estimates are estimated" (p. 418). Therefore, they recommend and stress the importance of confirmation for the soundness of the measurement model prior to examining the structural model (Anderson & Gerbing, 1988; Tomarken & Waller, 2003).

Measurement Model Solutions. A pure measurement model analysis is similar to confirmatory factor analysis (CFA) in which the model is saturated and all of the latent constructs are allowed to freely correlate. But a pure measurement model utilizes principle axis factoring (PAF) rather than principle components analysis (PCA) (Tomarken & Waller, 2003). PAF allows the researcher to determine if the indicators load onto the latent constructs as predicted. In comparison with PCA which only allows the researcher to determine the hierarchical order of the factors according to variance explanation.

Examination of the measurement model is included through analysis of the saturated model with Anderson and Gerbing's (1988) five nested model procedure. If the saturated model does not fit the data then:

> No structural model would give acceptable fit ... [and it] would suggest a fundamental misspecification of the measurement model needs to be remedied, rather than a need to estimate additional structural models. (p. 418)

However, usage of the saturated model to test the measurement model with single indicators is not appropriate given the degrees of freedom equals zero in the saturated model and therefore the model cannot be tested against any constraints (Hayduk,

1987). It is the constraints within a model that provide testing power. With single indicators Hayduk (1987) recommends utilizing a sensitivity analysis to examine the measurement model. In a sensitivity analysis the measurement error for each manifest indicator is individually respecified and the model rerun. First the measurement error is halved and then it is doubled. In the resulting reruns of the model if the parameter estimates remain similar then it can be concluded the model is not overly sensitive or insensitive to measurement specifications and thereby the measurement model is supported.

Parameter Estimates

A population covariance matrix is estimated based on the restrictions of the theoretically derived model. Parameter estimates indicate how likely it is that we would observe this *sample* covariance matrix given this *population* covariance matrix (derived from the hypothesized model). They are calculated through Maximum Likelihood Estimate (ML), Generalized Least Squares (GL), Ordinary Least Squares (OLS), Two Stage Least Squares (TSLS), Weighted Least Squares (WLS), Unweighted Least Squares (ULS), Asymptotically distribution-free (ADF; Browne, 1984), Elliptical distribution theory (EDT), or Bootstrapping.

One of the most popular estimation procedures is ML (Yuan & Bentler, 2001). ML estimates parameters, based on multivariate normality assumptions, through computing the most likely value provided the sample represents the population from which it is drawn (Breckler, 1990; Martens, 2005; Yuan & Bentler, 2001). The second most popular is ADF (Yuan & Bentler, 2001). ADF computes estimates from the raw data rather than the covariance matrix and does not assume multivariate normality.

Yuan and Bentler (2001) recommend that ADF should be utilized when normality is significantly violated. However, ML demonstrates robust estimates even with small to moderate violation of normality, which is likely the reason for its popularity (Amemiya & Anderson, 1990; Anderson & Amemiya, 1988; Browne, 1987; Browne & Shapiro, 1988; Chou, Bentler & Satorra, 1991; Hu & Bentler, 1998; Martens, 2005; McDonald & Ho, 2002; Mooijaart & Bentler, 1991; Satorra, 1992; Satorra & Bentler, 1990; Shapiro, 1987; Yuan & Bentler, 1999). As Yuan and Bentler (2001) summarize:

> The basic conclusion of this literature is that, under some special conditions, the parameter estimates based on ML are consistent, some standard errors remain consistent, and the likelihood ratio statistic can still follow a chi-square distribution asymptotically even when the observed data are non-normally distributed. (p. 162)

As well, ML estimates are "less likely to be influenced by various sources of irrelevant effects and less likely to depart from their true-population values ... and ... [therefore] ...should be preferred indicators for model selection and evaluation" (Hu & Bentler, 1998, p. 447). But, despite this robustness "lack of attention to data quality...can lead to biased estimators and highly significant test statistics" (Yuan & Bentler, 2001, p. 162) and could inflate Type I error, meaning that even if the model fits it would be rejected (Martens, 2005). Yuan and Bentler specifically indicate outliers in the data can highly influence the determination of a model's appropriateness through influencing the calculation of parameter estimates in ML.

Thus, Yuan and Bentler (2001) recommend two approaches to reducing the negative influence of outliers. First the researcher could identify the outliers and "then

apply classical procedures after outlier removal" or use a "robust approach to downweight the influence of outliers" (p. 171). If there is evidence the assumption of a multivariate data distribution may be violated then correction to the data is necessary (Martens, 2005) through transforming variables, alternative estimation procedures (e.g. Asymptotically Distribution Free Estimator), bootstrapping and correction to the statistical estimates (Satorra Bentler χ^2). If there is no evidence the assumption of multivariate data distribution may be violated then no correction is necessary. Utilization of one of these procedures for attenuating the negative effect of outliers is critical given the parameter estimates for the covariance matrix is the crux of SEM analysis. Appropriate estimates are critical to interpretation of the subsequent findings regarding the adequacy of the model (Allison, 2003).

Model Testing

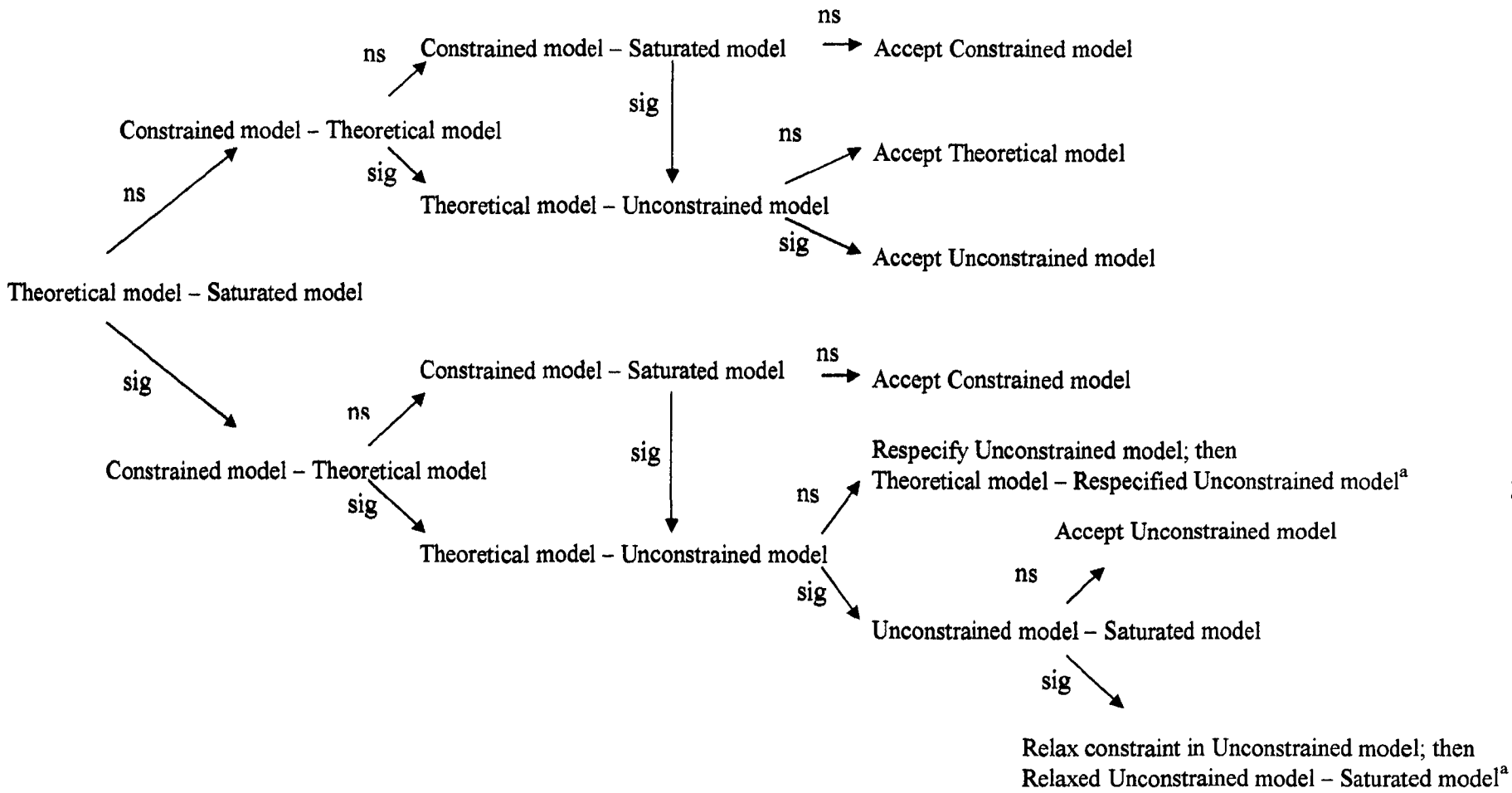
Chi-square (χ^2) statistic. The decision to reject or accept the model can be determined with the χ^2. χ^2 is one of the most frequently used because it is based on a known distribution and as such "a probability level can be calculated as in traditional hypothesis testing procedures" (McMurtry, 2004, p. 73). χ^2 provides a test of the null hypothesis that there is a perfect model fit in the population (Jaccard & Wan, 1996). As well, χ^2 is used to test nested models [recall Anderson & Gerbing's (1988) five model recommendation]. To test nested models χ^2 difference is computed, which is the difference between the χ^2 statistics for two *passing* models. The known distribution of χ^2 values is based on degrees of freedom and as such the χ^2 difference is also on this known distribution with the degrees of freedom equal to the difference in degrees of

freedom between the two models (McMurtry, 2004). Therefore, when two models adequately fit the data:

> The probability of the χ^2 difference can be derived, providing a statistical test of the difference in fit. The interpretation of a significant difference is such that given two models the one with fewer estimated parameters (higher degrees of freedom) fits better, if the difference in χ^2 between the models is not statistically significant. Conversely, a statistically significant difference in χ^2 values indicates that the model with more specified parameters (fewer degrees of freedom) is superior. In other words, a statistically significant drop in χ^2 with the addition of (a) path(s) to a model indicates that the addition was warranted and that the path(s) are important to the model. A failure to find a significant drop in the χ^2 value with the addition of (a) path(s) indicates that the added path(s) are not warranted. (p. 75-76)

Anderson and Gerbing (1988) recommend utilization of a decision tree to facilitate interpretation of the χ^2 difference with their five nested model strategy (see Figure 6). Importantly, the χ^2 difference and the decision tree are only useful and appropriate for use when comparing well-fitting models.

Figure 6. The decision-tree framework for the set of sequential chi-square difference test



sig = significant ns = non-significant [a]The modeling approach shifts from confirmatory to being increasingly exploratory.

From "Structural Equation Modeling in Practice: A Review and Recommended Two-Step Approach," by J. C. Anderson and D. W. Gerbing, 1988, *Psychological Bulletin, 103*(3), p. 420. Copyright 1988 by the American Psychological Association.

A frequent criticism of χ^2 is the effect of sample size on power (Fan & Wang, 1998; Marsh, Balla & McDonald, 1988). Power increases in conjunction with sample size so with large sample sizes the incidence of Type II error increases and with small sample sizes the incidence of Type I error increases: "As a result, model fit assessment using this narrow approach becomes stringent when sample size is large and lenient when sample size is small" (Fan & Wang, 1998, p. 703). The majority of criticism for the use of χ^2 focuses on this aspect citing the necessity of a large sample size for SEM analysis to reduce Type I error at the cost of increasing Type II error where this necessary large sample size may lead to unjust rejection of 'correct' models due to trivial differences between the sample covariance matrix and the population covariance matrix (Fan & Wang, 1998).

However, Hayduk (1987) notes "χ^2 is instructive for *N*'s ranging from about 50 to 500" depending on "the kinds of models estimated" (p. 169). Further, Anderson and Gerbing (1984) note comparability between χ^2 and the results of other goodness of fit indices, suggesting no added risk. Therefore, considering the utility of χ^2 for hypothesis testing and its known distributional properties its' use is recommended to determine viability of models. However, given the power concerns it should be paired with other goodness of fit indices especially with small or large sample sizes.

Fit Indices. As an alternative to complement χ^2 goodness of fit indices were developed (Jöreskog & Sörbom, 1996). The purpose of these fit indices is to expand upon the χ^2 dichotomous decision of acceptance or rejection of the model through quantifying the degree of fit or discrepancy between the proposed model (theoretically derived population variance/covariance matrix) and the data (sample

variance/covariance matrix obtained) (Fan & Wang, 1998; Marsh et al., 1988; Satorra & Saris, 1985; Tanaka, 1993). Tomarken and Waller (2003) suggest, in general, the smaller the discrepancy between the two covariances matrices the better the model fits. Therefore:

> For many researchers, the ideal fit index would have the following properties:
>
> 1. indicate degree of fit along a continuum bounded by values such as 0 and 1, where 0 reflects a complete lack of fit and 1 reflects perfect fit.
> 2. be independent of sample size (higher or lower values would not be obtained simply because the sample size is large or small).
> 3. have known distributional characteristics to assist interpretation and allow the construction of a confidence interval. (Gerbing & Anderson, 1992, p. 134)

However, Gerbing and Anderson (1992) conclude no goodness of fit index demonstrates all of these properties and, further, there is a distinct lack of agreement on the respective importance of all these criteria. As a result, a plethora of alternatives have been developed. For example, in LISREL—version 8 there are 23 fit indices reported in the output. These include the Minimum Fit Function Chi-Square (χ^2), Normal theory weighted least squares χ^2 (Wheaton, Muthen, Alwin & Summers, 1977), Estimated Non-centrality Parameter (NCP; Steiger, Shapiro & Browne, 1985), Minimum Fit Function Value, Population Discrepancy Function Value (F0), Root Mean Square Error of Approximation (RMSEA; Steiger & Lind, 1980), P-value for Test of Close fit when RMSEA<0.05 (P-close), Expected Cross-Validation Index (ECVI; Browne & Cudeck, 1993), χ^2 for Independence Model (the null model),

Akaike Information Criterion (AIC; Akaike, 1974, 1987), Consistent Akaike Information Criterion (CAIC; Bozdogan, 1987; Cudeck & Browne, 1983), Normed Fit Index (NFI; also called the Bentler-Bonett index [BBI]; Bentler & Bonett, 1980), Non-Normed Fit Index (NNFI; also called the Tucker-Lewis Index [TLI]; Bentler & Bonett, 1980; Tucker & Lewis, 1973), Parsimony Normed Fit Index (PNFI; James, Muliak & Brett, 1982), Comparative Fit Index (CFI; Bentler, 1990), Incremental Fit Index (IFI; also called the DELTA2; Bollen, 1989), Relative Fit Index (RFI; also called the Relative Noncentrality Index [RNI]; McDonald & Marsh, 1990), Critical N (CN; Hoelter, 1983), Root Mean Squared Residual (RMR; Bentler, 1995), Standardized Root Mean Squared Residual (SRMR; Bentler, 1995), Goodness of Fit Index (GFI; Jöreskog & Sörbom, 1981), Adjusted Goodness of Fit Index (AGFI; Bentler, 1983; Jöreskog & Sörbom, 1981; Tanaka & Huba, 1985), and the Parsimony Goodness of Fit Index (PGFI; also called the Parsimony adjusted CFI; Arbuckle & Wothke, 1999). This is not a comprehensive listing of the goodness of fit indices available in other SEM analysis programs but it does summarize the indices available within the LISREL 8 program.

There is also little consensus regarding which measures of goodness of fit are most robust. Specifically, indices are differentially robust when deviations from multivariate normality exist in the data, small/large sample sizes are utilized, and when there is a lack of parsimony (Hu & Bentler, 1999). In fact, sample size is not a problem unique to χ^2 analyses with small samples attenuating power with other goodness of fit indices as well (Tomarken & Waller, 2003). As well, "when misspecifications are quite small, even sample sizes larger than those typically used in

practice (e.g., $N = 1{,}000$) can be associated with very low power" (p. 592). Further, interpretations of these indices can be idiosyncratic and are generally based on generally accepted standards of practice (i.e., 'rules of thumb') rather than stringent empirically-based invariable standards (Browne & Cudeck, 1993).

Recommended Indices. The consensus, based on the above literature review, is the known properties of χ^2 make it one of the most popular measures of fit and it provides an excellent foundation from which to assess fit. Specifically, χ^2 allows for acceptance or rejection of the model and the other goodness of fit indices quantify this degree of fit. However, the choice of the appropriate fit indices to use in addition to χ^2 is unclear and contentious (Bentler, 1990; Bollen, 1990; Gerbing & Anderson, 1993; Martens, 2005; McDonald & Marsh, 1990).

Researchers have recommended using different combinations. Hu and Bentler (1998) recommended SRMR with TLI, RNI, CFI or RMSEA. Later they recommended SRMR with one other index such as the CFI or RMSEA (Hu & Bentler, 1999). But they suggested the NNFI and RMSEA be interpreted with caution in small sample sizes because the NNFI tends to be large in these circumstances and the RMSEA tends to over-reject substantially true models.

There is consensus to ***not*** use AIC, CN, NFI, GFI, χ^2/df ratio or AGFI because these indices are negatively affected by sample size and number of indicators per factor and do not generalize well across samples (Anderson & Gerbing, 1984; Hu & Bentler, 1998; Marsh et al., 1988). In direct comparison the NNFI, IFI, CFI, RMSEA and SRMR were found to be less sensitive to these extrinsic factors and generalized

relatively better (Hu & Bentler, 1998; Hu & Bentler, 1999; Martens, 2005; Steiger, 2000).

In an attempt to bring order to the debate, Sivo, Fan, Witta and Willse (2006) tried to determine which goodness of fit indices would simultaneously retain correct models and reject misspecified models "in a manner that is invariant across both sample size and data distribution" (p. 284). The summary of their findings suggests it is possible to set cut-off values for some goodness of fit indices to retain all correct models and to reject all misspecified models but the different fit indices require alternative cut-off values irrespective of their metric. They concluded the SRMR and RMSEA "may do the best job in aiding an applied researcher in distinguishing correct and incorrect (misspecified) models" (p. 286), which may also optimize power calculations based on these indices. They found the SRMR and RMSEA "required less stringent cut-off values as sample size increased" (p. 286). As well, they noted a sample of 150 provides an acceptable level of power to a SEM analysis.

Modification Indices

Once the fit of a model is assessed the modification indices are examined to provide information "about the direction and strength of specific relationships among the variables" (Fan & Wang, 1998, p. 702). Modification indices represent a series of univariate Lagrange multiplier (LM) tests indicating the degree of change that would occur in fit through the inclusion or omission of a path between variables (Bentler, 1995). Some researchers utilize the modification indices to improve passing models or modify a failing model so it passes goodness of fit tests (Jöreskog & Sörbom, 1993).

For both purposes there are two methods of specification searches based on modification indices alteration (Green, Thompson & Babyak, 1998). The first method is stepwise modification, where modification indices are examined to identify the parameter that if freed would most improve the fit of the model. This parameter is freed through model respecification and run a second time. The examination, identification and respecification are repeated in this stepwise fashion until the constrained parameters are all non-significant (0.5 level). An alternative approach is to identify the parameters that if freed would result in the greatest improvement in fit and free these parameters without respecifying the model at each step. However, utilization of modification indices relies heavily on the sample data. As Jöreskog (1993) note, it is a misuse of the indices and moves SEM closer to model generation rather than model testing. As such, researchers who alter their models based on these specification searches "should be concerned about committing Type I error, that is, adding parameters to their model that should not be added" (Green et al., 1998, p. 386).

Specifically, there is an increased risk for Type I error when the sample size is small, there are many parameters added in a specification search and when the original model does not fit the data well (Green et al., 1998). The implications of Type I error is it may increase standard errors for the other parameters and therefore create spuriously high fit indices (Green et al., 1998; Yuan & Bentler, 2001). Therefore, Green et al. (1998) suggest when completing model modification based on a specification search it is important to use the adjusted Bonferroni approach to control for Type I error, where the Bonferroni value is adjusted with each step of

respecification. As such Green et al. (1998) suggest a minimal criterion for the addition of a parameter be defined a priori and redefined with each specification search. However, Green et al. (1998) also warn utilization of this stringent criterion increases risk of Type II errors, where parameters that should be included in the model are not identified. As well, as an implication of Type II error, the resulting model may be biased due to the order the parameters were added given there are a myriad of equivalent models from which one could have started.

Therefore, it is important decisions to modify a model be based upon theory and not simply the modification indices (Streiner, 2005). When theoretically supported modifications are made the adjusted Bonferroni is recommended to control for Type I error (Green et al., 1998). Further, theory-driven limitations are recommended for the number of modifications allowed (Green et al., 1998). As well, any model including specification searches must be cross-validated in an independent sample to ensure generalizability to the population and increase the probability the resulting model is not an artifact of the idiosyncratic sample.

CHAPTER 4: Procedure

In this chapter, the rationale for the procedures for data collection, preparation and analysis are explained to ensure utilization aligns with the recommendations outlined in the chronic pain and SEM literature. Further, all procedures for the study were reviewed for adherence to ethical guidelines outlined in the *Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans* (Canadian Institutes of Health Research, Natural Sciences and Engineering Research Council of Canada, & Social Sciences and Humanities Research Council of Canada, 1998) and approved by the Faculties of Education, Extension and Augustana Research Ethics Board (EEA REB) at the University of Alberta.

Data Collection

Participants

Inclusion criteria. To qualify for participation the following criteria were utilized:

- Report experiencing chronic pain for at minimum 6 months;
- Age 18 to 65 (inclusively);
- Chronic pain not due to burn or co-occurring with a burn;
- Chronic pain not due to cancer and no current diagnosis of active cancer;
- No traumatic brain injury noted;
- Participants able to complete the questionnaires independently

These criterions were utilized to facilitate obtaining a sample representative of the heterogeneous population of interest but homogeneous in regards to chronic pain

experience. As well, the exclusion criteria were chosen to ensure selection of a homogenous chronic pain sample that conforms to samples utilized in the extant literature. Specifically, both cancer- and burn-related pain were excluded because these conditions are studied separately from other chronic pain conditions and head injuries were excluded because a head injury may influence thinking patterns (i.e., beliefs). However, in alignment with the literature reviewed, there were no restriction or inclusion criteria based on other pain conditions and diagnoses because previous findings found no significant differences in beliefs based on these variables (Paul, 2003).

Recruitment. To increase representativeness and generalizability of the results a dual sampling procedure was utilized, where participants were recruited from the community and a treatment facility. In accordance with Statistics Canada (2002) the aim was to obtain approximately 33.3% of people indicating a current compensation/litigation claim. The compensation/litigation sample was recruited from Millard Health Centre in Edmonton, Alberta, Canada, a treatment facility for the Workers' Compensation Board of Alberta (WCB-Alberta). Millard Health Centre focuses on providing treatment for people with work-related injuries. At the WCB-Alberta, a Research Assistant approached potential participants in a chronic pain treatment program.

The community participants were recruited through advertisements in local newspapers, on a local television station, and through posters in hospitals, physical therapy offices, psychologists' offices, and treatment facilities in and around Edmonton, Alberta, Canada. In the community advertisements and posters provided a

brief outline of the study and contact information. The potential participants were instructed to contact the researcher by telephone to request the material for participation. Once a request was made, the Primary Researcher contacted the potential participant via telephone.

During the initial contact, the Primary Researcher for the community sample and the Research Assistant for the treatment sample provided verbal information on the study. Specifically the oral overview outlined the purpose of the study, nature of the study, research procedures, expected duration/nature of participation, potential risks/benefits, the rights of the participant, confidentiality, as well as potential uses and the secure storage procedures of the data. For the treatment sample the Research Assistant also verbally ensured voluntary participation and provided additional information regarding confidentiality procedures. Specifically, the Research Assistant ensured voluntary participation through indicating participation or non-participation would not influence their WCB claim nor would it influence the services/treatment they would receive.

In this initial conversation the Primary Researcher/Research Assistant obtained a self-report diagnosis for chronic pain and screened for the inclusion criteria. If participation was not contraindicated a research package was sent/given to the participant including two copies of a cover letter/consent form (one for the participant and one to be signed and sent back to the researcher) (see Appendix A), the questionnaire (see Appendix B) and a self-addressed, stamped envelope to return the completed questionnaires. Community participants completed the questionnaire and returned it via mail. In the treatment facility, participants were not provided with

envelopes as they returned the completed questionnaire to the Research Assistant during their program.

All participants were provided with the contact information (a telephone number, an e-mail address and a mailing address) for the Primary Researcher if further debriefing was required or if questions, comments or problems arose regarding the study. Participants were also provided with the phone number and e-mail address for the Primary Researcher's supervisor, Dr. Derek Truscott, and the phone number for the Faculties of Education, Extension and Augustana Research Ethics Board (EEA REB) at the University of Alberta if they had any questions regarding their rights and/or the ethical conduct of the researcher. As well, upon request the Primary Researcher provided referral information for low-to-no cost community-based support groups, counselling and crisis intervention services.

Response Rate. For the community sample, 217 people were sent a research package. From the community sample, 136 returned the completed questionnaire to the Primary Researcher. List-wise deletion was utilized for cases endorsing exclusion criteria (i.e., cancer or head injury). As a result 22 were excluded from the community sample (1 for over age restriction, 20 for endorsement of head injury and 1 for indication that pain was caused by cancer). For the treatment sample, a total of 31 questionnaires were returned to the Research Assistant. After list-wise deletion for endorsement of questions related to exclusion criteria, 6 were excluded from the analysis (1 for over age restriction and 5 for endorsement of head injury question).

Sample. The community sample totalled 114 and the treatment group totalled 25. The final sample was 139 (41 men and 98 women). The dual recruitment

procedure resulted in 18% of the sample coming from the treatment recruitment procedure and 82% of the sampling coming from the community recruitment procedure. However, 7 people from the community sample indicated current participation in a treatment program for their pain as well. This brings the distribution up to 30% indicating participation in a treatment program for their pain. As aforementioned, the SEM literature is unclear about the ideal number of subjects required for power but in order to calculate the covariance matrix 136 subjects were needed (Sivo et al., 2006). The current sample meets the criteria for the calculation of the covariance matrix. Demographic data are summarized in Table 1.

Table 1. Descriptive data for demographics of sample.

	Feature	Total sample N_{tot} = 139	
AGE	**Mean**	48.55	
	Standard Deviation	11.616	
	Range	0-65	
DURATION OF PAIN (years)	**Mean**	9.96	
	Standard Deviation	9.59	
	Range	0.5 – 39.2	
	Missing (frequency)	12	8.6%
PAIN RATING	**Mean**	6.030	
	Standard Deviation	1.9505	
	Range	1 – 10	
	Missing (frequency)	7	5.0%
		frequency	**%**
GENDER	**Male**	41	29.5
	Female	98	70.5
	Missing	0	0
MARITAL STATUS	**Married/Common-Law**	77	55.4
	Divorced/Separated	28	20.1
	Single	27	19.4
	Widowed	6	4.3
	Missing	1	.7
EDUCATION	**Less Than Grade 9**	3	2.2
	Partial High School	18	12.9
	High School Diploma	24	17.3
	Partial Technical Or Trade School	4	2.9
	Technical Or Trade School	34	24.5
	Some University	23	16.5
	University Degree	33	23.7
	Missing	0	0
WORK STATUS	**Full-Time**	45	32.4
	Part-Time	19	13.7
	Student	3	2.2
	Volunteer	2	1.4
	Retired	13	9.4
	Unemployed Due To Pain	42	30.2
	Unemployed Other Reason	13	9.4
	Missing	2	1.4

		frequency	%
LIVING SITUATION	**Urban**	117	84.5
	Rural	20	14.4
	Missing	2	1.4
CAUSE OF PAIN	**Work-Related Accident**	40	28.8
	At Work No Accident	9	6.5
	Motor Vehicle Accident	13	9.4
	Post-Surgery	10	7.2
	No Reason	13	9.4
	Other	52	37.4
	Missing	2	1.4
FREQUENCY OF PAIN	**Constant**	104	74.8
	Intermittent	32	23.0
	Missing	3	2.2
PAIN LOCATION	**Head, Face and/or Neck**	65	46.8
	Cervical Region	35	25.2
	Upper Shoulders, Arms and/or Hands	87	62.6
	Thoracic Region	25	18.0
	Abdominal Region	20	14.4
	Low Back	82	59.0
	Pelvic Region	37	26.6
	Legs and/or Feet	87	62.6
	Anal, Perianal and/or Genitals	11	7.9
	Other (E.G., Joint Pain, Diffuse Muscle Pain, Etc.)	51	36.7
NUMBER OF PAIN LOCATIONS ENDORSED	**1**	33	23.7
	2	30	21.6
	3	25	18.0
	4	14	10.1
	5	16	11.5
	6	8	5.8
	7	8	5.8
	8	4	2.9
	9	1	0.7

Instruments

Participants completed the following questionnaires: A Demographic Survey, the Chronic Pain Acceptance Questionnaire (CPAQ), Chronic Pain Intrusion and Accommodation Scale (CPIAS), the Fear-Avoidance Belief Questionnaire (FABQ), Pain Beliefs and Perception Inventory (PBPI), Pain Beliefs Questionnaire (PBQ), Pain Catastrophizing Scale (PCS), Pain Locus of Control Questionnaire (PLC) and the Pain Self-Efficacy Questionnaire (PSEQ).

These questionnaires were chosen for their relevance to the component beliefs of the theoretically-based models. Every effort was taken to minimize the number of questionnaires to reduce participant burden while maintaining the integrity of the study. Each instrument was evaluated for construct valid, reliablity, demonstrated validity for use with chronic pain populations, presence and use in the extant literature and time to complete.

Demographic Survey. A 14-item questionnaire was administered to obtain demographic information, screen for inclusion, and ensure comparison with the extant and future research. To ensure appropriate inclusion the following information was collected: traumatic brain injury, chronic pain due to burn or co-occurring with a burn, and chronic pain not due to cancer or co-occurring with active cancer. To ensure differentiation of the treatment sample from the community sample the following factors were collected: current participation in a rehabilitation program, cause of pain (i.e., work-related). To ensure comparison with future studies the following demographic factors were collected in line with the findings of significant differences between chronic pain populations based on these criteria: Education level, marital

status, pain site, pain constancy, current pain rating, work status and region of habitation (urban or rural) (Hoffman, Meiser & Council, 2002; Kerr et al., 2004).

CPAQ. The CPAQ measures acceptance of pain, which is a willingness to engage in the satisfying and rewarding aspects of life even when experiencing sensations, such as pain, or thoughts that might otherwise direct efforts elsewhere, away from valued activities (Geiser, 1992; McCracken, 1999; McCracken et al., 2004). The CPAQ was originally derived from the Acceptance and Action Questionnaire (Hayes et al., 2003) and included 34 items, of which only 24 were used to calculate the level of Acceptance (Geiser, 1992). The reliability coefficient for the orginal CPAQ was 0.85 (Geiser, 1992).

Factor analytic examination of the item content for this original CPAQ revealed four factors within the overarching construct of acceptance of pain: (1) activity engagement; (2) cognitive pain control; (3) pain chronicity; and (4) pain control through avoidance (McCracken, 1999). But further analysis revealed the second (McCracken, 1999) and third factor (McCracken et al., 2004) were divergent from the other two factors and divergent from the overarching construct of Acceptance. As a result, McCracken et al. (2004) proposed a revision of the CPAQ that limited the content to the first and fourth factors, given research supported relevance and found both factors to be significant predictors of pain-related disability and distress.

This revised version of the CPAQ focused on activity engagement and avoidance, aptly naming the factors Activity Engagement and Pain Willingness, respectively. Activity Engagement measures "the pursuit of life activities in a normal

manner even while pain is being experienced" and Pain Willingness measures the "recognition ... that avoiding or controlling pain are strategies that are often patently ineffective" (McCracken et al., 2004, p. 164). It has 20 items rated on a 7-point Likert scale, where 0 represents never true and 6 represents always true (McCracken et al., 2004). Eleven items measure Activity Engagement (items 1, 2, 3, 5, 6, 8, 9, 10, 12, 15 and 19) and are summed to produce a score measuring participation in usual daily activities regardless of the pain. Nine items measure Pain Willingness (items 4, 7, 11, 13, 14, 16, 17, 18 and 20) and are reversed scored with the total indicating willingness to have pain present without trying to avoid or reduce it. The sum of Activity Engagement and Pain Willingness subscales indicates the degree of Pain Acceptance. The higher the Acceptance score the greater degree of acceptance of pain. This newest version demonstrates adequate content validity (McCracken & Eccleston, 2006) and adequate reliability with Cronbach's alphas of 0.82 for Activity Engagement, 0.78 for Pain Willingness and 0.78 for the total scale (McCracken et al., 2004).

CPIAS. The CPIAS is a 14-item self-report measure of Pain Intrusion and Pain Accommodation beliefs:

> Pain Intrusion, is endorsed by patients who report their pain fluctuations and emotional well-being as reciprocally influenced and predictable. These patients appear to see this kind of predictability as negative and intrusive.... The Pain Accommodation Scale, consisting of items that reflect a perceived ability to live a satisfactory life despite chronic pain, suggests a successful accommodation to the diagnosis of chronic pain, consistent with the common clinical message of 'you must learn to live with it'. (Jacob et al., 1993, p. 525)

The Pain Intrusion subscale consists of items 1 through 8 and the Pain Accommodative subscale consits of items 9 through 14. The items are rated on a 7-point Likert scale, where 0 represents strongly disagree, 6 represents strongly agree and 3 representing neither agreement nor disagreement. The higher the score the stronger the belief. The scales demonstrate discriminant distinctiveness, "good internal consistency" and have test-retest coefficient alphas ranging from 0.71 to 0.80 for the Pain Intrusion Scale and 0.64 to 0.82 for the Pain Accommodation Scale (Jacob et al., 1993, p. 523).

FABQ. The FABQ is a 16-item self-report measure of avoidance of work and other activities due to fears of injury or re-injury (Waddell et al., 1993). It is a good measure of specific fears about pain and activity. This level of specificity is a better predictor of pain severity, disability, and pain behaviour than more general measures of fear or anxiety (McCracken et al., 1996). There are two subscales: beliefs about possible harm from physical activity (FABQ-Physical) and beliefs about possible harm from work-related tasks (FABQ-Work). Factor analytic studies support four items on the FABQ—Physical subscale (iems 2, 3, 4 and 5) and seven items on the FABQ-Work subscale (items 6, 7, 9, 10, 11, 12 and 15) (Waddell et al., 1993). The FABQ has demonstrated good psychometric properties (Crombez et al., 1999; Fritz et al., 2001; Waddell et al., 1993) with Cronbach alpha coeffients of 0.84 for FABQ-Work subscale and 0.57 for FABQ-Activity (Crombez et al., 1999).

PBPI. The PBPI is a 16-item self-report instrument measuring peoples' beliefs about their chronic pain (Williams & Thorn, 1989). In the original version three factors were presented: Pain Stability, Pain as a Mystery and Self-Blame (Williams &

Thorn, 1989). However, a four factor structure is now recommended due to inconsistent findings replicating the factor structure (Herda et al., 1994; Strong et al., 1992; Williams et al., 1994). The four factors are Pain Permanence (reflecting the belief pain is enduring), Pain Constancy (reflecting the belief pain is constant and pervasive in its effects on daily living), Pain as a Mystery (reflecting the belief pain is mysterious and poorly understood) and Self-Blame (reflecting the belief pain is caused by and maintained by the person). In accordance with these findings Williams et al. (1994) recommended the scales consist of the following items: The Pain Permanence subscale consists of items 2, 5, 9(R), 12(R) and 15(R), Pain Constancy consists of items 3(R), 6, 10 and 16. The Pain as a Mystery subscale consists of items 1, 4, 8 and 14. The Self-Blame subscale consists of items 7, 11 and 13. Each item is rated on a scale from -2 to 2 (with no zero point) with -2 indicating strongly disagree and 2 indicating strongly agree. Those items depicted with an R are reversed scored for the subscale score. The PBPI has demonstrated adequate reliability with Cronbach alphas of 0.81 for Pain Permanence, 0.80 for Pain Constancy, 0.83 for Mystery and 0.89 for Self-Blame (Morley & Wilkinson, 1995).

PBQ. Edwards et al. (1992) created the PBQ. It has two scales: The Organic Belief Scale has eight items (1, 2, 3, 5, 7, 8, 10 and 11) and the Psychological Belief Scale has four items (4, 6, 9, and 12). The Organic Belief Scale is associated with the belief that other people (usually doctors) control their pain. The Psychological Beliefs Scales is associated with the belief that internal states influence the experience of pain. The PBQ has demonstrated good reliability with chronic pain populations with

Cronbach alphas of 0.71 for the Organic subscale and 0.73 for the Psychological subscale (Edwards et al., 1992).

PCS. The PCS is a 13-item self-report questionnaire that measures exaggerated and negative interpretations of pain (Sullivan et al., 1995). In the PCS Catastrophizing is viewed as a general construct encompassing Magnification, Rumination and Helplessness. Research into these dimensions has supported the distinctiveness of these three factors (Osman et al., 2000; Sullivan et al., 1995; Sullivan et al., 2005; Sullivan et al., 2001; Van Damme et al., 2002). The PCS utlizes a 5-point Likert scale and the total scores range from 0 to 52, with higher scores indicating stronger Catatrophizing beliefs. The Magnification subscale consists of three items (6, 7 and 13), Rumination four items (8, 9, 10 and 11) and Helplessness six items (1, 2, 3, 4, 5 and 12).

The PCS and each subscale individually has demonstrated adequate to excellent reliability coefficients (Chibnall & Tait, 2005; D'Eon et al., 2004; Osman et al., 2000; Osman et al., 1997; Sullivan et al., 1995). These studies report coefficient alphas ranging from 0.60 to .88 for Magnification, 0.85 to 0.87 for Rumination, .79 and .89 for Helplessness and between .87 and .92 for the entire PCS. The reliability indices indicated in the PCS manual are 0.66 for the Magnification subscale, 0.87 for Rumination, .78 for Helplessness and .87 for the entire PCS (Sullivan, 2004).

PLC. The PLC is a 20-item self-report questionnaire developed specifically for use with people with chronic pain to assess the degree people believe they can control their pain (Pain Control [PC] subscale: items 2, 4, 8, 10, 11, 12, 14, 16, 18 and 20) and the degree they believe they are responsible for their pain (Pain Responsibility [PR]

subscale: items 1, 5, 9, 17 and 19) (Main & Waddell, 1991). The scores range from 0 to 30 on the PC subscale and 0 to 15 on the PR subscale. The higher the score the stronger beliefs regarding Pain Control and Pain Responsibility. The PLC has demonstrated sensitivity to treatment changes (Main & Waddell, 1991) and internal consistency and reliability (Asghari & Nicholas, 2001). The test-retest reliability coefficients are 0.95 for the PC subscale and 0.67 for the PR subscale. The Cronbach alphas for the PC and PR subscales are 0.72 and 0.83, respectively.

PSEQ. Nicholas (1989) developed the PSEQ based on Bandura's (1977) concept of self-efficacy. It is a 10 item self-report inventory where people rate their confidence about their ability to complete each task despite the pain. The scale utilizes a 7-point likert scale, with 0 representing not at all confident and 6 representing completely confident. The scores range from 0 to 60, with higher scores indicating more confidence and, therefore, stronger self-efficacy beliefs. The PSEQ has demonstrated excellent reliability with Cronbach alphas of 0.92 (Nicholas, 2007).

Structural Equation Modeling Procedure

In line with the concerns presented in the SEM literature review, Martens (2005) indicated some common misuses of the method in Counselling Psychology. Specifically citing the following concerns: (1) failure to assess for multivariate normality prior to completing SEM analysis, (2) lack of identification of alternative models, (3) failure to assess the measurement model separately from the structural model, (4) misuse or confusion regarding which fit indices to utilize, (5) failure to fully disclose parameter estimates, and (6) utilizing SEM to generate the model or modifying the model based on empirical rather than theoretical criteria. In the next

section the SEM procedure for the current study is presented to address each area of concern and common misuse. Specifically the procedures address the following areas: multivariate normality, identification concerns, measurement model assessment, fit indices, disclosure of parameter estimates, and modification of the model.

Multivariate Normality

The parameter estimation procedure, maximum likelihood estimation (ML) and Multiple Imputation (MI) for missing data assume multivariate normality (Allison, 2003). To address multivariate normality concerns it is recommended outliers for possible removal be identified in the sample data (Yuan & Bentler, 2001). Once this is complete the skewness and kurtosis for each variable is examined through univariate and multivariate tests for normality (Mardia's coefficient in LISREL). Evaluation of the data for outliers and multivariate normality must always be completed on the raw data rather than imputed data.

Identification Concerns

A priori development of theoretically-based models addresses identification concerns. In the current study three models were presented: the Theoretical Model, the Parsimonious Theoretical Model and the Less Restricted Theoretical Model. The Theoretical Model is the most likely model based on the available information. But, this model was developed based on subjective analysis of the literature examined and no studies examined all the identified variables simultaneously. Therefore, it is possible that other equivalent or non-equivalent models may exist (Tomarken & Waller, 2003).

An adapted version of Anderson and Gerbing's (1988) two-step procedure facilitated development of the alternative nested models. The Parsimonious Theoretical Model is "the next most likely" constrained model where fewer relationships are modelled than the Theoretical Model. On the other hand, the Less Restricted Theoretical Model is "the next most likely" model where more direct relationships are included to test the mediation restrictions of the Theoretical Model.

For all three models the exogenous variables are Organic Causation beliefs, Psychological Influence beliefs, Pain as a Mystery beliefs, Pain Permanence beliefs, Pain Constancy beliefs, Self-Blame beliefs and External Pain Responsibility beliefs. The endogenous variables are Internal Pain Control beliefs, Accommodative beliefs, Assimilative beliefs, Worry, Catastrophizing, Acceptance beliefs, Pain Self-Efficacy beliefs, Activity-Related Fear-Avoidance beliefs and Work-Related Fear-Avoidance beliefs.

Measurement Model Assessment

In Anderson and Gerbing's (1988) original formulation analysis of a Saturated Model and Null Model allowed for separate examination of the measurement model, which aligns with Martens' (2005) recommendation. However, the current study utilizes single indicators and with single indicators the Saturated and Null Models do not provide the best strategy for evaluation of the measurement model. Specifically, the Saturated Model has low testing power because there are zero degrees of freedom (it is the pattern of constraints and lack of constraints placed within a model that increase testing power). As well, there are too many constraints in the Null Model to provide testing power (no variable is related to the other variables). Therefore, in this

study Anderson and Gerbing's two-step procedure was adapted and alternative modalities were utilized to test the measurement model; such as instrument selection, a sensitivity analysis, colinearity analysis and through examination of the structural model.

Instrument Selection. The primary tests of the measurement model are the test construction and construct validation studies for each questionnaire: Demonstrated statistical robustness through standardization and validation procedures with a chronic pain population were the primary criterion for instrument selection. Each instrument was chosen as indicator based on the construct validity of the subscale in relation to the latent constructs of interest. In addition, to maximize variation in the indicator scores and provide more robust results formulative indicators, where the items are added to create a total score, were utilized rather than the standardized subscale score.

As well, the error variance for each indicator was set to correspond with the externally derived reliability coefficient for the subscale (Schumacker & Lomax, 2004). In accordance with Joreskog and Sorbom's (1997) procedure, error terms were based on the alpha coefficient for the respective standardized subscales or parent scales. Cronbach's coefficient alphas were utilized for all instruments, except the CPIAS where only test-retest alpha coefficient was available. If a range of reliability coefficients were provided then the lowest reliability coefficient was utilized. Error terms were calculated through multiplying the result of 1 minus the alpha coefficient for the subscale by the variance of the variable (Anderson & Gerbing, 1988; Lackner, Jaccard & Blanchard, 2005) (see Table 2).

Table 2. Hypothesized constructs, instrumentation and error variance.

Variable	Latent Variable	Indicator	Reliability for scale	Variable Variance	Error Variance
Y1	Internal Pain Control beliefs	PC subscale of PLC	0.72	34.349	9.61772
Y2	Accommodative beliefs	Accommodative subscale of CPIAS	0.64	54.387	19.57932
Y3	Assimilative beliefs	Pain Intrusion subscale of CPIAS	0.71	79.331	23.00599
Y4	Acceptance beliefs	CPAQ	0.78	376.129	56.41935
Y5	Pain Self-Efficacy beliefs	PSEQ	0.92	215.277	17.22216
Y6	Worry beliefs	Rumination subscale of PCS	0.87	22.955	2.98415
Y7	Catastrophizing beliefs	Magnification subscale of PCS	0.66	8.698	2.95732
Y8	Activity-Related Fear-Avoidance beliefs	Activity subscale of FABQ	0.57	31.712	13.63616
Y9	Work-Related Fear-Avoidance beliefs	Work subscale of FABQ	0.84	177.606	28.41696
X1	Organic beliefs	Organic Beliefs subscale of PBQ	0.71	37.204	10.78916
X2	Psychological Influence beliefs	Psychological Influence subscale of PBQ	0.73	18.280	4.9356
X3	Pain as a Mystery beliefs	Pain as a Mystery subscale of PBPI	0.83	13.117	2.22989
X4	Pain Permanence beliefs	Pain Permanence subscale of PBPI	0.81	12.673	2.40787
X5	Pain Constancy beliefs	Pain Constancy subscale of PBPI	0.80	8.432	1.6864
X6	Self-Blame beliefs	Self-Blame subscale of PBPI	0.89	4.812	0.52932
X7	External Pain Responsibility beliefs	PR subscale of PLC	0.83	8.084	1.37428

Sensitivity Analysis. To expand measurement model examination a sensitivity analysis was completed, through altering measurement error specification for each indicator (Hayduk, 1987). Hayduk (1987) recommended running the model repeatedly with the measurement error for each indicator ($\Theta\varepsilon$ and $\Theta\delta$ values) halved and then run again with the error term doubled. Hayduk reported that if the parameter estimates remain similar than it can be concluded the measurement model is not *overly sensitive* or *insensitive* to measurement specifications. If the sensitivity analysis passes then any subsequent difficulties can be attributed to the structural model because different measurement specification would not have produced better results (Hayduk, 1996; Hayduk, 1987).

ColinearityAnalysis. Further, a common problem in the measurement model is colinearity. Colinearity can be problematic because the parameter estimates for variables that are highly correlated may be inaccurate or unstable (Hayduk, 1987). Hayduk recommends examining correlations for exogenous variables to check for colinearity problems. Examining the ξ correlations provides evidence the exogenous constructs are independent. However, high correlations only provide partial support for endogenous colinearity problems because the structure of the model highly influences the correlations between the endogenous variables; the correlations among the endogenous latent variables are implications of several parameters not simply a single coefficients/parameter (Hayduk, 1987). As a result a very high correlation is necessary to indicate colinearity problems. Hayduk (1987) cites 0.90 as an acceptable criterion for colinearity; however in the literature a more conservative criterion of 0.80 is typically utilized.

If colinearity is indicated, Lackner et al. (2005) note three strategies are available to remedy it: (1) the correlated indicators be modeled as indicators of a common more general latent variable (see also Hayduk, 1987); (2) the error terms associated with each of the indicators be allowed to correlate to recognize the influence of a common unmodeled unknown variable on the indicator; or (3) or reciprocal causality be introduced, whereby a non-recursive relationship is modeled between the variables of interest.

The first strategy of utilizing the constructs as multiple indicators of a broader construct is chosen if the variables are modeled to be similarly related to other constructs in the model. If in theory they are differentially related to other constructs then this method is rejected. The second strategy is "justified theoretically if one can specify variables outside of the theoretical system that might serve as common causes of the two constructs" (Lackner et al., 2005, p. 213). If in theory there are no plausible common causes then this method is rejected. The third strategy is chosen if it is theoretically justifiable the variables represent an independent feedback loop. However, the addition of a feedback loop into a model does not add effects but rather "loops multiplicatively enhance any basic direct or indirect effects they touch" (Hayduk, 1987, p. 272). If in theory it is not plausible the variable influences itself then this method is rejected.

Examination of the Structural Model. Martens (2005) cited failure to assess the measurement model separately from the structural model as a misuse of SEM. But, according to Hayduk (1987) the test for the endogenous measurement model *is* the structural model. Specifically Hayduk indicates when utilizing single indicators the

researcher is asserting the meaning for each latent variable through fixing the error variance. Thus, if the structural model is found to fit well then this is strong support for the endogenous constructs.

Fit Indices

To address Martens' (2005) concerns regarding inappropriate utilization of fit indices a thorough review of the fit indices available was completed and the indices utilized were chosen based on robustness of findings for a small sample size. In accordance with the recommendations of Hayduk (1987) as well as Anderson and Gerbing (1984) χ^2 was utilized; and in accordance with the recommendations of Sivo et al. (2006) the SRMR and the RMSEA were utilized.

χ^2. For a model to fit the χ^2 would have to be insignificant, within 2 standard deviations from 0, meaning there is no significant difference between the sample covariance matrix and the model implied population covariance matrix. The criterion for usage of the χ^2 difference test was as follows: If more than one model passes then the χ^2 difference test can be computed to determine the 'best' model and Anderson and Gerbing's (1988) decision tree can be utilized. This procedure provides an objective way to determine the best fitting model when more than one model fits (Anderson & Gerbing, 1988). If no models fit then the χ^2 difference test can not be used because comparison of two ill fitting models does not facilitate distinction of the appropriate model (McMurtry, 2004).

SRMR. The SRMR is a standardized version of the RMR which reflects average discrepancy between predicted and observed covariance (Jaccard & Wan, 1996). RMR are estimated based on the assumption the model is correct. The RMR is

the coefficient resulting from taking the square root of the mean of the squared residuals, which is the amounts the sample variances/covariances differ from the corresponding model implied variances/ covariances (Garson, 2006). The SRMR puts all of the variables in the same metric to allow comparison and determination of the location of the largest residuals.

RMSEA. The RMSEA quantifies the "discrepancy per degree of freedom" through comparing the hypothesized model to a saturated model and therefore the RMSEA prefers more parsimonious models (Garson, 2006). This is problematic if the modeled relationship structure is complex then a parsimonious model may be statistically sounder according to the RMSEA but may not reflect the real world. In fact, a parsimonious model may be an equivalent or non-equivalent model for the 'true' model. However, despite reliance on parsimony the RMSEA does *not* tend to over-reject complex models (Cheung & Rensvold, 2002).

Optimal Index Values. In a thorough analysis of the robustness of fit indices Sivo et al. (2006) recommend for a sample size of 150 the optimal index values of 0.12 for SRMR and 0.06 for RMSEA be utilized to ensure no correct models are rejected. As well, they recommend the optimal index values for rejecting all misspecified models is 0.06-0.12 for the SRMR and 0.00-0.05 for the RMSEA with a sample size of 150. Therefore, the criteria of 0.06 for RMSEA and 0.12 for SRMR were utilized to ensure that no correct models are rejected.

Disclosure of Parameter Estimates

Parameter estimates are calculated based on the indicator loading, regression weight for paths between the latent variables, error terms for the endogenous variables

and the variance for the exogenous variables. ML estimates utilize these four components to provide the best guess that the discrepancy between the estimate and the observed sample is a result of sampling fluctuations. It is "the quantification of the magnitude and seriousness of the departures between what is observed and the various estimates of the population coefficients.... Maximizing the likelihood minimizes what must be attributed to sampling fluctuations" (Hayduk, 1987, p. 131-132). The question answered with ML estimates is: "How likely is it that the observed sample information could appear if the population parameters took on any particular set of values" (p. 132). Each ML estimate is associated with a standard error of estimate quantifying the accuracy of each free parameter. The standard errors are correct under multivariate normal distributions: symmetric, smooth and static distribution with dynamic values between the variables. But they are also "robust against moderate departures from normality" (Jöreskog & Sörbom, 2001, p. 26; see also Amemiya & Anderson, 1990; Anderson & Amemiya, 1988; Browne, 1987; Browne & Shapiro, 1988; Chou, Bentler & Satorra, 1991; Hu & Bentler, 1998; Martens, 2005; McDonald & Ho, 2002; Mooijaart & Bentler, 1991; Satorra, 1992; Satorra & Bentler, 1990; Shapiro, 1987; Yuan & Bentler, 1999).

If the parameter is estimated accurately the standard errors should be small. Large standard errors may indicate unidentified coefficients and/or identification problems. However, the estimates are provided in the units of the corresponding latent variable so magnitude of the size of standard errors is determined through *t*-values (*t*-value = parameter estimate/standard error). If the *t*-value is between -1.96 and +1.96, it is not significantly different from zero, so fixing it to zero (indicating no chain of

influence) will not make the fit of the model significantly worse (Jöreskog & Sörbom, 2001). However, it is not recommended to fix a parameter to zero based solely on parameter estimates:

> Eliminating a parameter on the basis of its *t*–value may...be dangerous, especially in a small sample. Even non-significant parameters may be of practical importance. If the substantive theory suggests that a particular parameter should be included in the model, it is probably better to retain it even though it is not significant, because the sample size may be too small to detect its real significance. (Jöreskog & Sörbom, 2001, p. 275)

Therefore, interpretation of the parameters with non-significant *t*-values must be done with caution as it is likely that the parameter estimates are not estimated accurately (Jöreskog & Sörbom, 2001).

Potential Problems. Along with disclosing the fit indices for the model it is crucial the parameter estimates be disclosed because at times a model can fit well but the parameter estimates are outside of the expected range or opposite valence than expected (McMurtry, 2004). Two common unreasonable parameters occur with opposite value expected and Heywood cases. Heywood cases occur when the standardized value for a parameter is outside of the range of 0 to 1.

In general, these 'unreasonable' parameter estimates may indicate specification problems. Findings of significant parameters in the opposite direction hypothesized suggest "the data does not conform to the hypothesized theory, even if the model provides adequate fit to the data" (McMurtry, 2004, p. 78). These unexpected parameters may indicate problems with the structure of the data, the coding,

colinearity or identification problems. But, both can also be deemed acceptable under certain conditions. Specifically, unexpected parameter estimates may be a reflection of contention in the extant literature (i.e., contradictory findings).

On the other hand, a Heywood case can be acceptable if a variable has two predictors of opposite valence occurring simultaneously. Jöreskog (1999) remarked that the one-dimensional perception of Heywood cases as 'unreasonable':

> Probably stems from classical exploratory analysis where factor loadings are correlations if a correlation matrix is analyzed and the factors are standardized and uncorrelated (orthogonal). However, if the factors are correlated (oblique), the factor loadings are regression coefficient and not correlations and as such they can be larger than one in magnitude. This can indeed happen also for any factor loading or structural coefficient in any LISREL model. (p. 1)

However, most standardized parameter estimates are regression coefficients or structural coefficients. Unlike correlation coefficients, both regression coefficients and structural coefficients can exceed a magnitude of 1 and still be reasonable. Full disclosure of parameter estimates allows for a determination of the 'reasonableness' of a Heywood case and opposite valence coefficients.

Modification of the Model

In the event none of the models fit the data well a specification search with the best fitting or, alternatively, the most theoretically consistent model is not contraindicated in the literature presented. Modification indices (MIs) show where relationships can be added to the model to improve the overall fit. MIs are provided in χ^2 units and indicate how much χ^2 would change as a result of freeing this parameter

(Joreskog & Sorbom, 2001). This is paired with an expected change unit and a standardized expected change unit. These quantify the potential value of the parameter were it to be freed. The standardized version places the χ^2 units into a standardized metric with a standard deviation of 1 which means those values closer to 1 will result in greater change.

However, freeing a parameter increases the degrees of freedom by 1 and so it is recommended only those values approaching or closest to 1 standard deviation in χ^2 units, which is approximately 4, be considered for freeing. Utilization of MIs and expected change units in conjunction with theoretical soundness maximizes generalizability. This procedure increases the probability all changes are reflective of the population of interest rather than the idiosyncratic sample: It is recommended the parameter with the highest amount of χ^2 units that is consistent with theory be modified. It is recommended this modification be completed in a stepwise fashion to control for Type I error rate (Joreskog & Sorbom, 1989; Sorbom, 1989). This procedure is repeated until all the constrained parameters are non-significant.

But, this could result in the creation of an overly complex saturated model with limited testing power. Utilization of empirical data to generate or modify a model results in a model that can reflect primarily the idiosyncrasies of the sample. It reduces generalizability of the findings to the population of interest (Martens, 2005). Therefore, specification searches must be completed carefully to avoid Type I error (inclusion of parameters that should not be included) (Green & Babyak, 1997) and Type II error (deleting parameters that should be included) (Cohen, 1994; Kaplan, 1989; Schmidt, 1996). The best practice is full disclosure of the MIs and the

theoretical rationale for any adjustments. In other words, only those modifications in accordance with the literature are made. Further, Green et al.'s (1998) suggest a minimal criterion for the deletion or addition of a parameter (based on the modification indices) be set in advance. They also recommend utilizing an adjusted Bonferroni approach, with increasing rigour for each step in the specification search to reduce Type I error:

> A reduced alpha level is computed for a step by dividing the familywise error rate by the number of parameters tested at a step. Because the divisor for the reduced alpha level decreases by one at each successive step in the search process, the reduced alpha level increases across steps. The critical value for a parameter with the adjusted Bonferroni approach is a chi square with degrees of freedom of 1 and the reduced alpha level. (Green et al., 1998, p. 368)

The formula to calculate the adjusted Bonferroni is 0.05 divided by the number of parameters tested (number of free parameters added to the model). This equals the critical χ^2 value for an alpha level of 0.05 (see Table 3).

Table 3. Bonferroni adjustment indicating the critical χ^2 value for alpha level of 0.05.

	Significance level/ Number of tested parameters	**Adjusted Bonferroni**	**Critical χ^2value**
Step 1	0.05/1	0.05	3.84
Step 2	0.05/2	0.025	7.38
Step 3	0.05/3	0.017	9.35
Step 4	0.05/4	0.013	11.14
Step 5	0.05/5	0.01	15.09
Step 6	0.05/6	0.008	16.81
Step 7	0.05/7	0.007	18.48
Step 8	0.05/8	0.006	20.09
Step 9	0.05/9	0.006	21.67
Step 10	0.05/10	0.005	25.19
Step 11	0.05/11	0.005	26.76
Step 12	0.05/12	0.004	28.30
Step 13	0.05/13	0.004	29.82

On the other hand, it is recommended the criterion for parameter freeing be 1 standard deviation of change to χ^2, -1.96 and +1.96. This criterion offsets the effect of adding 1 degree of freedom. However, to reduce Type II error it is recommended outlying parameters within the acceptance criterion range also be considered for theory-based modification. Note only theoretically supported parameters are recommended for modification given extraneous factors can influence greatly parameter estimates and any changes not based on theory once again decrease generalizability and jeopardize the validity of the model.

To facilitate this process and provide procedural transparency the parameter estimates considered for constraint or freeing need to be fully disclosed, along with the error variances and the R^2 values (Martens, 2005). Finally, cross-validation needs to be explicitly recommended for any theoretically based adjustments made on the basis of the parameter estimates or modification indices. If all the models fail and can only be made to fit through a series of modifications then this cross-validation is essential. As well, this situation would necessitate a more detailed review of the results to determine if the difficulty was measurement or relational (Hayduk, 1987; 1996).

Chapter 5: Results

This chapter presents the results of the analysis. Beginning with the data preparation, including examination of missing data, testing for multivariate normality and completion of Multiple Imputation. This is followed with calculation of the covariance matrix and completion of the SEM analysis, focusing on both measurement model testing and structural model testing. Finally the section closes with a structural analysis of the passing model, The Less Restricted Theoretical Model, and a visual diagram of the significant paths for the passing model.

Data Preparation

Missing Data. Participants were coded for missing or no missing data on the 16 variables of interest. A frequency analysis was completed to determine the percentage of cases with missing data within each category of the variables of interest. A between subjects one-way analysis of variance (ANOVA) was performed on the 16 variables of interest to determine if the mean differences among participants with missing data and participants with no missing data are likely to have occurred by chance. None of the F-values reached significance (see Table 4) which supports the conclusion there are no systematic differences between participants with missing data and those with complete data.

Table 4. Between subjects one-way analysis of variance (ANOVA) for missing data.

Indicators	Latent Variable	f-values	significance
Y1	Internal Pain Control beliefs	1.106	0.295
Y2	Accommodative beliefs	0.616	0.434
Y3	Assimilative beliefs	0.747	0.389
Y4	Acceptance beliefs	0.10	0.919
Y5	Pain Self-Efficacy beliefs	0.013	0.909
Y6	Worry beliefs	2.007	0.159
Y7	Catastrophizing beliefs	0.449	0.504
Y8	Activity-Related Fear-Avoidance beliefs	0.006	0.937
Y9	Work-Related Fear-Avoidance beliefs	0.363	0.548
X1	Organic beliefs	2.231	0.138
X2	Psychological Influence beliefs	0.286	0.594
X3	Pain as a Mystery beliefs	0.285	0.595
X4	Pain Permanence beliefs	0.556	0.457
X5	Pain Constancy beliefs	0.748	0.389
X6	Self-Blame beliefs	0.113	0.737
X7	External Pain Responsibility beliefs	0.216	0.643

Multivariate Normality. To address multivariate concerns the sample raw data was examined for outliers for possible removal through the use of histogram frequency analyses. No outliers were located in the sample raw data. Then the skewness and kurtosis of each variable was examined through univariate and multivariate tests for normality in the raw data (Mardia's coefficient) in LISREL, which yielded a non-significant result (0.998, $p > 0.05$) (Mardia, 1970). These results indicate no correction to the data was necessary because there was insufficient evidence that the assumption of a multivariate data distribution was violated.

Multiple Imputation. Multiple Imputation was chosen as the best method to manage missing data, ensure efficient usage of the data and ensure accurate parameter estimates. Similar to pair-wise, list-wise and maximum likelihood techniques for missing data, Multiple Imputation assumes missing data are "ignorable" and assumes multivariate normality (Allison, 2003, p. 545). It computes new values through "iterated linear regressions in which each variable with missing data is regressed on other observed variables" (p. 550). It creates a regression line with the variables missing data and other variables in the data set with random variation in error based on the standard distribution. The result is a range of imputations for the missing values with each run. The variables utilized in this process must all be in the model and any other variables associated with the variables with missing data or with the probability that those variables will have missing data.

According to the data preparation analyses the missing data are likely to have occurred by chance and there was evidence that univariate and multivariate normality was not compromised, therefore, Multiple Imputation was utilized to compensate for missing values. All of the variables associated with the model were included in Multiple Imputation.

Ninety-four of the 139 participants had complete data sets. This equates to 67.6% with complete data sets and 32.4% with some missing information on the variables of interest. According to Allison (2003) this proportion of missing information requires three imputations for 90% efficiency, five imputations for 94% efficiency, 10 imputations for 97% efficiency and 20 imputations for 99% efficiency. To ensure more stable parameter estimates, standard error estimates and test statistics

20 imputations were completed, which provides the most efficient MI estimates based on 30% missing information (Allison, 2003).

After the imputation the kurtosis and skewness of the variables for the full sample remained within normal ranges and the Mardia's coefficient was 1.041. These results suggest the assumption of multivariate data distribution can be maintained after Multiple Imputation: There is insufficient evidence Multiple Imputation violated the assumption of a multivariate data distribution. Therefore, no correction to the data was necessary.

Covariance Matrix

The covariance matrix was computed, which is the foundation upon which the hypothesized population matrix is compared. The means, standard deviations and covariance matrix for the endogenous and exogenous indicators are presented in Tables 5 and 6.

Table 5. Means, standard deviations and variances for indicators.

Indicators	Latent Variable	Mean	Standard Deviation	Variance
Y1	Internal Pain Control beliefs (PC)	19.987	5.861	34.349
Y2	Accommodative beliefs (Accom)	24.113	7.375	54.387
Y3	Assimilative beliefs (Assim)	27.129	8.907	79.331
Y4	Acceptance beliefs (Accep)	58.806	19.394	376.129
Y5	Pain Self-Efficacy beliefs (PSEQ)	32.748	14.672	215.277
Y6	Worry beliefs (Worry)	7.953	4.791	22.955
Y7	Catastrophizing beliefs (Cat)	4.201	2.949	8.698
Y8	Activity-Related Fear-Avoidance beliefs (FA-PA)	13.367	5.631	31.712
Y9	Work-Related Fear-Avoidance beliefs (FA-W)	21.884	13.327	177.606
X1	Organic beliefs (Org)	24.568	6.099	37.204
X2	Psychological Influence beliefs (Psy)	13.099	4.276	18.280
X3	Pain as a Mystery beliefs (Mys)	9.492	3.622	13.117
X4	Pain Permanence beliefs (PPerm)	15.197	3.560	12.673
X5	Pain Constancy beliefs (PCon)	13.245	2.904	8.432
X6	Self-Blame beliefs (S-Blam)	4.640	2.194	4.812
X7	External Pain Responsibility beliefs (PR)	6.214	2.843	8.084

Table 6. Covariances for the indicators.

Indicator	Y1 PC	Y2 Accom	Y3 Asssim	Y4 Accep	Y5 PSEQ	Y6 Worry	Y7 Cat	Y8 FA-PA	Y9 FA-W	X1 Org	X2 Psy	X3 Mys	X4 PPerm	X5 PCon	X6 SBlam	X7 PR
Y1	34.349															
Y2	-17.084	54.387														
Y3	-24.494	2.493	79.331													
Y4	-18.809	109.079	-12.765	376.129												
Y5	-26.658	84.430	14.721	226.704	215.277											
Y6	12.480	-18.945	-0.183	-50.207	-31.514	22.955										
Y7	4.365	-8.865	3.445	-27.830	-14.297	8.749	8.698									
Y8	3.690	-19.354	-1.272	-55.551	-45.559	8.078	5.426	31.712								
Y9	8.314	-42.611	-10.585	-131.947	-113.079	12.860	8.613	34.271	177.606							
X1	-13.354	22.575	11.672	63.365	50.072	-15.100	-7.688	-16.203	-29.269	37.204						
X2	13.782	-8.854	-19.214	-8.213	-13.908	2.414	0.237	1.169	9.711	-4.858	18.280					
X3	6.187	-10.663	-2.256	-23.138	-15.743	8.453	3.939	2.957	6.721	-9.398	1.208	13.117				
X4	4.457	-6.498	6.983	-8.389	-7.384	4.815	2.864	-1.250	0.603	-0.723	0.488	1.048	12.673			
X5	5.387	-9.450	-0.655	-22.568	-21.699	4.806	2.168	4.265	11.557	-5.589	3.456	3.919	2.974	8.432		
X6	-3.066	-0.078	3.503	0.263	1.713	-0.588	0.348	0.459	-2.489	0.822	-0.888	-0.521	0.615	-1.310	4.812	
X7	-3.935	7.383	-2.246	20.856	14.902	-4.680	-2.846	-4.269	-7.265	4.349	0.309	-0.618	-1.852	-1.321	0.228	8.084

Structural Equation Modeling

Measurement Model Testing

Sensitivity Analysis. The sensitivity analysis was completed with the three models. Within the sensitivity analysis the majority of the parameter estimates remained similar. This provides evidence the measurement model was not overly sensitive or insensitive to measurement specifications and supports the measurement model (Hayduk, 1987). However, some of the altered models would not run due to Not Positive Definite fitted covariance matrices and all of the models that ran resulted in Not Positive Definite Ψ matrices. In all models the Not Positive Definite results indicated the explained variance of Y4 (the indicator for Acceptance Beliefs) exceeded 1. This suggests the error associated with the latent construct of Acceptance was over accounted for. However, the Ψ matrix was the source for the Not Positive Definite finding suggesting the measurement model was not the source of the problems. The source was most likely within the structural model of the latent variables and as such it is likely alternative measurement specifications would not have produced better results (Hayduk, 1987). These results are further explained in the discussion chapter.

Colinearity. A correlational analysis was completed to check for colinearity problems (see Table 7). None of the correlations exceeded the criterion of 0.90 (Hayduk, 1987) or the more conservative criterion of 0.80. The highest correlations were between Accommodation ($\eta 2$) and Acceptance ($\eta 4$) (0.765, $p<0.000$); Accommodation ($\eta 2$) and Pain Self-Efficacy ($\eta 5$) (0.783; $p<0.000$); and between Acceptance ($\eta 4$) and Pain Self-Efficacy ($\eta 5$) (0.797; $p<0.000$). These correlations

were within the borderline range and between endogenous variables and, therefore, no modifications were made to the measurement or structural models (Hayduk, 1987).

Table 7. Correlations between indicators.

Indicator	Y1 PC	Y2 Accom	Y3 Asssim	Y4 Accep	Y5 PSEQ	Y6 Worry	Y7 Cat	Y8 FA-PA	Y9 FA-W	X1 Org	X2 Psy	X3 Mys	X4 PPerm	X5 PCon	X6 SBlam	X7 PR
Y1	1.00															
Y2	***-0.395****	1.00														
Y3	**-0.469****	*0.038*	1.00													
Y4	*-0.165*	**0.763****	-0.074	1.00												
Y5	*-0.310***	**0.780****	*0.113*	**0.797****	1.00											
Y6	***0.444****	**-0.536****	*-0.004*	**-0.540****	**-0.448****	1.00										
Y7	*0.253*	**-0.408****	0.131	**-0.487****	-0.330**	**0.619****	1.00									
Y8	*0.112*	**-0.466****	*-0.025*	**-0.509****	**-0.551****	0.299*	0.327**	1.00								
Y9	*0.106*	**-0.434****	*-0.089*	**-0.511****	**-0.578****	0.201	0.219	**0.457****	1.00							
X1	**-0.374****	***0.502****	0.215	***0.553****	***0.560****	***-0.517****	***-0.427****	***-0.472****	***-0.360****	1.00						
X2	**0.550****	*-0.281**	**-0.505****	*-0.099*	*-0.222*	*0.118*	*0.019*	*0.049*	*0.170*	-0.186	1.00					
X3	*0.291**	**-0.399****	*-0.070*	-0.329**	-0.296*	**0.487****	**0.369****	0.145	0.139	***-0.425****	*0.078*	1.00				
X4	*0.214*	-0.248	0.220	-0.122	-0.141	0.282*	0.273	*-0.062*	0.013	*-0.033*	*0.032*	*0.081*	1.00			
X5	*0.317***	**-0.441****	*-0.025*	**-0.401****	**-0.509****	0.345**	0.253	0.261	0.299*	*-0.316**	*0.278*	***0.373****	0.288*	1.00		
X6	-0.238	-0.005	0.179	0.006	0.053	-0.056	*0.054*	*0.037*	-0.085	*0.061*	*-0.095*	-0.066	*0.079*	-0.206	1.00	
X7	-0.236	***0.352****	*-0.089*	***0.378****	***0.357****	-0.344**	-0.339**	-0.267	-0.192	0.251	*0.025*	*-0.060*	*-0.183*	-0.160	*0.037*	1.00

* = $p < 0.005$ ** = $p < 0.001$ *** = $p < 0.0005$ *italics* = contrary direction to hypothesized **bold** = significant

Observed Valence of Correlations. The correlations were evaluated utilizing a Bonferroni adjustment to correct for the number of tests completed. This adjustment ensured the results were not due to chance (Strong et al., 1992). To retain an overall Type I error rate of 0.05, the level of significance required was 0.0003 (0.05/128) (Miles & Shevlin, 2001). The findings suggest the directionality of some correlations was contrary to the hypothesized direction (denoted as italicized in Table 7). However, only a few of the reversed correlations were statistically significant. The following significant correlations demonstrated a negative relationship contrary to the hypothesized positive relationship:

- Internal Pain Control beliefs and Accommodative beliefs (-0.395, $p<0.0005$)
- Organic beliefs and Worry beliefs (-0.517, $p<0.0005$)
- Organic beliefs and Catastrophizing beliefs (-0.427, $p<0.0005$)
- Organic beliefs and Physical-Activity Fear-Avoidance beliefs (-0.472, $p<0.0005$)
- Organic beliefs and Work-Related Fear-Avoidance beliefs (-0.360, $p<0.0005$)
- Organic beliefs and Pain as a Mystery beliefs (-0.425, $p<0.0005$)

On the other hand, the following significant correlations demonstrated a positive relationship contrary to the hypothesized negative relationship:

- Internal Pain Control beliefs and Worry (0.444, $p<0.0005$)
- Organic beliefs and Accommodative beliefs (0.502, $p<0.0005$)
- Organic beliefs and Acceptance beliefs (0.553, $p<0.0005$)
- Organic beliefs and Pain Self-Efficacy beliefs (0.560, $p<0.0005$)
- External Pain Responsibility beliefs and Accommodative beliefs (0.352, $p<0.0005$)

- External Pain-Responsibility beliefs and Acceptance beliefs (0.378, $p<0.0005$)
- External Pain-Responsibility beliefs and Pain Self-Efficacy beliefs (0.357, $p<0.0005$)
- Pain as a Mystery beliefs and Pain Constancy beliefs (0.373, $p<0.0005$)

These reversed significant correlations are further explained in the discussion chapter.

Structural Model Testing

The Theoretical Model failed according to the χ^2 statistic ($\chi^2 = 255.811$, $df = 76$, $p = 0.0$), the RMSEA index (0.131 with a 90% confidence interval range from 0.113 to 0.149) and according to SRMR (0.134). The Parsimonious Theoretical Model failed according to the χ^2 statistic ($\chi^2 = 342.909$, $df = 82$, $p = 0.0$), the RMSEA index (0.151 with a 90% confidence interval range from 0.134 to 0.168), and according to SRMR (0.184). Contrary to the other models, the Less Restricted Theoretical Model passed according to the χ^2 statistic ($\chi^2 = 60.961$, $df = 48$, $p = 0.0992$), the RMSEA index (0.0430 with a 90% confidence interval range from 0.0 to 0.0740) and according to the SRMR (0.0311). Therefore, the Less Restricted Theoretical Model passed according to all fit indices and none of the other proposed models fit the data. Therefore, the χ^2 difference test was not utilized. However, similar to the sensitivity analysis, all three models resulted in Not Positive Definite Ψ matrices, which was the result of a R^2 value over 1 for Acceptance (indicating that more than 100% of the variance is explained in this latent variable). Examination of the parameter estimates, R^2 values and the standardized solution was completed to further explore the results of the SEM analysis for the Less Restricted Theoretical Model.

Less Restricted Theoretical Model Structural Analysis

Parameter Estimates. To obtain the maximum likelihood parameter estimates 91 iterations were completed. The parameter estimates, standard errors and *Z-scores* are provided below for Beta (β) and Gamma (Γ) matrices (see Tables 8 and 9).

Table 8. Parameter estimates, standard errors and *t-values* for β Matrix.

		1	2	3	4	5	6	7	8	9
1	**Internal Pain Control beliefs**									
2	**Accommodative beliefs**	*0.594* *(0.623)* *0.953*								
3	**Assimilative beliefs**	**-2.013** **(0.613)** **-3.283**								
4	**Acceptance beliefs**	**3.514** **(1.333)** **2.637**	**4.655** **(0.804)** **5.789**	*1.037* *(0.689)* *1.589*						
5	**Pain Self-Efficacy beliefs**	*-0.080* *(0.414)* *-0.194*	*-0.412* *(0.521)* *-0.791*	**0.491** **(0.217)** **2.261**	**0.866** **(0.154)** **5.625**					
6	**Worry beliefs**	**0.676** **(0.145)** **4.649**	**-0.258** **(0.071)** **-3.648**	**0.346** **(0.088)** **3.913**						
7	**Catastrophizing beliefs**	*0.159* *(0.113)* *1.411*	*-0.057* *(0.044)* *-1.302*	**0.152** **(0.064)** **2.366**			**0.285** **(0.097)** **2.928**			
8	**Activity-Related Fear-Avoidance beliefs**	*-0.342* *(0.352)* *-0.972*	**0.559** **(0.244)** **2.294**	*-0.297* *(0.249)* *-1.194*	*-0.118* *(0.124)* *-0.954*	*-0.277* *(0.155)* *-1.782*	*-0.239* *(0.316)* *-0.757*	**1.496** **(0.761)** **1.966**		
9	**Work-Related Fear-Avoidance beliefs**	*-0.859* *(0.626)* *-1.372*	*-0.115* *(0.460)* *-0.251*	*0.668* *(0.468)* *-1.426*	*-0.091* *(0.259)* *-0.350*	*-0.447* *(0.327)* *-1.367*	*-0.921* *(0.674)* *-1.366*	*2.357* *(1.490)* *1.582*		

Table 9. Parameter estimates, standard errors and *t-values* for Γ Matrix.

		Organic	Psychological Influence	Mystery	Pain-Permanence	Pain Constancy	Self-Blame	External Pain Responsibility
1	**Internal Pain Control beliefs**	**-0.261** **(0.121)** **-2.154**	**0.982** **(0.139)** **7.070**	**0.483** **(0.168)** **2.872**	**0.378** **(0.142)** **2.672**	**-0.568** **(0.229)** **-2.483**	**-0.477** **(0.184)** **-2.598**	**-0.347** **(0.169)** **-2.058**
2	**Accommodative beliefs**	**0.760** **(0.241)** **3.150**	*-0.800* *(0.669)* *-1.196*	*-0.495* *(0.389)* *-1.271*	*-0.580* *(0.328)* *-1.769*	*0.046* *(0.508)* *0.090*	*0.295* *(0.387)* *0.763*	**0.818** **(0.319)** **2.567**
3	**Assimilative beliefs**	*-0.302* *(0.252)* *-1.199*	*0.439* *(0.658)* *0.667*	**1.024** **(0.400)** **2.559**	**1.086** **(0.319)** **3.399**	*-0.472* *(0.496)* *-0.950*	*-0.168* *(0.398)* *-0.423*	**-0.833** **(0.347)** **-2.402**
4	**Acceptance beliefs**							
5	**Pain Self-Efficacy beliefs**							
6	**Worry beliefs**							
7	**Catastrophizing beliefs**							
8	**Activity-Related Fear-Avoidance beliefs**							
9	**Work-Related Fear-Avoidance beliefs**							

Many of the parameter estimates are associated with large standard errors (*t*-values between +1.96 and –1.96) which indicate the parameters are not significantly different from zero and fixing it to zero would not make the fit of the model significantly worse (Jöreskog & Sörbom, 2001). Based on the parameter estimates the following parameters could be fixed to zero without negatively influencing the fit of the model (in order of smallest significance to largest—absolute value):

- Pain Constancy beliefs leading to Accommodative beliefs (0.090)
- Internal Pain Control beliefs leading to Pain Self-Efficacy beliefs (-0.194)
- Accommodative beliefs leading to Work-Related Fear-Avoidance beliefs (-0.251)
- Acceptance beliefs leading to Work-Related Fear-Avoidance beliefs (-0.350)
- Self-Blame beliefs leading to Assimilative beliefs (-0.423)
- Psychological Influence beliefs leading to Assimilative beliefs (0.667)
- Worry beliefs leading to Activity-Related Fear-Avoidance beliefs (-0.757)
- Self-Blame beliefs leading to Accommodative beliefs (0.763)
- Accommodative beliefs leading to Pain Self-Efficacy beliefs (-0.791)
- Pain Constancy beliefs leading to Assimilative beliefs (-0.950)
- Internal Pain Control beliefs leading to Accommodative beliefs (0.953)
- Acceptance beliefs leading to Activity-Related Fear-Avoidance beliefs (-0.954)
- Internal Pain Control beliefs leading to Activity-Related Fear-Avoidance beliefs (-0.972)
- Assimilative beliefs leading to Activity-Related Fear-Avoidance beliefs (-1.194)
- Psychological Influence beliefs leading to Accommodative beliefs (-1.196)
- Organic beliefs leading to Assimilative beliefs (-1.199)

- Pain Mystery beliefs leading to Internal Pain Control beliefs (-1.271)
- Accommodative beliefs leading to Catastrophizing beliefs (-1.302)
- Worry beliefs leading to Work-Related Fear-Avoidance beliefs (-1.366)
- Pain Self-Efficacy beliefs leading to Work-Related Fear-Avoidance beliefs (-1.367)
- Internal Pain Control beliefs leading to Work-Related Fear-Avoidance beliefs (-1.372)
- Internal Pain Control beliefs leading to Catastrophizing beliefs (1.411)
- Assimilative beliefs leading to Work-Related Fear-Avoidance beliefs (-1.426)
- Catastrophizing beliefs leading to Work-Related Fear-Avoidance beliefs (1.582)
- Assimilative beliefs leading to Acceptance beliefs (1.589)
- Pain Permanence beliefs leading to Internal Pain Control beliefs (-1.769)
- Pain Self-Efficacy beliefs leading to Activity-Related Fear-Avoidance beliefs (-1.782)

Squared Multiple Correlations for Structural Equations. The Squared Multiple Correlations for the Structural Equations indicate the proportion of explained variance in the latent dependent variables accounted for by the structural equations (see Table 10). As aforementioned, the R^2 value for Y4 (Acceptance) is over the threshold of 1 (indicating that more than 100% of the variance is explained in the respective latent variable).

Table 10. R^2 values for the Structural Equations.

η1	η 2	η 3	η 4	η 5	η 6	η 7	η 8	η 9
0.894	0.798	0.853	1.323	0.866	0.658	0.767	0.737	0.514

Squared Multiple Correlations for Y-variables. These values were fixed according to the reliability studies for each questionnaire. The results indicated the percentage of variance in the dependent indicators attributed to the latent dependent variables rather than measurement error was quite high for all variables (i.e., the indicators all had acceptable reliability) (as seen in Table 11).

Table 11. R^2 values for the Y variables.

Y-Variable	Label	R^2	% of explained variance
Y1	Internal Pain Control beliefs	0.715	71.5
Y2	Accommodative beliefs	0.644	64.4
Y3	Assimilative beliefs	0.704	70.4
Y4	Acceptance beliefs	0.850	85.0
Y5	Pain Self-Efficacy beliefs	0.920	92.0
Y6	Worry beliefs	0.870	87.0
Y7	Catastrophizing beliefs	0.659	65.9
Y8	Activity-Related Fear-Avoidance beliefs	0.575	57.5
Y9	Work-Related Fear-Avoidance beliefs	0.841	84.1

Residuals. "The residuals compared the observed variances and covariances with those resulting from the model's parameter estimates. In a model that fits well, these will be small" (Joreskog & Sorbom, 2001, p. 107). Fitted residuals are in the metric of the indicators and, therefore, examination of the standardized residuals can be more helpful for diagnostics of why the model does not fit well. As well, they "may be helpful for power calculations" (Joreskog & Sorbom, 2001, p. 31). A large number

of the standardized residuals for the Less Restricted Theoretical Model are high, indicating some problems with specification may be present (McMurtry, 2004). However, the high standardized residuals are evenly distributed throughout the matrix and no one variable or group of variables was responsible for the difficulties. The Q-plot of the standardized residuals supports the normal distribution of the residuals but indicate the residuals are more variable than expected.

Standardized Solution. In the standardized solution five significant coefficients exceeded and/or approached 1 (see Tables 12 and 13; the significant parameters are depicted in bold). Recall Heywood cases may be a reflection of identification problem, colinearity, or may be acceptable as regression coefficients. The standardized effects approaching or exceeding 1 were associated with the following chains of influence:

- Internal Pain Control beliefs leading to Assimilative beliefs (-1.336)
- Internal Pain Control beliefs leading to Acceptance beliefs (0.964)
- Accommodative beliefs leading to Acceptance beliefs (1.549)
- Acceptance beliefs leading to Pain Self-Efficacy beliefs (1.099)
- Pain Self-Efficacy beliefs leading to Activity-Related Fear-Avoidance beliefs (-0.911)

Several other standardized coefficients also indicate an effect contrary to hypothesized, an opposite valence relationship (i.e., a positive effect was hypothesized and a negative effect was found or vice versa) (as depicted in italics in Tables 12 and 13).

Table 12. Standardized Solution for β Matrix.

		1	2	3	4	5	6	7	8	9
1	**Internal Pain Control**									
2	**Accommodative**	0.489								
3	**Assimilative**	**-1.336**								
4	**Acceptance**	**0.964**	**1.549**	*0.429*						
5	**Pain Self-Efficacy**	*-0.028*	*-0.174*	***0.257***	**1.099**					
6	**Worry**	***0.744***	**-0.343**	**0.574**						
7	**Catastrophizing**	*0.329*	-0.144	**0.471**			**0.534**			
8	**Activity-Related Fear-Avoidance beliefs**	-0.391	***0.776***	*-0.512*	-0.493	-0.911	*-0.249*	**0.830**		
9	**Work-Related Fear-Avoidance beliefs**	-0.345	-0.056	*-0.404*	-0.132	-0.515	*-0.336*	0.459		

Table 13. Standardized Solution for Γ Matrix.

		Organic	**Psychological Influence**	**Mystery**	**Pain-Permanence**	**Pain Constancy**	**Self-Blame**	**External Pain Responsibility**
1	**Internal Pain Control**	**-0.273**	**0.729**	***0.324***	***0.247***	**-0.300**	**-0.201**	**-0.183**
2	**Accommodative**	***0.655***	*-0.490*	-0.274	-0.311	*0.020*	*0.103*	***0.356***
3	**Assimilative**	*-0.209*	*0.216*	**0.456**	**0.470**	*-0.165*	*-0.047*	***-0.291***
4	**Acceptance**							
5	**Pain Self-Efficacy**							
6	**Worry**							
7	**Catastrophizing**							
8	**Activity-Related Fear-Avoidance beliefs**							
9	**Work-Related Fear-Avoidance beliefs**							

Of these contrary to expected relationships the following were significant:

- Internal Pain Control beliefs and Pain as a Mystery beliefs
- Internal Pain Control beliefs and Pain-Permanence beliefs
- Internal Pain Control beliefs and Worry beliefs
- Accommodative beliefs and Organic beliefs
- Accommodative beliefs and External Pain Responsibility beliefs
- Accommodative beliefs and Activity-Related Fear-Avoidance beliefs
- Assimilative beliefs and External Pain Responsibility beliefs
- Assimilative beliefs and Pain Self-Efficacy beliefs

Summary

The Less Restricted Theoretical Model passed according to χ^2, the RMSEA and the SRMR fit indices. Figure 7 depicts the Less Restricted Theoretical Model with all paths. The dashed paths were insignificant and the bold lines were significant. Interpretation of the findings is limited to those parameters displaying significance because insignificant parameters may be estimated inaccurately in LISREL (including the parameter estimates and standardized coefficients). In Figure 8 only the significant paths are depicted. The dashed paths attenuate and the bold lines strengthen the dependent construct.

Figure 7. Less Restricted Theoretical Model with significant parameters in bold and insignificant parameters in a dashed line.

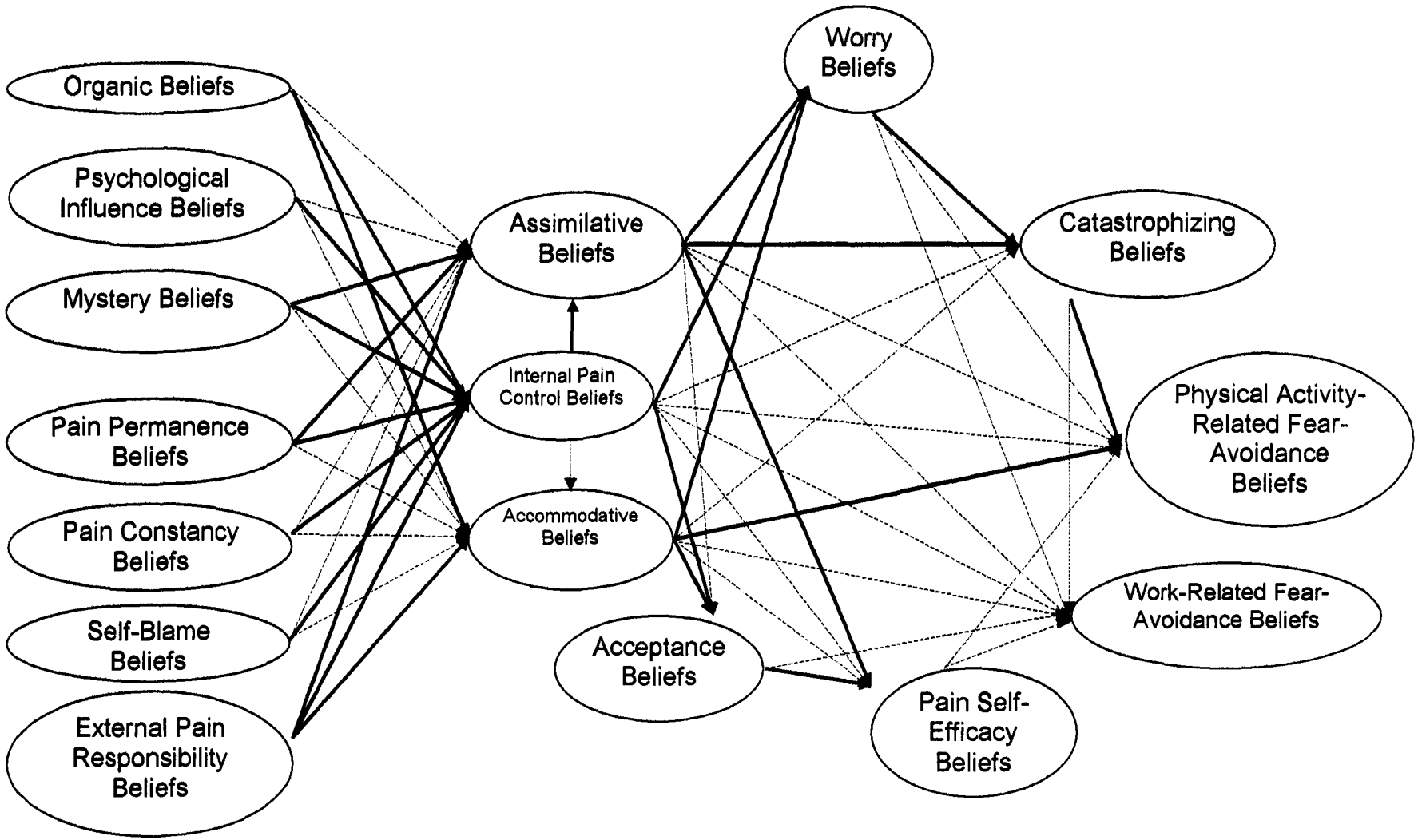
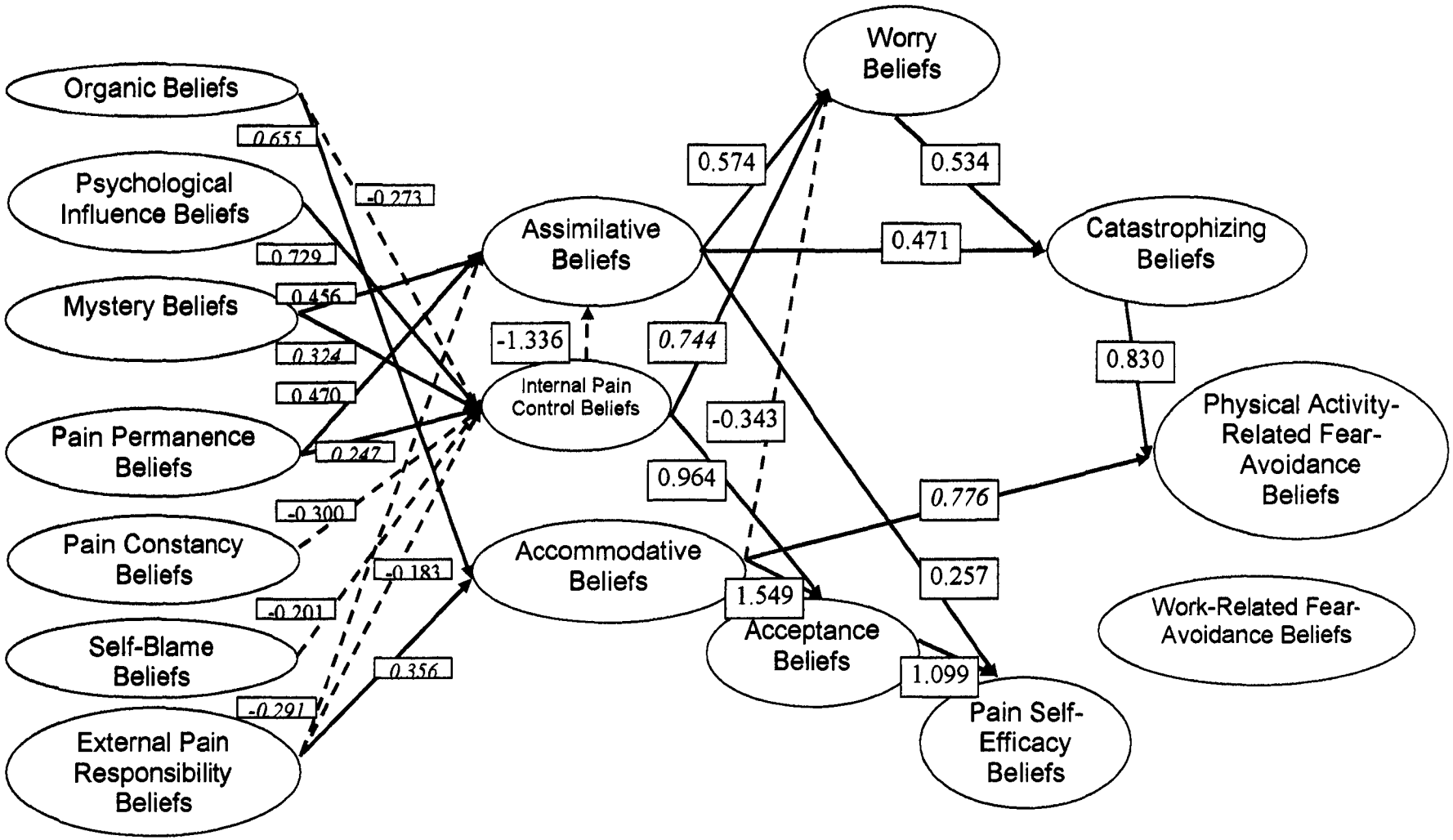


Figure 8. Less Restricted Theoretical Model with only significant paths and their respective standardized coefficients.



Chapter 6: Discussion

The purpose of this research is to bring order to the existing literature on peoples' beliefs about chronic pain and recovery through uniting the chronic pain belief literature into a consensual nomenclature and testing a model of chronic pain beliefs. Overall, the findings are consistent with the proposed nomenclature and support an integrated model of peoples' beliefs about chronic pain and recovery. In this chapter the results of the Less Restricted Theoretical Model are interpreted and discussed along with the clinical implications, the limitations of the analysis and directions for future research.

Interpretation of the Less Restricted Theoretical Model

The proposed nomenclature provides the basis for development of the models. It orients attention to the relevant beliefs, provides the foundational framework for integration of existing models and supports the operationalization of the relevant variables. The colinearity analysis supports the independence of all the exogenous belief variables. As well, independence of all the identified endogenous beliefs is supported through the unique patterns of influence each displayed in the model. Therefore, the results support inclusion of all the identified belief variables as unique contributors to the model. The following discussion focuses on the Less Restricted Theoretical Model specifically examining the structure of the model and the significant chains of influence for the identified belief taxonomies.

Structural Model

The significant structure indicates people possess various beliefs about the threat of chronic pain (i.e., pain beliefs). In line with Philips' (1987) research, all the

pain beliefs were influential. These pain beliefs collaboratively are associated with peoples' perception of personal control over the influence of pain on their lives (i.e., Internal Pain Control beliefs). The results suggest what people believe about the threat of pain are directly associated with their beliefs about personal control over the negative effects of their pain. The implication is people will believe they have less personal control over the negative influence of pain on their life when they perceive the pain as an organic, constant condition that is controlled by others and when they believe they are at fault for the experience of pain. On the other hand, the results imply people will believe they have personal control when pain is viewed as a mysterious, permanent problem that can be influenced by psychological factors.

Consistent with the Fear Avoidance Model, beliefs about the pain problem (i.e., Organicity, Permanence, Constancy and Mystery beliefs) are associated with perceptions of personal control (i.e., Internal Pain Control beliefs). However, contrary to the Fear Avoidance Model where perception of the pain problem (i.e., organicity) is the defining feature of personal control, the results suggest perception of the pain problem is not the sole defining feature of personal control. Rather, the defining feature for Internal Pain Control is the pairing of these perceived pain problem beliefs (i.e., organicity, permanent and/or mysterious) with treatment responsibility beliefs (i.e., Psychological Influence, Self-Blame and External Pain Responsibility). Recall, these three treatment beliefs were proposed to implicate who is responsible for treatment and pain management (Walsh & Radcliffe, 2002). The findings suggest these treatment beliefs also are associated with perceptions of personal control. Specifically, the implication of these findings are that beliefs such as "I am

responsible" (i.e., Psychological Influence beliefs) may exert a positive influence while the beliefs "Powerful others are in control" (i.e., External Pain Responsibility beliefs) and "It is my fault I am in pain" (i.e., Self-Blame beliefs) may exert a negative influence on Internal Pain Control beliefs.

Therefore, interpretation of these results suggests beliefs about the pain problem (i.e., Organicity, Permanence, Constancy, and Mystery beliefs) are associated with perception of personal control. But, these pain problem beliefs function in tandem with beliefs about the controllability of the situation or treatment beliefs (i.e., Psychological Influence, Self-Blame and External Pain Responsibility beliefs). Together these two categories of pain beliefs are associated with perceptions of personal control (i.e., Internal Pain Control beliefs) (McCracken & Eccleston, 2003; McCracken et al., 2004). In turn, all three categories (i.e., pain problem, treatment and personal control beliefs) are associated with peoples' beliefs about how to meet their goals considering the presence of pain (i.e., Goal-Pursuit beliefs).

There are two goal-pursuit reactions; change the situation to maintain the integrity of the goal (i.e., Assimilative beliefs) or change the goals to meet the limitations of the situation (i.e., Accommodative beliefs). The results suggest they are independent and co-occur rather than representing a continuum of decision making. However, they share common chains of influence according to the results. Specifically, the perception of the problem (i.e., Organic, Mystery, Permanence and Constancy beliefs), responsibility for treatment/pain management (i.e., Psychological Influence, Self-Blame and External Pain Responsibility beliefs) and personal control (i.e., Internal Pain Control beliefs) are associated with both goal-pursuit beliefs. In

other words, the results suggest people tend to believe they need to change their goals to meet the limits of the situation when they perceive the problem as organic and believe others are responsible for the management/treatment of their pain. The implication of this result may be that believing a powerful other is responsible for curing the organic problem of pain may increase people's beliefs in the need for their goals to change to meet the situational limitations of chronic pain. On the other hand, the results imply people may tend to believe the situation must be altered rather than their goals when they perceive their pain as a mysterious, permanent condition that neither they nor others can control. Therefore, the results suggest when the influence of others and personal control on the pain is limited and the pain is a mysterious permanent phenomenon then people focus on changing the situation.

These findings suggest the combined effect of these seven pain beliefs, one pain control beliefs and two goal-pursuit beliefs is to either attenuate or intensify an 'adaptive' or 'maladaptive' response. This dual-reaction supports the Fear-Avoidance Model, the Dual Process Model of Coping and Goal-Pursuit theory (Schmitz et al., 1996; Viane et al., 2004; Vlaeyen, Kole-Snijders, Boern, et al., 1995; Vlaeyen, Kole-Snijders, Rotteveel, et al., 1995). The proposed 'adaptive' reaction focuses on Acceptance of the presence of pain and beliefs about self-confidence to perform daily activities despite the pain (i.e., Pain Self-Efficacy beliefs). The proposed 'maladaptive' reaction focuses on ruminative heightened awareness (i.e., Worry), magnification of the negative impact of chronic pain (i.e., Catastrophizing) and fear that physical activity will worsen chronic pain symptoms (i.e., Physical Activity-Related Fear-Avoidance beliefs).

However, the firm distinction between a purely 'adaptive' and 'maladaptive' reaction is not supported: The current study found associations between the 'adaptive' and 'maladaptive' beliefs. For example, believing in personal control over the negative effects of pain (i.e., Internal Pain Control) increased both Acceptance and rumination about pain and its consequences (i.e., Worry). These findings contradict the control literature indicating Internal Pain Control beliefs are not related to Acceptance (Jensen et al., 2001), negatively related to Acceptance (Viane et al., 2004) and not predictive of 'maladaptive' reactions (Wu et al., 2004). The current findings suggest qualification is necessary for the commonly assigned positive influence of personal control on recovery. Specifically, the findings suggest there are times when believing in personal control may increase rumination about the negative consequences of pain.

Reconciling the contrary to expected relationships for Control on 'maladaptive' beliefs is difficult. The contributing factor that provides clarity is the role of goal-pursuit beliefs. With this framework, the results suggest Worry is the result of believing in personal control in a situation they believe cannot be changed but also believe needs to be changed (i.e., Assimilative beliefs). On the other hand, according to the results, Acceptance is a reflection of believing in personal control and goal modification to accommodate to situational limitations that are believed to be beyond their personal control (i.e., Accommodative beliefs). The extant research findings are consistent with this interpretation in that rigidly struggling to control pain in an uncontrollable situation is associated with greater pain, distress and disability (McCracken et al., 2004; McCracken & Eccleston, 2003). Whereas, internal control

beliefs are related to positive outcomes in general (Couglin et al., 2000; Jensen et al., 2001; Miller, 2000).

The results presented thus far support the research indicating the goal-pursuit beliefs are critical in shaping peoples' beliefs about their pain and their recovery. Implication of both 'adaptive' and 'maladaptive' reactions is contrary to Brandtstädter and Rothermund's (2002) proposal that Accommodative beliefs influence adaptive and Assimilative beliefs influence maladaptive. Rather, the results support Schmitz et al.'s (1996) proposal that the goal-pursuit beliefs are independent and interact to either attenuate or intensify the subsequent 'adaptive' or 'maladaptive' chains of influence.

However, the positive influence of Accommodative beliefs on Physical Activity-Related Fear-Avoidance beliefs and the positive influence of Assimilative beliefs on Pain Self-Efficacy are difficult to explain. The question is how can Assimilative beliefs increase both "an exaggerated negative 'mental set'" (Sullivan et al, 2001, p. 53) and confidence to complete tasks despite the pain? As well, how can Accommodative beliefs increase both Acceptance and Fear-Avoidance beliefs?

The answers to these questions may lay in redefining the respective adaptiveness of the identified 'adaptive' and 'maladaptive' beliefs. In line with the literature the results suggest 'maladaptive' beliefs reflect a response to chronic pain that has negative repercussions but are not intrinsically negative (Eccleston et al., 2001; Linton et al., 2000; Sullivan, 2004). Meaning the threat of pain requires problem solving and attempts to solve the problem of pain simultaneously contributes to both negative and positive repercussions (McCracken et al., 2004; McCracken & Eccleston, 2006; Viane et al., 2003).

Therefore, Worry and Catastrophizing may not reflect a pre-existing psychopathology, a deficit in problem solving or incompetence. Rather, Worry may encompasses a heightened awareness and vigilance to threat that is "the natural by-product...of repeated attempts to solve the insoluble problem of chronic pain". The results suggest Worry is linked to perceptions of the pain as uncontrollable and unpredictable. In turn Worry "influences catastrophizing by inflating negatively skewed beliefs that form the basis for catastrophizing" which then in turn increase Physical Activity-Related Fear-Avoidance beliefs.

On the other hand, accepting the presence of pain and having confidence to perform the tasks of daily living does not reflect a more adaptive person with no fear of pain. Rather, Acceptance and Pain Self-Efficacy reflect awareness that the threat of pain is under the control of others paired with perceptions of personal control over the influence of pain and the belief that goals can be modified to offset the negative influence of pain to complete daily activities. But this is accompanied by the belief that over exertion can result in increased pain which is an outcome that is feared and avoided.

In other words, in the current study the results suggest both 'maladaptive' and 'adaptive' reactions share associations and both implicate Fear-Avoidance beliefs. As such, Fear-Avoidance beliefs may not be irrational beliefs with no foundation in reality. Rather they may be specific anticipated outcomes of future harm or outcome expectancies to the threat of pain. Further, supporting the current findings and this interpretation, the literature suggests pain and re-injury expectancies (i.e., Fear-Avoidance beliefs) are a product of one's confidence to perform functional tasks (i.e.,

Pain Self-Efficacy). Although the results of this study fail to support a direct effect between Pain Self-Efficacy and Fear-Avoidance beliefs the results do suggest similar chains of influence (implied through similar associations with modelled a priori variables) and the parameter estimates for the direct paths were near significance.

Summary. Overall the passing model suggests chronic pain is a unique threat to which peoples' beliefs may have positive or negative repercussions. Specifically, the significant results suggest what people believe about their pain and what people believe about the controllability of the situation or treatment is associated with the degree of personal control and perception of goal modifiability. These beliefs then are associated with both 'maladaptive' and 'adaptive' beliefs. Therefore, implying that in order to facilitate recovery all four areas can/should be addressed: (1) Perception of the pain problem, (2) Perception of responsibility for treatment, (3) Perception of personal control over the negative effects of pain and (4) Perception of the extent of goal modification possible/required given situational limitations.

Clinical Implications

The results of this study question the contention that the mere presence of some beliefs hinders recovery and others facilitate. The diametric view of some beliefs as intrinsically 'adaptive' and 'maladaptive' does not capture the gestalt of the chronic pain experience. Rather, the results indicate both reactions result from beliefs about perceived threat, responsibility, personal control and goal modifiability. Clinically this implies facilitation of recovery may be improved through responsively addressing beliefs in all of these four areas. The best practice may be responsive application of interventions focusing on each of the identified beliefs to shift their respective

influence from 'maladaptive' to 'adaptive'. This conclusion represents a diversion from the traditional conceptualization focused on extinguishing or reducing the *presence* of a presumed 'maladaptive' belief. This focus here is on *shifting the pattern of influence* of the identified beliefs.

Thus, believing pain is the result of something organically wrong with the body and the treatment/cure is controlled by chance or powerful others may increase the belief that goals need to change to meet the limitations of the situation. This adaptive approach then decreases Worry and increases Acceptance. However, because the cure is in someone else's hands it is proposed the person has to decide how to effectively change their goals to meet the chronic pain situation. This process is proposed to be through trial-and-error. Through this process one can learn how to live one's life in a manner that does not increase pain: Learn how to have a life *with* the pain. But, with the trial-and-error orientation, one can also learn to fear activities that increase their pain. Specifically, matching goals with the situation provides ample opportunity to learn physical activity or "over doing it" will increase pain or cause a "flare-up".

Clinically, the person with the 'adaptive' reaction is accepting of the presence of pain and therefore it may be helpful to provide tools to facilitate the trial-and-error approach to learning pain management. These tools and strategies may help to reduce the degree of negative learning instances suggested to be associated with fear-avoidance beliefs. Examples include pain awareness training, pacing strategies, specific sleep hygiene strategies, goal-setting, etc.. As well, it may be helpful to

facilitate the determination and communication of realistic expectations given the situational limitations.

On the other hand, the 'maladaptive' chain of influence contains the same four belief areas but some different pain beliefs are implicated and an altered pattern of influence is suggested for the similar beliefs, compared to the 'adaptive' reaction. Specifically, the results suggest people may ruminate (i.e., Worry) about their pain and magnify (i.e., catastrophize about) the negative influence of pain on their lives if they believe their pain is poorly understood, will be part of their future, the treatment/fix is not controlled by powerful others and not under their control. But, if they believe no one can control the pain or the negative effects of the pain then the only way to function is to do activities despite the pain. Therefore, people may believe in their confidence (i.e., Pain Self-Efficacy) to perform the activities of daily living *despite* the pain rather than in *recognition* of the pain. In this situation they feel confident about their abilities but still Worry and Catastrophize about the negative consequences of pain.

Therefore, with the 'maladaptive' approach interventions can focus on facilitating a shift from how to have a life *despite* pain to how to have a life *with* pain. Living despite pain suggests ignoring the influence of pain and function as though it were not present. The subsequent influence increasing Worry, Catastrophizing, Fear-Avoidance and Pain Self-Efficacy beliefs suggest the person feels confident in his/her ability but experiences negative repercussions (may be similar to a negative trial-and-error learning situation). On the other hand, living *with* pain requires acceptance of the situational limitations and the presence of pain and acting accordingly.

But recall the person believes neither he/she nor anyone else can control the pain and the negative effects of the pain on his/her life. As well, the person believes the situation must change to maintain the integrity of his/her goals. The possible implication of this pattern of beliefs, although not examined, is usage of medication or distraction as the primary pain-management tools. Although these tools can be effective the negative belief repercussions suggested in this research implies the efficacy of this approach is suspect.

Therefore, based on this research it is possible helpful interventions may focus on increasing Acceptance of the presence of pain in conjunction with increasing perceived control over the negative effects of pain (i.e., cognitive behavioural therapy focused on personal control and relaxation training focused on reducing physical effects of Worry and Catastrophizing). This may entail grief counselling regarding loss associated with the impact of pain. Further, to reduce the interpersonal costs for people, communication training focused on increasing specificity and prioritizing concerns may be helpful with significant others, family, employers, co-workers and health professionals. Finally, it may be helpful to provide education regarding effective usage of pain medication (i.e., pain managers not 'pain killers').

Limitations of the Analysis

Interpretation of these findings as the definitive model for peoples' beliefs about chronic pain and recovery is premature for several reasons. First, the above discussion is limited to significant parameters. Second, the Less Restricted Theoretical Model lacks parsimony. Third, there is a possibility of equivalent models. Fourth, the fitting model was associated with several interpretation and admissibility concerns

such as, wrong signed coefficients, standardized coefficients over 1 (i.e., Heywood cases) and one variable was associated with an R^2 value of over 1 (η4: Acceptance beliefs).

Insignificant Parameters

Only a portion of the proposed relationships were supported with significant parameters. Some of the mediation restrictions posited in the Less Restricted Theoretical Model obtained insignificant coefficients. Insignificant parameters do not necessarily indicate the parameter is wrong or not relevant but does suggest it may be unstable and estimated inaccurately, in terms of magnitude and/or valence. As well, due to the small sample size in the current study insignificance may indicate irrelevant parameters or attenuation due to lack of power.

Difficulties in interpretation of the insignificant parameters arise because determination of which reason is most plausible is impossible from the results of the current study. But, regardless of the reason, the valence and magnitude indicated are not trustworthy. As a result discussion of the Less Restricted Theoretical Model is limited to significant findings and all insignificant parameters are omitted from the interpretation. But, the insignificant parameters are not discarded without prejudice. Specifically, because each parameter was included based on the literature it is contraindicated to delete the parameters without empirical evidence from an alternative sample. Future research is necessary to conclusively rule out any of the nonsignificant findings in the Less Restricted Theoretical Model. Therefore, all the estimated parameters included in the Less Restricted Theoretical Model should be tested in future replication studies.

Lack of Parsimony

The Less Restricted Theoretical Model was supported according to the goodness of fit indices and χ^2. Although, it is important to note a model with more hypothesized paths will generally, in SEM analysis, obtain better fit results than those with fewer parameters (Martens, 2005). This may suggest the fit of the Less Restricted Theoretical Model (over the Parsimonious and Theoretical Models) may be in part due to the greater number of hypothesized paths. But the Less Restricted Theoretical Model was the *only* fitting model and no determination of 'better' fit needed to be made. It was not a matter of better fit. It was the only model to fit.

Further, the goodness of fit indices chosen and stringent goodness of fit criteria utilized provide support for the veracity of the Less Restricted Theoretical Model. Specifically, the RMSEA tends to prefer more parsimonious models. The fact the RMSEA prefers parsimony yet still obtained the criterion values suggests the Less Restricted Theoretical Model findings are not a statistical artifact of the number of parameters estimated.

Providing additional support of the validity of the Less Restricted Theoretical Model no modification was necessary to make it fit. This supports the importance of and the strength of the solid theoretical substrate for the model; thereby increasing the probability this model can be generalized from this sample to the population of interest, people in the community with chronic pain.

Equivalent Models

The SEM results in conjunction with the literature suggest the model is acceptable. The results also suggest the beginning portion of the model is correct in

implicating four areas of beliefs (i.e., pain problem, responsibility, personal control and goal pursuit beliefs) in the chain of influence for 'adaptive' and 'maladaptive' reactions. But, as aforementioned, there remain some questions regarding the order of the four areas of beliefs identified.

The strength and primary test within The Less Restricted Theoretical Model is regarding the mediation characteristics of Internal Pain Control beliefs in the relationship between the pain beliefs and the goal-pursuit beliefs. Failure of the Theoretical and Parsimonious Theoretical Models in addition to the significant direct effects from various pain beliefs to one or both of the goal-pursuit beliefs contradicts the full mediation of Internal Pain Control on goal-pursuit beliefs (cf., Schmitz et al., 1996). But, Internal Pain Control did demonstrate a direct effect on Assimilative beliefs thus suggesting Internal Pain Control beliefs partially mediate the relationship between pain beliefs and goal-pursuit. It is possible this parameter may be spurious because both Internal Pain Control and Assimilative have two common identical influences in terms of magnitude and valence (Mystery and Permanence beliefs). But, spuriousness is unlikely considering the two pain beliefs are not the sole influence for either Internal Pain Control or Assimilative beliefs. To rule out this explanation further testing of the a priori status of these three taxonomies is necessary.

Admissibility Concerns

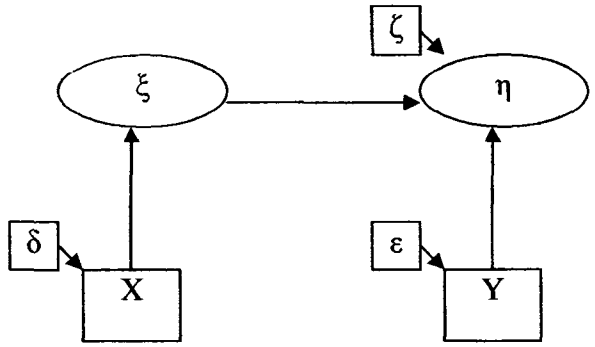
The results of the SEM analysis in the current study reveal some admissibility concerns: Not Positive Definite matrices, R^2 exceeding 1, borderline colinearity, opposite to expected correlations, Heywood cases and insignificant parameters. Admissibility concerns need to be addressed because they may indicate the results are

not trustworthy. To interpret and determine the level of concern the location of the finding, in the context of the specific analysis completed, is critical. Therefore, the admissibility concerns are addressed in the following section and a conclusion about the degree of threat to the findings to the structural model are presented.

Not Positive Definite Matrices and R^2 Exceeding 1. The Not Positive Definite Matrices (impossible covariances) in the sensitivity analysis suggest the error associated with the latent construct of Acceptance was over accounted for. The Ψ matrix is the source for the Not Positive Definite finding suggesting the measurement model is not the source of the problem. Therefore, the source is most likely within the structural model of the latent variables and alternative measurement specifications would not have produced better results (Hayduk, 1987).

However, in the current study single indicators were utilized with the error variance for the manifest variables (δ and ε) set in accordance with externally derived reliability studies. The only portion of the model that is free to vary and demonstrate concerns is the error variance associated with the latent level variables (ζ). Figure 9 demonstrates this restriction. Recall, with single indicators δ and ε are specified according to externally derived reliability studies; ξ is exogenous and specified through the data; η is endogenous and specified through the data; and ζ is free and allowed to vary. ζ is the unaccounted for error variance in the latent variables. We include ζ because no model can reflect the real world perfectly and this inclusion allows for the unidentified influences to be accounted for.

Figure 9. Not Positive Definite.



However, it is unlikely this finding is reflective of a measurement problem though because the Not Positive Definite matrices were present throughout the sensitivity analysis. Two other explanations are possible. First, the admissibility concerns may reflect problems in the structural model for the chain of influence for Acceptance beliefs. The second and more probable explanation is the operationalization of the latent variable of Acceptance encompasses too broad of a construct. Recall the CPAQ full score was utilized which includes both the Activity Engagement and Pain Willingness subscales. It is possible the model fits because these two constructs are related but the admissibility concerns indicate the two subscales have differential influences and have different chains of influence.

The CPAQ is based on factor analytic studies indicating the two subscales contribute to the underlying factor of Acceptance (McCracken et al., 2004). However, if the error terms are not independent for these two subscales (an assumption of factor analysis) it would mean they share common causes. If this were the case then the Not Positive Definite matrices may indicate unique influences on the two subscales. As

well it may indicate use of the broad construct of Acceptance masks the independent influence these two subscales have on other variables in the model.

The operationalization interpretation for the admissibility concerns is supported with other findings in this analysis. It is supported with the R^2 of over 1 indicating the latent variable of Acceptance is overexplained within this model. And, it is supported with the borderline colinearity findings implicating the chain of influence for Acceptance (Accommodative, Acceptance and Pain Self-Efficacy beliefs). Granted the borderline correlations may also suggest Accommodative, Acceptance, and Pain Self-Efficacy beliefs are indicators for a broader construct, are influenced by a common unmodeled variable(s), represent non-recursivity in the model or may simply be an artifact of their endogenous operationalization that encompasses shared chains of influence.

However, these alternative explanations for the borderline colinearity are not theoretically supported. The literature does not imply these variables have similar influences and effects. There is no research to support or suggest a shared unmodeled cause for the three variables. Finally, the literature does not suggest a feedback loop or a non-recursive relationship. The presence of non-recursivity does not align with conceptualization of the 'adaptive' process as outlined in the literature review. There is no empirical evidence to support Pain Self-Efficacy as a predictor of Acceptance or Acceptance as a predictor of Accommodative beliefs. Further, the literature does not suggest any single variable loops whereby the effect of a belief is magnified or attenuated by a function of the presence of the belief.

The most plausible explanation is the correlations between these endogenous constructs are due to shared chains of influence. But given the pattern of findings it is not probable. The Not Positive Definite matrices in the sensitivity analysis and the R^2 findings for Acceptance suggest it is likely operationalization for Acceptance is responsible for the problems rather than measurement or structural problems. The degree of threat for these admissibility concerns is minimal to interpretation of the resulting model. However, further explication of the role of each of the subscales of Acceptance in the model is necessary.

Opposite to Expected Correlations. The opposite valence relationships occurred both within the colinearity analysis and in the structural model and with both exogenous and endogenous variables. This suggests despite displaying adequate fit the data supports the Less Restricted Theoretical Model but does not fully support the foundational hypothesized theory. Specifically, the conceptualization of the dual processes outlined may not be accurate. In other words, the 'adaptive' or 'maladaptive' dichotomy may not capture the complex influences for some of the identified beliefs; specifically in relation to Organic beliefs, Accommodative beliefs, Internal Pain Control beliefs, and External Pain Responsibility beliefs. But, the structural model is not the cause of these findings because the correlation analysis also indicated wrong signed coefficients in the absence of the model restrictions. Therefore, these coefficients do not reflect a problem with the model but rather are valid and suggest the influence of the identified beliefs may not align with theoretical expectations. The results also suggest the opposite valence coefficients represent minimal threat to the validity of the structural model.

Heywood Cases. Recall that Heywood cases may be a reflection of identification problem, colinearity, or may be acceptable as regression coefficients. Generally a standardized effect can exceed 1 (or -1) whenever complimentarily or suppressor relationships occur in the data (e.g., if a negative covariance is obtained between two predictors which have positive regression weights or when one predictor has a negative regression weight and the other one has a positive regression weight and the covariance between the two predictors is positive) (Arbuckle, 1996). With all of the variables involved in the Heywood cases there are both complimentary and suppressor effects involved in the chains of influence and therefore the standardized coefficients exceeding 1 are not necessarily problematic. However, two special cases are found in the results: (1) the relationship between Internal Pain Control and Assimilative beliefs is unexpected and (2) the nonsignificant borderline Heywood case for the relationships between Pain Self-Efficacy and Activity-Related Fear-Avoidance beliefs.

In terms of the relationship between Internal Pain Control and Assimilative it was contrary to expected and both are influenced almost identically by Mystery and Pain Permanence beliefs. These two beliefs demonstrated similar effects in terms of both magnitude and valence. Typically, similar patterns of influence suggest colinearity which is in line with previous research that found people who endorsed both Pain Permanence and Pain as a Mystery beliefs tend to report high levels of Catastrophizing and low levels of internal locus of control (Williams & Keefe, 1991). However, the operational definitions provided in the literature, the factor analytic studies of the parent questionnaire (the PBPI), and the colinearity analyses within the

current study support their independence and suggest Pain Permanence and Mystery beliefs represent different constructs (Dysvik et al., 2004; Herda et al., 1994; Strong et al., 1990; Williams et al., 1994; Williams & Thorn, 1989). Therefore, it is unlikely the similar effects for Permanence and Mystery are the result of colinearity.

Alternative reasons for similarities in chains of influence with independent constructs are available. First, it is possible the two variables are independent indicators for a broader construct. Second, it is possible both are influenced by (an) unidentified variable(s) (i.e., pain intensity, diagnosis, age, gender, etc.) and the similarities in effect reflect the unmodeled mediation of the unidentified variable(s) on Internal Pain Control and/or Assimilative beliefs. Third, the observed similarities reflect spuriousness in identification and, therefore, (an) unidentified variable(s) influence these two beliefs and Internal Pain Control and/or Assimilative beliefs directly. Fourth, both variables are operationalized from the PBPI and the similar effects may be the result of shared measurement error. Finally it is possible that a combination of these situations exists.

Based on the results of the SEM analysis, it is most likely the similar parameters for Mystery and Permanence beliefs reflect spuriousness because both displayed effects in an unexpected direction with Internal Pain Control beliefs, in terms of the valence. Therefore, it is most probable that a third variable influences these pain beliefs, Internal Pain Control beliefs and/or the goal-pursuit beliefs and the relationship between the exogenous and endogenous variables reflect spurious coefficients. But, since Pain as a Mystery and Pain Permanence beliefs both act as

exogenous variables it is beyond the scope of this analysis to determine the plausibility of this interpretation or to attempt to identify the other variable(s).

Further research is necessary to illuminate the reason for the similarities. First, to clarify the independence and chain of influence for Pain as a Mystery beliefs and Pain Permanence beliefs specifically outlining the influence of these two beliefs on Internal Pain Control and goal-pursuit beliefs. Second, future research needs to examine the influence of other variables on these chains of influence. This examination should expand beyond beliefs to incorporate the biological and social elements of the pain experience, in alignment with the Biopsychosocial Model. Finally, research replicating the factor structure for the PBPI is necessary to rule out shared measurement error as the possible culprit. However, it is likely this Heywood case is reflective of spuriousness and does not invalidate the findings for the Less Restricted Theoretical Model.

The second special case refers to borderline Heywood case for the relationship between Pain Self-Efficacy and Activity-Related Fear-Avoidance beliefs. The notable consideration is the insignificant findings for the standardized coefficient value approaching 1 in magnitude. Insignificant parameters are problematic for interpretation because insignificance does not by definition mean the parameter is wrong or irrelevant but rather that the coefficients are unstable and may be inaccurate, in terms of magnitude and/or valence. In the current study, insignificance may indicate irrelevant parameters or attenuation due to lack of power given the relatively small sample size. Either way the characteristics (i.e., magnitude and valence) for the parameter are not trustworthy and are quite possibly inaccurate. Therefore,

interpretation of this relationship as a Heywood case is not appropriate. However, even insignificant parameters influence the fit of a model and, therefore, influence the validity of the Less Restricted Theoretical Model so determination of the level of threat is still important.

The most probable explanation for this finding is attenuation due to lack of power because the relationship was near significance with a *t*-value of -1.782. This is inline with the literature indicating Pain Self-Efficacy influences disability (Altmaier et al., 1993; Asghari & Nicholas, 2001; Buckelew et al., 1994; Denison et al., 2004). But, one of the predictor variables for Pain Self-Efficacy (Accommodative beliefs) directly influences Physical Activity-Related Fear-Avoidance beliefs and therefore this insignificant yet substantial effect may also be reflective of a spurious relationship. Future research with a larger sample is necessary to clarify this relationship. Therefore, this borderline Heywood case does not invalidate the Less Restricted Theoretical Model but it does limit discussions because an insignificant parameter is not included in the discussion regardless of the magnitude because the estimate is not trustworthy.

Directions for Future Research

Based on this study it is suggested that replication of this model in a larger sample is necessary to substantiate the tentative conclusions made based on these results. As well, future research will need to verify and test the Less Restricted Theoretical Model through comparing it with equivalent and/or competing models.

However, prior to replicating the model it would be helpful to examine the nomenclature concerns via the instruments chosen. Although the nomenclature was

substantially supported it is suggested examination of the factor structure for the PBPI be completed to rule out shared measurement error as the possible culprit for similar patterns of influence for Pain as a Mystery beliefs and Pain Permanence beliefs. As well, examination of the operationalization and factor structure of Acceptance beliefs may be beneficial to rule out measurement or construct validity problems as the culprit given the statistical anomalies that occurred around this variable.

After this measurement examination is completed then a replication could be completed. The replication should not discount the insignificant parameters because the sample size for the current study was relatively small and it is possible some of the non-significant parameters may have reached significance with a larger sample. Therefore, prior to making statements against these chains of influence further research is necessary regarding each of the non-significant parameters. The replication should also test a series of models with altered mediational characteristics to rule out equivalent models and allow for examination and clarification of the characteristics of the relationships between the variable identified (e.g., alter the a priori status of Internal Pain Control over the goal-pursuit beliefs to rule out equivalent models).

As well, the samples utilized in the replications should include three groups (a non-chronic pain group, a community sample chronic pain group and a work-related chronic pain group). This is important because the current study had a small work-related sample which may have attenuated the influence of the identified variables on Work-Related Fear-Avoidance Beliefs. As well, this may be beneficial to clarify the presence and chain of influence of these beliefs as a specific response to chronic pain rather than a global fear.

Finally, further research is necessary to clarify the relationships between the variables identified, the influence of other unidentified variables and should also expand beyond beliefs. The following is a summary of suggestions for future research based on the current study:

1. Examination of Mystery, Permanence, Internal Pain Control and Assimilative beliefs for potential spuriousness in identification or to determine the reason for the identical chains of influence.
2. Examination of the mediational characteristics of Internal Pain Control beliefs on the relationship between the pain beliefs and the goal-pursuit beliefs (i.e., further testing the claims of the Dual-Process Model asserting full mediation of control beliefs and a priori status of control beliefs over goal-pursuit beliefs). (e.g., Are Assimilative beliefs mediated differently than Accommodative?)
3. Clarification of the respective influence of the pain beliefs on External Pain Responsibility and Internal Pain Control beliefs specifically focusing on operationally differentiating treatment responsibility beliefs from external control beliefs. (i.e., Are External Pain Responsibility beliefs more accurately modeled as external control beliefs rather than as a pain belief?)
4. The literature suggests that Catastrophizing, which Turk and Rudy (1992) characterized as of central importance in exacerbating pain problems, may be a by-product of patients' confidence in their performance capabilities insofar as anticipated outcome of future harm are outcome expectancies (Bandura, 1978; Lackner et al., 1996). This hypothesis was untested within this study but given the current findings it may be helpful to note if the effects of Pain Self-

Efficacy on Fear-Avoidance beliefs is mediated (fully or partially) through Catastrophizing beliefs in future examinations.

5. Examination of the influence of Pain Self-Efficacy beliefs on Fear-Avoidance beliefs, specifically in relation to pain expectancies and actual disability.
6. Examination of the independence and differential operationalization for the Fear-Avoidance beliefs given that the sample selection may have influenced the findings for Work-Related Fear-Avoidance beliefs.
7. Clarification of the independence and differential operationalization for Fear-Avoidance beliefs and pain expectancies
8. Examination of the independence and comparing the respective influence of depression and Self-Blame on the model.
9. Incorporation of biological and social elements of the pain experience, in alignment with the Biopsychosocial Model (i.e., pain intensity, duration, diagnosis, etc.).
10. Inclusion of variables to examine the impact of an Axis I or Axis II diagnosis on these beliefs to clarify the reality or psychopathology of the perception of threat.
11. Examination and testing the hypothesis regarding the sensitization of threat awareness through inclusion of a comparison sample of people with Anxiety disorders.

Limitations of This Study

Replication should also aim to remedy some of the limitations within the current study which restrict the strength of the conclusions and make suggestions

regarding the findings tenuous. The main limitation was this study was cross-sectional in design which limits conclusions regarding causality and the temporal order for the variables despite the implicit suggestion of order and sequence inherent in the model.

As well, a few measurement limitations are present. First, sending out the questionnaires provided no opportunity to standardized administration procedures which may have compromised reliability of the instruments to an unknown extent. However, allowing participants to complete the questionnaires from home provided an opportunity to utilize a community sample rather than relying on a convenience treatment sample.

Further, this study relied on self-report instruments which may result in response bias, repetitive questions and/or shared method variance which may inflate correlations between constructs. However, no modifications linking the error terms within the manifest level of the model were necessary and therefore it is assumed these concerns were not influential on the results. Further replication would strengthen this assumption.

Finally, this study only utilized subscales and not individual items which means generalizations beyond the subscale level of the instruments is tentative and any suggestion regarding the constructs does not reflect on the individual items included in each subscale. Conclusions beyond the latent level constructs to the individual items for each subscale would require examination with a new sample: It was determined examination of the individual items for each subscale with this sample may result in sample-based modifications to the instruments which may limit generalizability to the chronic pain population.

On a conceptual level, this study utilized several ill-defined constructs that merged many psychological constructs under the umbrella term of 'beliefs'. This study did not aim to remedy, correct, or make judgements against current umbrella usage of the term belief but rather focused on bringing order to the current self-identified belief literature. Therefore, future research may benefit from a more precise definition of what constitutes a belief to further elucidate the core dimensions of peoples' beliefs about chronic pain and recovery. It may also be helpful to delineate the different levels and types of psychological cognitive and affective processes that may be involved. Future research can utilize the literature review and passing model presented in this study to facilitate development and testing of alternative models.

Conclusion

The current research represented an exigent challenge in the creation of a consensual nomenclature and, as well, in the development and testing of the models. Every effort was made to acknowledge and integrate contradictory findings to clarify and elucidate the underlying beliefs structure. The results suggested that the Less Restricted Theoretical Model is supported and it provides a framework for further analysis of the chain of influence for beliefs about chronic pain and recovery. It also provides a framework for clinical intervention. Finally, the results of the current study support the suitability of this synthesis of beliefs into a consensual nomenclature, which future research can expand upon to try to clarify peoples' beliefs about chronic pain and recovery.

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Appendix A

INFORMATION/CONSENT LETTER

PRINCIPAL INVESTIGATORS: Tula Paul, M.Ed., University of Alberta
Derek Truscott, Ph.D., Associate Professor and Supervisor, University of Alberta

STUDY TITLE: Testing a model of the progression of peoples' beliefs about their chronic pain and their recovery

INTRODUCTION

The current research study is conducted by Tula Paul, a Ph.D. candidate of the Department of Educational Psychology, University of Alberta. This research study is conducted as a requirement for the Ph.D. degree, under the supervision of Dr. Derek Truscott, Associate Professor at the University of Alberta.

We invite you to participate in the research study. The purpose of this research is to better understand peoples' beliefs about recovering from their chronic pain. Results of this study will be helpful for providing more effective treatment to help reduce the personal, economic and social costs of chronic pain. Through your participation you will be helping us develop a better understanding of how people experience and recover from the negative effects of chronic pain, thus helping us to develop more effective treatment interventions. In addition it will help to contribute further knowledge in the area for educational purposes for students and future practitioners in the health care professions. The information gathered will be utilized in Tula Paul's doctoral dissertation, as well as in research articles, presentations and for teaching purposes.

You will be asked to complete a paper-and-pencil self-report questionnaire including basic demographic and pain information, as well as questions about your beliefs about your pain and recovery. **Your participation will require about 45 minutes.** Your participation in this study will not affect your medical treatment or your WCB claim. **Whether or not you decide to participate will have no effect on the services you receive.** You are under no obligation to participate. You may withdraw from the study at anytime with no consequences to you. A copy of the final findings can be provided to you at your request upon completion of the project.

Any input you provide for this project will be **confidential**. The identity of participants will not be revealed in any report produced from this study. The information extracted from the questionnaires will be treated confidentially and will be stored in a secure facility in compliance with the University of Alberta Standards for the Protection of Human Research Participants.

If any problems or questions arise with regard to this study, with regards to your rights as a participant in this research, or with regard to the research staff you should contact the principal investigator Tula Paul, M.Ed. (phone: (or e-mail:) or Dr. Derek Truscott of the University of Alberta (phone: (780) or e-mail:). The plan for this study has been reviewed for its adherence to ethical guidelines and approved by the Faculties of Education, Extension and Augustana Research Ethics Board (EEA REB) at the University of Alberta. For questions regarding participant rights and ethical conduct of research, contact the Chair of the EEA REB at

Authorization: I, _____, have read the above and decided to participate in the research project described above.

Signature of Participant: _____ Date: _____

Appendix B

QUESTIONNAIRE

This questionnaire consists of 10 parts. It is very important that all questions are completed. Please read each question carefully and choose the response or number that best answers the question for you. Please note that the answer choices change for each section.

Date: _____

Are you currently involved in a rehabilitation program? Yes No
If yes, _____ Intake _____Discharge (___ weeks on rehabilitation program)

Section A. Demographics

1. Gender: ☐Male ☐Female

2. Date of Birth: _____(mm/dd/yy)

3. Marital Status:
   ☐Single ☐Married ☐Divorced ☐Widowed
   Common-law Separated

4. Highest level of education:
   ☐less than Grade 9 ☐technical/trade school
   ☐partial high school ☐some University
   ☐high school diploma ☐university degree
   ☐partial technical/trade ☐other:_____

5. Current work status:
   ☐full time ☐voluntary
   ☐part time ☐retired
   ☐student ☐unemployed due to pain
   ☐unemployed other reason:_____

6. Living situation: ☐Urban ☐Rural

7. Have you had a head injury? ☐No ☐Yes

8. Do you have cancer? ☐No ☐Yes
   If yes, is it in remission? ☐No ☐Yes

Section B. The next set of questions provides some information about your pain.

9. Date of onset for chronic pain: _____(mm/dd/yy)

10. Cause of pain:

☐Work-related accident	☐Cancer
☐At work-no accident	☐Burn
☐Motor vehicle accident	☐No reason
☐Post-surgery	☐Other:_____

11. Frequency of pain: ☐Constant ☐Intermittent

12. On a scale from 0 to 10, where 0 represents no pain and 10 represents the worst pain imaginable, where is you pain most of the time? Please circle one number.

0 1 2 3 4 5 6 7 8 9 10

13. Pain location: (check all that apply)

☐head, face, neck	☐low back
☐cervical region	☐pelvic region
☐upper shoulder, arms	☐legs
☐thoracic region	☐anal, perineal, genital
☐abdominal region	☐other: _____

Section C. PCS. Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

0 **Not at all**	**1** **To a slight degree**	**2** **To a moderate degree**	**3** **To a great degree**	**4** **All the time**

1.	I worry all the time about whether the pain will end.	0	1	2	3	4
2.	I feel I can't go on.	0	1	2	3	4
3.	It's terrible and I think it's never going to get any better.	0	1	2	3	4
4.	It's awful and I feel that it overwhelms me.	0	1	2	3	4
5.	I feel I can't stand it anymore.	0	1	2	3	4

6.	I become afraid that the pain will get worse.	0	1	2	3	4
7.	I keep thinking of other painful events.	0	1	2	3	4
8.	I anxiously want the pain to go away.	0	1	2	3	4
9.	I can't seem to keep it out of my mind.	0	1	2	3	4
10.	I keep thinking about how much it hurts.	0	1	2	3	4
11.	I keep thinking about how badly I want the pain to stop.	0	1	2	3	4
12.	There's nothing I can do to reduce the intensity of the pain.	0	1	2	3	4
13.	I wonder whether something serious may happen.	0	1	2	3	4

Section D. PBPI. Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following rating scale to make your choices. For instance, if you strongly disagree with a statement you would circle the -2 scale below that statement.

		Strongly Disagree			Strongly Agree
1.	No one's been able to tell me exactly why I'm in pain.	-2	-1	1	2
2.	I used to think my pain was curable but now I'm not so sure.	-2	-1	1	2
3.	There are times when I am pain-free.	-2	-1	1	2
4.	My pain is confusing to me.	-2	-1	1	2
5.	My pain is here to stay.	-2	-1	1	2
6.	I am continuously in pain.	-2	-1	1	2
7.	If I am in pain it is my own fault.	-2	-1	1	2
8.	I don't know enough about my pain.	-2	-1	1	2
9.	My pain is a temporary problem in my life.	-2	-1	1	2
10.	It seems like I wake up with pain and I go to sleep with pain.	-2	-1	1	2
11.	I am the cause of my pain.	-2	-1	1	2
12.	There is a cure for my pain.	-2	-1	1	2
13.	I blame myself if I am in pain.	-2	-1	1	2
14.	I can't figure out why I'm in pain.	-2	-1	1	2
15.	Someday I'll be 100% pain free again.	-2	-1	1	2
16.	My pain varies in intensity but is always with me.	-2	-1	1	2

Section E. PLC. This is a questionnaire to find out how you see the causes and control of your pain. Just rate each statement by the number on the right which best shows how much you currently feel the statement applies to you.

		Very true	True	Untrue	Very untrue
1.	I need my medication to control my pain.	0	1	2	3
2.	My pain will often go away if I let myself relax physically.	0	1	2	3
3.	No matter what I do, I cannot seem to have any effect on my pain.	0	1	2	3
4.	I can make my pain decrease if I concentrate on pain-free parts of my body.	0	1	2	3
5.	I need the help of others to control my pain.	0	1	2	3
6.	I can sometimes reduce pain by imagining the pain is really a pleasant stimulation.	0	1	2	3
7.	Only I can help myself with the pain.	0	1	2	3
8.	My pain level will go down if I remain passive and don't respond to it.	0	1	2	3
9.	My pain professionals can help with my pain.	0	1	2	3
10.	Sometimes I can reduce my pain by not paying attention to it.	0	1	2	3
11.	I am responsible for how the pain affects me.	0	1	2	3
12.	I can make pain go away by believing it will go away.	0	1	2	3
13.	May pain just comes and goes, regardless of what I do or think.	0	1	2	3
14.	My pain will decrease if I think of things going on around me.	0	1	2	3
15.	Being in pain is never my choice.	0	1	2	3
16.	I can reduce pain if I imagine a situation in which I have been pain free in the past.	0	1	2	3
17.	Medication helps me control my pain.	0	1	2	3
18.	My pain will get better if I think of pleasant thoughts.	0	1	2	3
19.	My pain is out of control.	0	1	2	3
20.	Just slowing down and regulating my breathing pattern often helps my pain.	0	1	2	3

Section F. CPIAS. Please indicate the extent to which you agree or disagree with each of the following items.

		Strongly Disagree			Unsure			Strongly Agree
1.	When I'm feeling down or sad my pain usually bothers me more.	0	1	2	3	4	5	6
2.	When I am bothered by daily problems and I feel stressed, my pain is usually worse.	0	1	2	3	4	5	6
3.	The intensity of my pain is affected by how I am feeling emotionally.	0	1	2	3	4	5	6
4.	At the times that my pain is worst, I usually feel helpless and depressed.	0	1	2	3	4	5	6
5.	I can predict when my pain is going to get worse.	0	1	2	3	4	5	6
6.	When I'm feeling excited or when I'm enjoying myself my pain doesn't bother me as much.	0	1	2	3	4	5	6
7.	When I'm too active, I know I'll suffer more later.	0	1	2	3	4	5	6
8.	My pain is affected by changes in the weather.	0	1	2	3	4	5	6
9.	All things considered, I think of myself as able to deal with life's problems and hassles.	0	1	2	3	4	5	6
10.	Despite my pain problem I think I'm quite able to handle my daily affairs and problems.	0	1	2	3	4	5	6
11.	Despite my pain problem, I still feel that I'm in control of my life.	0	1	2	3	4	5	6
12.	Despite my pain problem, I know how to have a good time and lift my spirits.	0	1	2	3	4	5	6
13.	I know that I'll be able to overcome my pain problem and live a satisfactory life.	0	1	2	3	4	5	6
14.	At times I am able to get my mind off my pain.	0	1	2	3	4	5	6

Section G. CPAQ. Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following rating scale to make your choices. For instance, if you believe a statement is 'Always True,' you would circle the 6 on the scale below that statement.

0 Never true	1 Very rarely true	2 Seldom true	3 Sometimes true	4 Often true	5 Almost always true	6 Always true

1.	I am getting on with the business of living no matter what my level of pain.	0	1	2	3	4	5	6
2.	My life is going well, even though I have chronic pain.	0	1	2	3	4	5	6
3.	It's OK to experience pain.	0	1	2	3	4	5	6
4.	I would gladly sacrifice important things in my life to control this pain better.	0	1	2	3	4	5	6
5.	It's not necessary for me to control my pain in order to handle my life.	0	1	2	3	4	5	6
6.	Although things have changed, I am living a normal life despite my chronic pain.	0	1	2	3	4	5	6
7.	I need to concentrate on getting rid of my pain.	0	1	2	3	4	5	6
8.	There are many activities I do when I feel pain.	0	1	2	3	4	5	6
9.	I lead a full life even though I have chronic pain.	0	1	2	3	4	5	6
10.	Controlling pain is less important than any other goals in my life.	0	1	2	3	4	5	6
11.	My thoughts and feelings about pain must change before I can take important steps in my life.	0	1	2	3	4	5	6
12.	Despite the pain, I am now sticking to a certain course in my life.	0	1	2	3	4	5	6
13.	Keeping my pain level under control takes first priority whenever I'm doing something.	0	1	2	3	4	5	6
14.	Before I can make any serious plans, I have to get some control over my pain.	0	1	2	3	4	5	6
15.	When my pain increases, I can still take care of my responsibilities.	0	1	2	3	4	5	6
16.	I will have better control over my life if I can control my negative thoughts about pain.	0	1	2	3	4	5	6
17.	I avoid putting myself in situations where my pain might increase.	0	1	2	3	4	5	6

18.	My worries and fears about what pain will do to me are true.	0	1	2	3	4	5	6
19.	It's a relief to realize that I don't have to change my pain to get on with my life.	0	1	2	3	4	5	6
20.	I have to struggle to do things when I have pain.	0	1	2	3	4	5	6

Section H. PBQ. For each item please indicate your opinion by underlining one of the following words in each sentence: always, almost always, often, sometimes, rarely, never. There are no right or wrong answers; it is important that you respond according to your actual beliefs, not according to how you feel you should believe, or how you think we want you to believe.

1	2	3	4	5	6
Always	Almost always	Often	Sometimes	Rarely	Never

1.	Pain is the result of damage to the tissue of the body.	1	2	3	4	5	6
2.	Physical exercise makes pain worse.	1	2	3	4	5	6
3.	It is impossible to do much for oneself to relieve pain.	1	2	3	4	5	6
4.	Being anxious makes pain worse.	1	2	3	4	5	6
5.	Experiencing pain is a sign that something is wrong with the body.	1	2	3	4	5	6
6.	When relaxed pain is easier to cope with.	1	2	3	4	5	6
7.	Being in pain prevents you from enjoying hobbies and social activities.	1	2	3	4	5	6
8.	The amount of pain is related to the amount of damage.	1	2	3	4	5	6
9.	Thinking about pain makes it worse.	1	2	3	4	5	6
10.	It is impossible to control pain on your own.	1	2	3	4	5	6
11.	Pain is a sign of illness.	1	2	3	4	5	6
12.	Feeling depressed makes pain seem worse.	1	2	3	4	5	6

Section I. FABQ. Please answer these questions concerning the relationship between pain and activities. Please circle the number on the scale that best corresponds to your belief.

		Strongly Disagree			Unsure			Strongly Agree
1.	My pain was caused by physical activity.	0	1	2	3	4	5	6
2.	Physical activity makes my pain worse.	0	1	2	3	4	5	6
3.	Physical activity might be harmful.	0	1	2	3	4	5	6
4.	I should not do physical activity which (might) make my pain worse.	0	1	2	3	4	5	6
5.	I cannot do physical activities which (might) make my pain worse.	0	1	2	3	4	5	6
6.	My pain was caused by my work or by an accident at work.	0	1	2	3	4	5	6
7.	My work aggravated my pain.	0	1	2	3	4	5	6
8.	I have a claim for compensation for my pain.	0	1	2	3	4	5	6
9.	My work is too heavy for me.	0	1	2	3	4	5	6
10.	My work makes or would make my pain worse.	0	1	2	3	4	5	6
11.	My work might harm me.	0	1	2	3	4	5	6
12.	I should not do my normal work with my present pain.	0	1	2	3	4	5	6
13.	I cannot do my normal work with my present pain.	0	1	2	3	4	5	6
14.	I cannot do my normal work til my pain is treated.	0	1	2	3	4	5	6
15.	I do not think that I will be back to my normal work within 3 months.	0	1	2	3	4	5	6
16.	I do not think that I will ever be able to go back to that work.	0	1	2	3	4	5	6

Section J. PSEQ. Please rate how **confident** that you are that you can do the following things **at present** despite the pain. To indicate your answer circle one of the numbers on the scale under each item, where 0 = not at all confident and 6 = completely confident.

		Not at all confident						Completely confident
1.	I can enjoy things, despite the pain.	0	1	2	3	4	5	6
2.	I can do most of the household chores (e.g., tidying up, washing dishes, etc.) despite the pain.	0	1	2	3	4	5	6
3.	I can socialize with my friends or family members as often as I used to do, despite the pain.	0	1	2	3	4	5	6

		Not at all confident						Completely confident
4.	I can cope with my pain in most situations.	0	1	2	3	4	5	6
5.	I can do some form of work, despite the pain. (Work includes housework, paid and unpaid work.)	0	1	2	3	4	5	6
6.	I can still do many of the things I enjoy doing, such as hobbies or leisure activity, despite the pain.	0	1	2	3	4	5	6
7.	I can cope with my pain without medication.	0	1	2	3	4	5	6
8.	I can still accomplish most of my goals in life despite the pain.	0	1	2	3	4	5	6
9.	I can live a normal lifestyle, despite the pain.	0	1	2	3	4	5	6
10.	I can gradually become more active, despite the pain.	0	1	2	3	4	5	6

Thank you for your time!

Please send the completed information/consent letter and the completed questionnaire to the principal investigator, Tula Paul, M.Ed., using the enclosed self-addressed envelope.