AN EMPIRICAL AND THEORY-BASED EVALUATION OF THE FEAR AVOIDANCE MODEL OF PAIN

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June 2012

A thesis submitted to McGill University in partial fulfillment of the requirements of the degree of Doctor of Philosophy

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ABSTRACT

Persistent pain and disability are commonly associated with musculoskeletal injury and can result in considerable personal suffering and societal burden. The Fear Avoidance Model provides a theoretical account of how pain-related disability develops, and has inspired a large body of research that aims to mitigate the negative consequences of musculoskeletal injury. While the Fear Avoidance Model is currently the leading theory of pain-related disability, there are several theoretical and empirical aspects of the model that have yet to be fully addressed; this manuscript-based thesis aims to explore such aspects. This thesis consists of five chapters: a general introduction, three empirical studies and a general discussion. The general introduction provides a broad theoretical context for the three empirical studies. The introduction begins with an historical account of pain-related theories that shaped the development of the Fear Avoidance Model. Next, a detailed discussion of each of the theoretical constructs included in the Fear Avoidance Model is provided. The introduction concludes with a brief review of the clinical applications of the model and by highlighting current holes in the model's supporting literature. The studies included in this thesis aim to address two empirical gaps; the studies evaluate specific prospective relationships proposed by the Fear Avoidance Model and assess alternate relationships among model-relevant variables.

Study 1 provides an analysis of the sequential relationships proposed in the Fear Avoidance Model. Specifically, this study evaluates whether changes in pain catastrophizing precede changes in (1) pain-related fear, (2) depression or (3) pain severity, and whether these changes subsequently influence pain-related disability. Analyses were conducted on a sample of 121 individuals with work-related musculoskeletal injuries and high baseline levels of catastrophizing and pain-related fear. Contrary to the predictions of the Fear Avoidance Model, results from Study 1 failed to support prospective sequential relationships among model-relevant constructs. These findings suggest that model-relevant constructs, such as pain catastrophizing, pain-related fear and depression, may be less inter-dependent than predicted by the Fear Avoidance Model.

Study 2 evaluates the Fear Avoidance Model prediction that pain-related fear acts as a common psychological conduit for multiple pain-related outcomes. This study tests alternate hypotheses by examining whether model-relevant constructs act as differential predictors of pain-related outcomes and whether pain self-efficacy, a construct not addressed in the Fear Avoidance Model, contributes any unique predictive value. The study sample consisted of 202 individuals with subacute, work-related musculoskeletal injuries. Contrary to model predictions, results revealed that pain catastrophizing, pain-related fear and pain self-efficacy had unique relationships with different model-relevant outcomes, and that pain-related fear failed to predict multiple outcomes. These findings complement results from Study 1, and raise concerns about the validity of the inter-relationships proposed within the Fear Avoidance Model.

Results from Study 1 and 2 suggest that model-relevant constructs may interact through alternate relationships that have not been proposed by the Fear Avoidance Model. Study 3 explores one type of alterative relationship by testing whether model-relevant constructs relate to one another in a cumulative fashion. Specifically, this study evaluates whether the number of elevated scores on model-relevant constructs relates to the level of risk for problematic recovery. The study sample, which was the same sample used in Study 2, consisted of 202 individuals with subacute, work-related musculoskeletal injuries. Inconsistent with Fear Avoidance Model relationships, results from Study 3 suggest that model-relevant variables inter-relate in a cumulative fashion.

Considered together, findings from these three empirical studies suggest that the specific inter-relationships proposed within the Fear Avoidance Model may not accurately portray the experiences of people living with musculoskeletal pain conditions. The general discussion provides a detailed exploration of various theoretical assumptions that are made within the Fear Avoidance Model that may help account for the observed lack of empirical support. For instance, the discussion explores how the model's central emphasis on pain-related phobia and avoidance may limit its validity and generalizability; how the model's implied co-presentation of chronic pain and disability make it difficult to explain the

observed variance in these two states; and how the model's failure to integrate pain-related physiological mechanisms is at odds with a large body of biopsychosocial research. The thesis is concluded with a discussion of the potential benefits of exploring new models of pain-related disability.

RÉSUMÉ

La douleur et l'invalidité persistante sont souvent associées avec des blessures musculo-squelettiques et peuvent entrainer une souffrance personnelle majeure ainsi qu'un fardeau social considérable. Le Modèle Cognitivo-Comportemental de la Peur liée à la douleur (MCCP) donne un compte rendu théorique de la manière dont l'invalidité associée à la douleur se développe et a inspiré une grande partie de la recherche qui vise à atténuer les conséquences de blessures musculo-squelettiques. Tandis que le MCCP est la théorie de premier ordre sur l'invalidité associée à la douleur, plusieurs aspects théoriques et empiriques du modèle n'ont pas encore été pleinement adressés. Cette thèse vise à examiner ces aspects. Cette thèse consiste en cinq chapitres : une introduction générale, trois études empiriques et une conclusion générale. L'introduction présente le cadre théorique pour les trois études empiriques. L'introduction débute avec un compte-rendu historique des théories liées à la douleur qui ont influencées le développement du MCCP. Ensuite, une discussion de chacune des constructions théoriques incluses dans le MCCP est donnée. L'introduction se conclut avec une révision des applications cliniques du modèle, et surligne les lacunes actuelles dans la documentation qui appui ce modèle. Les études incluses dans cette thèse visent à combler deux lacunes empiriques; les études évaluent des relations prospectives spécifiques proposées par le MCCP, et examinent les relations alternatives entre les variables pertinents au modèle.

Étude 1 fournit une analyse des relations séquentielles proposées dans le MCCP. Plus précisément, cette étude évalue si des changements dans la catastrophisation de la douleur précèdent les changement dans (1) la peur liée à la douleur, (2) la dépression, ou (3) l'intensité de la douleur, et si ces changements influencent l'invalidité associée à la douleur. Des analyses ont été effectuées sur un échantillon de 121 individus qui ont subi une blessure musculo-squelettiques en milieu de travail et qui ont des taux élevés de catastrophisation et de peur liée à la douleur. Contrairement aux prédictions du MCCP, les résultats de l'étude 1 ne supportent pas les relations séquentielles prospectives parmi les constructions théoriques du modèle. Ces résultats suggèrent que les constructions théoriques du

modèle, tels que la catastrophisation de la douleur, la dépression, pourraient être moins interdépendants que prédit par la MCCP.

Étude 2 évalue la prédiction du MCCP que la peur liée à la douleur agit comme une conduite commune psychologique pour une multitude de conséquences liées à la douleur. Cette étude met à l'épreuve des hypothèse alternatives en examinant si les constructions théoriques liées au modèle agissent comme prédicteurs différentielles des conséquences liées au douleur, et si l'auto-efficacité relié à la douleur, une construction théorique qui n'est pas inclus dans la MCCP, contribue une valeur prédictive unique. L'échantillon de groupe était composé de 202 individus qui ont subi des blessures musculo-squelettiques de phase subaigüe en milieu de travail. Contrairement aux prédictions du modèle, les résultats ont démontré que la catastrophisation de la douleur, la peur liée au douleur et l'auto-efficacité relié à la douleur ont des relations uniques avec différentes conséquences liées au modèle; la peur liée à la douleur n'a pas prédit une variété de résultats. Ces résultats sont cohérents avec ceux observés dans l'étude 1, et soulève des questions de la validité des inter-relations proposées dans le MCCP.

Les résultats des études 1 et 2 suggèrent que les constructions théoriques liées au modèle peuvent interagir par des relations alternatives qui ne sont pas proposées dans le MCCP. Étude 3 examine une sorte de relation alternative en mettant à l'épreuve si les constructions liées au modèle sont reliées d'une manière cumulative. Spécifiquement, cette étude évalue si le nombre de scores élevés sur les constructions théoriques est relié au niveau de risque de récupération problématique. L'échantillon de groupe était composé de 202 individus qui ont subi des blessures musculo-squelettiques de phase subaigüe en milieu de travail. Contrairement aux relations du MCCP, les résultats de l'étude 3 suggèrent que les variables liées au modèle sont inter-reliées d'une manière cumulative.

Considérés dans leur ensemble, les résultats de ces trois études empiriques suggèrent que les inter-relations spécifiques proposées dans le MCCP ne représentent pas avec précision les expériences des individus qui vivent avec des conditions de douleur musculo-squelettiques. La discussion générale fournit une

exploration détaillée de diverses hypothèses théoriques développées dans le MCCP qui peuvent aider à expliquer le manque d'appui empiriques observé dans les trois études. Par exemple, la discussion examine la manière dont l'emphase centrale du modèle sur la phobie liée à la douleur et l'évitement peuvent limiter la validité et la généralisabilité du modèle ; la manière dont la co-présentation de douleur chronique et d'invalidité impliqué dans le modèle font qu'il est difficile d'expliquer les différences observées dans ces deux états ; et de la manière dont l'échec du modèle à intégrer les mécanismes physiologiques liés à la douleur est en opposition avec une grande partie de recherche dans le domaine de la biopsychologie sociale. La thèse se conclut avec une discussion des bénéfices potentiels de remplacer le MCCP avec une théorie de l'invalidité associée à la douleur qui est plus compréhensive.

CONTRIBUTIONS OF AUTHORS

This thesis consists of three multi-authored manuscripts. Mr. Timothy Wideman, the author of this thesis, was the primary author for each of these manuscripts. Study 1 was co-authored by Ms. Heather Adams and Dr. Michael Sullivan, Mr. Wideman's PhD supervisor. Study 2 and Study 3 were co-authored by Dr. Sullivan. As lead author, Mr. Wideman planned and performed all data analyses; he also wrote the manuscripts as well as all letters of response in the peer-review process. Dr. Michael Sullivan provided guidance and advice throughout this process. Dr. Sullivan coordinated data collection for all studies. Ms. Heather Adams assisted with data collection for Study 1.

ACKNOWLEDGEMENTS

I feel honoured to have been supported by so many kind-hearted and thoughtful people throughout my graduate studies. My supervisor, Mick Sullivan, has provided me with an education in research that will long outlast my time spent at McGill. Thank you Mick for facilitating such diverse and rich research opportunities. I truly appreciate your faith in my abilities and your mentorship throughout my studies. Thank you also to other McGill faculty members who provided me with invaluable support and guidance. I am grateful to Maureen Simmonds for shaping my understanding of the rehabilitation field. I am also thankful for the kindness and support that I received from Laura Stone and Richard Koestner; it was hard to not feel a little happier and more confident in my abilities each time we met. Thank you also to my fellow lab members and classmates. You have filled the last five years with fond memories that will certainly last a lifetime.

Thank you to the generous funding organizations that have enabled me to pursue full-time studies. I am particularly grateful for support from FRSQ, CIHR, IRSST and the Physiotherapy Foundation of Canada.

I am also indebted to my patients. You first inspired me to pursue higher education and continue to motivate my learning. You have also humbled me with your grace and perseverance in the face of adversity. It is to each of you that I dedicate this thesis.

Finally, thank you to my friends and family. You are my foundation. Your unconditional love, support and understanding have helped me weather countless academic storms. Thank you Mom and Dad for keeping me focused on what matters most and for teaching me the importance of faith. Lastly, thank you to my loving wife, Megan Bradley. My greatest achievement in life has been marrying you. You make me profoundly happy and have helped me become a better person. Thank you for your endless patience and support throughout my graduate studies. It is a real blessing to share my life with you.

CHAPTER 1: GENERAL INTRODUCTION

Pain is a ubiquitous state that is frequently characterized by intense discomfort and personal suffering. Recent estimations suggest that 20% of adults have experienced pain within the last month and that one out of 12 adults experiences pain on a daily basis (Langley, 2011). Pain is also associated with significant societal burden, particularly in the context of musculoskeletal injuries. For instance, back pain is cited as the fourth most common reason for consulting a healthcare professional, and patients suffering from this condition use twice the amount of healthcare resources than individuals who are pain-free (Goetzel, Hawkins, Ozminkowski, & Wang, 2003; Langley, 2011).

Pain is also enigmatic. It has been alternately characterized as a simple sensory correlate of tissue damage and as a complex manifestation of emotional and cognitive distress. The study of pain is thus an exercise in making sense of the fundamental relationships between the mind and the body. An additional layer of complexity is added by trying to understand how pain relates to disability. For example, severe disability can be paradoxically associated with both high and low levels of pain intensity.

The Fear Avoidance Model is the current leading theory of pain-related disability. This model explains how disability develops among individuals with musculoskeletal pain and, through its various incarnations, has inspired a significant body of research over the past 30 years. The goal of this manuscript-based thesis is to evaluate aspects of this theoretical model that have yet to be fully addressed in the literature. This introduction aims to provide a broad theoretical context for the three empirical studies presented within this thesis. The introduction is divided into three main sections. First, early scientific models that shaped the contemporary understanding of mind-body relationships in the context of pain are reviewed. Second, early theories that related fear, avoidance and pain-related disability are reviewed, and their influence on the contemporary Fear Avoidance Model is explored. Third, a detailed description of the Fear Avoidance Model is provided, with an emphasis on its theoretical constructs and the current state of empirical investigations.

From Descartes to Fordyce: Historical Perspectives on Pain and Mind-Body Relationships

While the Greek and Roman physicians of antiquity documented the importance of pain symptoms in medical practice, the earliest scientific study of pain is commonly attributed to the 17th century French philosopher, René Descartes. Descartes is credited with founding the modern age of intellectual thinking and is commonly known for approaching philosophies of humankind from a dualistic perspective. Descartes believed that humans consist of a body and a mind (or soul)[1], which have distinct compositions (Kenny, 1997). The body, Descartes proposed, is a physical entity that is bound by the rules of mechanics. The mind, on the other hand, is a spirit-like, immaterial entity that is controlled by free will rather than physics. While Descartes delineated these two dimensions of humankind, and commonly discussed them as disparate substances, he also argued that they were intimately united (Kenny, 1997). Descartes' writings on humankind created a paradox that never fully resolved how the material body and immaterial mind could inter-relate (Audi, 1995; Kenny, 1997). Descartes' conception of pain was characterized by a similar mind-body enigma.

Descartes' writings on pain are divergent and are scattered throughout several of his texts, but they can broadly be categorized into three perspectives, which are outlined in his introduction to Treatise of Man:

> "These men will be composed, as we are, of a soul and a body; and I must first separately describe for you the body; then, also separately, the soul; and finally I must show you how these two natures would have to be joined and united to constitute men resembling us." (p. 1; (Descartes, 1664/1972))

Consistent with his approach to describing humankind, Descartes attempted to describe pain from a three-fold perspective: 1) A bottom-up, mechanical description of the bodily movements associated with tissue-damage; 2) A top-

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[1] Descartes used the terms *mind* and *soul* interchangeably; they are similarly treated as synonyms throughout the text.

down explanation of how the mind and emotions influence pain; and 3) A unified portrayal of the pain experience occurring at the mind-body interface.

In the Treatise of Man (Descartes, 1664/1972), Descartes used two sketches to describe the physical dimensions of pain. The first sketch, shown in Figure 1, describes a mechanical response to tissue being heated by fire. In this diagram, heat from the fire triggers a sequential reaction in which threads, that resemble the nervous system, are pulled and a pore in the brain is opened. Animal spirits are released from the open pore, which trigger muscles to withdraw the foot, turn the head, focus the eyes and move the hands, all in an attempt to protect the heated limb from a burn (p. 34-35, (Descartes, 1664/1972)). This diagram and description are commonly used in the pain literature to characterize Descartes' conception of pain (e.g. (Melzack & Wall, 1965; Melzack & Wall, 1996; Waddell, 2004)). Descartes, however, continued his explanation by showing how pain-related information is processed upon reaching the brain (p. 103; (Descartes, 1664/1972)). Descartes used a second sketch, shown in Figure 2, to describe two pain-related pathways in the brain: pathway OR, which is associated with the previously described protective behaviours, and pathway OS, which trigger behaviours that accompany pain-related emotions (p. 103-105; (Descartes, 1664/1972)). Descartes described the latter pathway with the following text:

> "And through the other passage, OS, the spirits enter all those nerves that cause internal emotions like those that pain occasions in us, such as nerves that constrict the heart, agitate the liver, and other such. Through OS they also enter nerves that can cause external movements testifying [to the internal emotions], those for example that provoke tears, or that wrinkle the forehead and cheeks, or that dispose the voice to cry." (p. 106; (Descartes, 1664/1972))

In this text and the related figure, Descartes highlights two key characteristics of his understanding of pain. First, Descartes suggests that pain is processed in the brain. Descartes also emphasized this latter point in Principles of Philosophy by presenting a remarkably progressive case study of a young woman with phantom limb pain. He used this example to explain how the brain can perpetuate the

sensation of pain in the fingers, despite the amputation of the hand and forearm (p. 50-51; (Descartes, 1644/2008)). Processing pain in the brain is an important aspect of Descartes' description, because it permitted a close relationship with the mind, which Descartes also believed to be seated in the brain (specifically in the pineal gland) (Kenny, 1997).

Second, Descartes proposed that pain consists of both physical and emotional qualities, the latter of which were mediated by the mind. Descartes provided more information about the emotional dimension of pain in Passions of the Soul:

> "For the soul is immediately informed of things that harm the body only by the sensation it has of pain, which produces in it first the passion of Sadness, next Hatred of what causes the pain, and in the third place the Desire to get rid of it." (p. 92; (Descartes, 1649/1989))

In addition to emotional reactions to pain, Descartes also described mechanisms by which emotions, and the related brain processes, might moderate the pain experience; referring again to Figure 2, Descartes wrote:

> "Quite similarly, if the action of fire A is intermediate between actions that can conduct the spirits toward R and those that can conduct it toward P, that is, between those causing pain and those causing pleasure, it is easy to understand that it must be the inequalities of the spirits alone that direct them to the one or the other: just as the same action [stimulus] that is agreeable to us when we are in a good humor can often displease us when we are sad and sorrowful." (p. 107; (Descartes, 1664/1972))

Together, these writings have led researchers to conclude that Descartes conceptualized pain as a physical and emotional experience that stemmed from the intersection of the mind and body (p. 73; (Duncan, 2000; Rey, 1995)). For instance, Rey, a scholar in medical history, argued that Descartes believed pain and suffering to be uniquely human experiences. While both animals and humans could experience protective movements that limited tissue damage (as described in relation to Figure 1), only humans had the prerequisite soul to feel the

discomfort and emotion that may accompany such an injury (Rey, 1995). Descartes highlighted this union between the mind and body when he wrote:

> "Nature also teaches me, by these sensations of pain, hunger, thirst and so on, that I am not merely present in my body as a sailor is present in a ship, but that I am very closely joined and, as it were, intermingled with it, so that I and the body form a unit. If this were not so, I, who am nothing but a thinking thing, would not feel pain when the body was hurt, but would perceive the damage purely by the intellect, just as a sailor perceives by sight if anything in his ship is broken." (p. 458-459; (Duncan, 2000), quoting Descartes)

Despite this description, however, Descartes never fully reconciled *how* the mind and body inter-relate to produce pain (Duncan, 2000). In effect, Descartes' perspectives on pain remained fragmented: his bottom-up mechanical description of pain-related behaviours failed to unite with his top-down description of the immaterial mind and emotions. Descartes ultimately failed to achieve his stated goal of explaining how the mind and body are "joined and united", thus leaving an unresolved, dichotomous view of pain – a characterization, that the present author will argue, has yet to be fully reconciled.

While the specific influence of Descartes' writings on subsequent pain theory remains unclear (Duncan, 2000), the leading paradigms throughout the 19th and first half of the 20th century can be roughly construed as extensions of Descartes' bottom-up, mechanical description of pain (Asmundson, Vlaeyen, & Crombez, 2004; Melzack & Wall, 1996; Waddell, 2004). One theory, described as the 'Specificity Model of Pain', proposed a direct, one-to-one relationship between noxious stimuli and perceived pain. Noxious stimuli activated peripheral pain receptors and pain was transmitted, via distinct pathways through the nervous system, to a pain centre in the brain. The clinical influence of the Specificity Model was augmented through advances in physiological research that lent support for certain aspects of the model, such that by the late 19th century it was the leading medical model of pain (Melzack & Wall, 1996).

Despite its increased recognition, however, key findings of the early 20th century led some researchers to question the validity of the Specificity Model of Pain. For instance, findings from the animal studies of Ivan Pavlov suggested that pain-related suffering could be dissociated from noxious stimuli. Similarly, research by Henry Beecher, an American Anesthesiologist who studied soldiers injured on the battlefield of the Second World War, highlighted the influence of meaning and context on pain perception. Both of these observations contradicted the 'straight-through' processing of pain that was proposed by the Specificity Model. In the early 1960's Ronald Melzack, a psychologist from McGill University, and Patrick Wall, a physiologist from MIT, cited this research as evidence that the Specificity Model of Pain was fundamentally flawed. Melzack and Wall argued that the Specificity Model contained a psychological fallacy that equated one's pain experience to the activation of a physiological receptor. A valid pain theory, they argued, needed to account for both sensory *and* psychological factors to adequately explain complex pain conditions such as phantom limb pain or causalgia (i.e. complex regional pain syndrome).

Melzack and Wall aimed to present such a model when they introduced the Gate Control Theory of pain in their seminal 1965, *Science* article (Melzack & Wall, 1965). The Gate Control Theory was the first physiological model of pain that contained a mechanism to account for psychological factors. Melzack and Wall proposed a gating mechanism, shown in Figure 3, that integrated information from both peripheral and central sources (Melzack & Wall, 1965). The gating mechanism was believed to be located in the substantia gelatinosa of the dorsal horn and to receive sensory information via two peripheral sources: small diameter fibers that had an inhibitory effect on the gating mechanism, and large diameter fibers that had the reverse effect on the same mechanism. The gating mechanism was also understood to receive information from the central nervous system, such as cognitive or emotions factors, which could further modulate the sensory system. The net balance of the central and sensory inputs determined whether the gating mechanism conveyed pain signals throughout central nervous and motor systems.

The Gate Control Theory appeared to solve Descartes' 300 year-old mind-body enigma. Unlike previous models, the Gate Control Theory successfully extended Descartes' fledgling bottom-up description of pain physiology to account for the cognitive and emotional factors that he associated with the mind. Thus, the Gate Control Theory provided the "joined and united" perspective of pain that Descartes failed to deliver. The Gate Control Theory revolutionized how pain was conceptualized and was a major catalyst for research in the field. Some researchers, however, believed that this model still failed to resolve key aspects of the pain experience.

During roughly the same period that Melzack and Wall introduced and developed the Gate Control Theory, Wilbert Fordyce advanced and elaborated a fundamentally different conceptualization of chronic pain. While the Gate Control Theory provided a revolutionary conception of how the neurological system processed pain, Fordyce, an American behavioural psychologist, criticized the model for only addressing *internal*, physiological processes and failing to account for *external*, environmental influences (Fordyce, 1976). While Fordyce acknowledged the existence of internal pain processes, he described his behavioural approach to pain as exclusively focused on environmental factors:

> "In behavioral analyses of pain, attention is focused on the relationship between the emission of the pain behaviors and the occurrence of the reinforcing contingencies; no attention is paid to the antecedent events subsumed by the term 'nociception'" (p. 115; (Fordyce, Roberts, & Sternbach, 1985)).

Fordyce reasoned that since the pain experience was wholly subjective, it could never be directly observed or measured by others. Instead of focusing on the experience of pain, Fordyce emphasized the central role of pain behaviour in perpetuating physical and social disability; Fordyce believed that regardless of any unobservable pain-related processes (e.g. nociception or pain-related thoughts and feelings), there could be no pain *problem* in the absence of observable pain behaviour (Fordyce, 1984). Thus, Fordyce's approach to pain can be broadly construed as a top-down account of how environmental factors shape one's pain-

related behaviour, and distinct from the physiological processes delineated in the Gate Control Theory of Pain.

Fordyce's conceptualization of pain behaviour was central to his treatment and research paradigms. Fordyce defined pain behaviour as observable and countable action that is related, at least tangentially, to the pain experience; he delineated two sub-dimensions: respondent and operant pain behaviour (Fordyce, 1976). Respondent pain behaviour was a reflexive response to nociceptive processes, while operant pain behaviour was a learned response to anticipated or contingent social stimuli (Fordyce, 1976). According to Fordyce, acute pain could typically be classified as respondent behaviour, however, chronic pain was almost always characterized as operant behaviour (Fordyce, et al., 1985). In this manner Fordyce suggested that as pain became more chronic, there was an increasing discrepancy between disease processes and illness behaviour.

Fordyce's conceptualization of pain fueled a unique approach to the treatment of patients with chronic pain. The behavioural interventions advanced by Fordyce aimed to reduce disability and pain behaviour, rather than alleviate pain or suffering (Fordyce, 1984). Based on the principles of learning theory, Fordyce proposed three avenues by which pain behaviour could increase and become problematic: 1) direct positive reinforcement of pain behaviour (e.g. praise for pain-contingent rest); 2) indirect positive reinforcement through the avoidance of negative consequences associated with pain (e.g. avoiding physical activity to prevent pain-related distress); and 3) limited positive reinforcement for well behaviours (Fordyce, 1976). The goal of Fordyce's treatments was to replace pain behaviours with well behaviours that were, ideally, mutually exclusive.

Both Melzack and Wall's Gate Control Theory, and Fordyce's behavioural principles of pain were highly influential in shaping subsequent theoretical development in the field. For instance, the Gate Control Theory paved the way for more broad conceptualizations of how the brain modulates the pain experience, while behavioural approaches led to the development of more specialized models of pain-related disability; both paradigms influenced and shaped early fear avoidance models of pain.

"Fear precedes pain and sorrow comes after"[2]

Early Fear Avoidance Models

While themes of fear and suffering have been written about since antiquity, early versions of the contemporary Fear Avoidance Model were first introduced throughout the 1980s and 1990s. Lethem (Lethem, Slade, Troup, & Bentley, 1983), Philips (Philips, 1987) and Waddell (Waddell, 1987; Waddell, Newton, Henderson, Somerville, & Main, 1993) each proposed models that would later influence the theory developed and refined by Vlaeyen and his colleagues (Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995a). While each of these early fear avoidance models linked fear to various aspects of the pain experience, they differed in their content and emphasis.

Lethem et al.'s Fear Avoidance Model of Exaggerated Pain Perception

In 1983, Lethem and his colleagues published a description of their Fear Avoidance Model of Exaggerated Pain Perception (Lethem, et al., 1983). This model attempted to explain the relationship between fear and pain by integrating Fordyce's behavioural principles within the broad conceptual framework of the Gate Control Theory. Lethem's model focused on the inter-relationship between the Gate Control Theory's sensory and emotional dimensions of pain (Melzack, 1975). Lethem and his colleagues proposed that a *normal* pain experience is characterized by an emotional response that is commensurate with the sensory experience (i.e. nociception); emotional distress (governed by psychological processes) heightens or subsides as nociceptive input (governed by physiological processes) increases or decreases, respectively. Some individuals, however, experienced a *desynchronous* relationship between these two dimensions of pain. These individuals experienced increasing levels of emotional distress despite decreasing or stable nociceptive input. This desynchrony resulted in exaggerated pain perception that was beyond what could be explained by the sensory experience alone.

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[2] 4 Maccabees, chapter 1, verse 23; circa 1st century Common Era

Lethem et al. proposed that fear-related coping strategies were influential factors in determining whether the balance between emotional and sensory components of pain would be synchronous or desynchronous (Lethem, et al., 1983). Lethem and his colleagues posited that all patients experience fear in the face of pain and that certain patients confronted this fear while others avoided it. Confrontation was characterized as an adaptive response to fear that facilitated recovery, while avoidance was a maladaptive response to fear that led to exaggerated and prolonged pain perception. Four factors governed the psychological context through which individuals responded to fear, namely: life experiences, pain history, coping strategies, and personality. An avoidant coping strategy was facilitated through (1) stressful life events prior to the pain episode, (2) a history of severe and disabling pain, (3) a passive approach to pain management that focused on rest and analgesics, and (4) personality characteristics that included hypochondria, hysteria and depression. Consistent with Fordyce's research (Fordyce, 1976), avoidance was conceptualized within an operant conditioning framework, thus initial levels of avoidance behaviour would increase if they were negatively rewarded through decreased emotional stress or pain. In this manner, pain and disability could be perpetuated even in the absence of nociceptive input.

Philips' Model of Chronic Pain Avoidance Behaviour

Building on her previous research, Philips concluded that avoidance behaviour was a defining characteristic of patients with chronic pain, and that Melzack's nociceptive and Fordyce's behavioural models could not adequately explain this type of pain behaviour (Philips, 1987). Specifically, she highlighted that previous models failed to address the importance of cognitive factors in perpetuating pain behaviour. Philips addressed this issue by proposing a psychological model of chronic pain which suggested that, in addition to sensory and environmental factors, cognitive processes contributed to the development and maintenance of avoidance behaviour (Philips, 1987). Philips emphasized pain-related expectations, memories, and self-efficacy beliefs as cognitive factors

that influenced avoidance behaviour. While Philips' model did not specifically focus on fear as a precursor to avoidance, she did suggest that exposure interventions, similar to those used in the phobia literature, may be an effective treatment in mitigating avoidant behaviour (Philips, 1987). In sum, Philips' avoidance model recognized the influence of nociceptive and behavioural factors, but emphasized the central role of cognitive processes in chronic pain behaviour. In contrast to Lethem's model (Lethem, et al., 1983), Philips' focus was on avoidance rather than pain perception. Philips also suggested that fear was not the sole precursor of avoidance behaviour.

Waddell's Fear Avoidance Research and Biopsychosocial Model of Back Pain

Building on the work of Lethem (Lethem, et al., 1983) and Philips (Philips, 1987), Waddell added specificity to the fear construct by developing sub-dimensions for fear of physical activity and fear of work activity (Waddell, et al., 1993). Waddell and his colleagues argued that, in addition to fear of pain, fear of different activities contributed to pain-related disability. His research in this area focused on the development of a fear avoidance measurement tool and on the integration of fear of activity into early avoidance models. Prior to this work, Waddell developed a biopsychosocial model of low back pain, which served as the broad conceptual framework for his research on fear avoidance (Waddell, 1987). In addition to behavioural and cognitive factors, Waddell stressed the importance of nociceptive processes in precipitating *and* perpetuating chronic pain and disability. For example, in his seminal paper exploring fear avoidance constructs Waddell and his colleagues used the following text to emphasize the biological influences on chronic pain:

> "Chronic pain is sometimes described as persisting beyond normal healing time: if there is no longer any evidence of tissue damage it is sometimes implied that there is no remaining nociception. This would incorrectly imply that there is no longer any sensory component to the pain. This is

> neither theoretically nor clinically acceptable." (p. 165; (Waddell, et al., 1993))

In this manner, Waddell advanced a model of fear avoidance that equally emphasized underlying biological, psychological, and social factors.

"*Pain-related fear is more disabling than pain itself*"[3]

Vlaeyen et al.'s Fear Avoidance Model of Pain

The Cognitive-Behavioral Model of Fear of Movement/(Re-)Injury

In 1995 Vlaeyen and his colleagues (Vlaeyen, et al., 1995a) presented The Cognitive-Behavioral Model of Fear of Movement/(Re-)Injury that addressed the principal factors and relationships that are included in contemporary versions of the Fear Avoidance Model; a graphical depiction of Vlaeyen's original model is shown in Figure 4. This model aimed to explain prolonged pain and pain-related disability through a series of sequentially related cognitive, affective, and behavioural factors. Vlaeyen and his colleagues proposed that most individuals who experienced increased pain during physical activity responded with adaptive appraisals (i.e. non-threatening appraisals of pain-related stimuli) that facilitated confrontation and recovery. Some individuals, however, catastrophically interpreted such pain experiences. These maladaptive appraisals precipitated a downward spiral that was characterized by increased levels of fear and avoidance. With time, avoidance of pain-related movements led to increased levels of disuse, disability, and depression, which in turn fed back into increased levels of pain and catastrophic thoughts.

Relationship Between Early and Contemporary Fear Avoidance Models

Vlaeyen's model both developed and disregarded different elements of early fear avoidance models. Directly related to previous work by Lethem (Lethem, et al., 1983), Philips (Philips, 1987), and Waddell (Waddell, et al., 1993) is Vlaeyen's central focus on confrontation and avoidance and the preceding pain-related cognitive and affective variables (Vlaeyen, et al., 1995a; Vlaeyen &

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[3] (Crombez, Vlaeyen, Heuts, & Lysens, 1999)

Linton, 2000). Vlaeyen extended this research by further delineating the mechanisms and consequences of avoidance behaviour. For instance, Vlaeyen added specificity to the psychological context through which avoidance occurred by incorporating constructs of pain catastrophizing and fear of movement/(re-) injury into his model (Vlaeyen, et al., 1995a). Vlaeyen's model also introduced the *disuse syndrome* construct as a conceptual link between avoidance behaviour and prolonged pain-related disability.

Unlike early fear avoidance models, however, Vlaeyen's did not address the potential influence of nociceptive or biological processes. Consistent with Fordyce's principles of avoidance learning (Fordyce, 1976), Vlaeyen suggested that in chronic cases, disability was independent from organic pathology, nociceptive processes, and pain intensity (Vlaeyen, et al., 1995a; Vlaeyen, Kole-Snijders, Rotteveel, Ruesink, & Heuts, 1995b). While each of the early fear avoidance models addressed, and at least partially integrated, the principles of behaviourism *and* Gate Control Theory, Vlaeyen et al.'s model (Leeuw, et al., 2007a; Vlaeyen, et al., 1995a; Vlaeyen & Linton, 2000; Vlaeyen & Linton, 2012) failed to incorporate research related to the latter theory. In this manner, the contemporary Fear Avoidance Model can be broadly framed as a cognitive-behavioural, rather than a biopsychosocial, model of pain-related disability.

Overview of Amendments to the Fear Avoidance Model

Since its first introduction in 1995 (Vlaeyen, et al., 1995a; Vlaeyen, et al., 1995b), Vlaeyen and his colleagues have published three journal articles and one edited collection focusing on reviewing and refining the Fear Avoidance Model. In 2000, Vlaeyen and Linton published the first comprehensive review of the fear avoidance literature and included a slightly revised version of the 1995 model (Vlaeyen & Linton, 2000). Building on his earlier work, the amended model introduced three new factors, namely *negative affectivity*, *threatening illness information,* and *hypervigilance*; the revised model is shown in Figure 5.

In 2004, more extensive revisions to the model were published in a book edited by Asmundson and his colleagues (Asmundson, et al., 2004). This version

of the Fear Avoidance Model introduced several new variables and relationships, but also eliminated some of the key elements included in previous models. In general, this model integrated Asmundson's previous work on fear-related physiological arousal (Norton & Asmundson, 2003) with Vlaeyen and Linton's 2000 model (Vlaeyen & Linton, 2000); a graphical depiction of this model is shown in Figure 6. Compared to previous versions, this model is much more specific. For instance, pain-related fear was distinguished from anxiety, and both constructs were further characterized by cognitive, behavioural, and physiological dimensions. However, several of the amendments presented in the 2004 model were at odds with Vlaeyen's earlier models. For instance, the 2004 model did not address depression or disability, and added bi-directional relationships that directly linked pain perception and disuse/de-conditioning.

A more recent presentation of Vlaeyen's Fear Avoidance Model was published in 2007 (Leeuw, et al., 2007a). This model was virtually identical to Vlaeyen and Linton's 2000 model (Vlaeyen & Linton, 2000), with the addition of Asmundson's multidimensional constructs of fear and anxiety (Asmundson, et al., 2004). The constructs of disability and depression were reintroduced to the model and the direct relationship linking pain and disuse was eliminated; the 2007 model is shown in Figure 7.

Vlaeyen and Linton's most recent review of the Fear Avoidance Model was published in 2012 (Vlaeyen & Linton, 2012). While this review introduced several new theoretical mechanisms for the acquisition of pain-related fears, the graphical depiction of the model was identical to the figure used in the 2000 review (see Figure 5). The following text provides a detailed description of the theoretical constructs and processes that have been included, and maintained, in Vlaeyen et al.'s Fear Avoidance Model.

Theoretical Constructs of the Fear Avoidance Model

Pain-Related Fear and Anxiety

The conceptualization of fear within the Fear Avoidance Model has undergone significant development since Vlaeyen's original model. In his first

model, Vlaeyen and his colleagues introduced the construct as *fear of movement/(re-) injury*, which they regarded as a synonym for the term *kinesiophobia*. Kinesiophobia was defined as "an excessive, irrational, and debilitating fear of physical movement and activity resulting from a feeling of vulnerability to painful injury or reinjury" (Vlaeyen, et al., 1995a).[4] Consistent with this definition, Vlaeyen contextualized the fear of movement/(re-) injury construct within the phobia literature by proposing 1) conceptual links with phobic complaints, such as fear of bodily injury, illness and death and 2) phobia interventions, such as graded exposure, for individuals with elevated levels of fear (Vlaeyen, et al., 1995a; Vlaeyen, et al., 1995b). In more recent work on the model, Vlaeyen and his colleagues label the fear construct as either pain-related fear (Vlaeyen & Linton, 2000) or fear of pain (Leeuw, et al., 2007a); the three terms are frequently used synonymously.

The most significant amendments to the conceptualization of fear within the Fear Avoidance Model stemmed from research conducted by Asmundson and his colleagues. These amendments include, 1) anchoring the fear construct within a three-factor model of fear and 2) delineating fear and anxiety constructs. The former amendments were a direct extension of prior work by Lang et al (Lang, 1968). Lang advanced a three-factor model of fear that was designed to facilitate the assessment and treatment of phobic disorders (Lang, 1968). Lang's model suggested that the fear response was characterized by three partially overlapping and mutually reinforcing dimensions, namely verbal (or cognitive), motivational (or behavioural), and autonomic (or physiological) factors (Lang, 1968). Lang proposed that the cognitive component of the fear response was characterized by attention to fear-related stimuli, appraising fear-related coping resources, and creation of escape/avoidance action plans. The behavioural dimension consisted of escape from, or avoidance of, fear-related stimuli. Finally, the physiological

[4] While Vlaeyen credits Kori, Miller and Todd with first coining and developing the construct of kinesiophobia, early work on the development of this construct is not widely available. Vlaeyen's references to this work included an unpublished doctoral thesis and a conference abstract. Moreover, the seminal paper published by Kori et al., in 1990 (which is frequently cited in literature as a reference for the development of the Tampa Scale of Kinesiophobia) could not be acquired by searching online databases or several university libraries, or through personal requests to key authors.

component of the fear response was characterized by autonomic changes associated with the fight-or-flight response (e.g. increased muscle activation, heart rate, and respiratory rate). Lang argued that a comprehensive assessment should address all three dimensions of the fear response (Lang, 1968).

The second major modification to the conceptualization of fear focused on distinguishing anxiety from fear. Fear implies a response to an immediate threat that permits *escape*, rather than *avoidance* behaviour. Anxiety, on the other hand, relates to a threat that may occur in the future and, therefore, may be logically associated with avoidance behaviour. In an effort to more accurately reflect these conceptual distinctions, the 2004 Fear Avoidance Model discussed pain-related fear and anxiety as separate constructs, and related these constructs to avoidance and escape behaviour, respectively (Asmundson, et al., 2004). In this model, both pain-related fear and anxiety were couched in the three-factor response model previously proposed by Asmundson and his colleagues (Norton & Asmundson, 2003). Specifically, fear was characterized by defensive motivation, threat perception and arousal, while anxiety was characterized by preventative motivation, hypervigilance, and arousal. While these amendments to the fear construct were included in the 2007 review of the Fear Avoidance Model (Leeuw, et al., 2007a), their practical importance was qualified. Specifically, the model addressed the conceptual distinction between fear and anxiety, but conceded that the clinical differentiation between these two factors is likely unfeasible and is expected to have little functional importance (Leeuw, et al., 2007a).

Pain Catastrophizing, Negative Affectivity, and Anxiety Sensitivity

The Fear Avoidance Model proposes that negative, pain-related, cognitive processes precipitate fear and anxiety. In Vlaeyen's original model, the pain catastrophizing construct represented these cognitive processes (Vlaeyen, et al., 1995a). In later models, additional constructs, such as negative affectivity and anxiety sensitivity, were introduced to further describe the context through which pain-related thoughts and feelings developed (Asmundson, et al., 2004; Leeuw, et

al., 2007a; Vlaeyen & Linton, 2000). Below is a description of each construct and their proposed inter-relations.

Within the context of the Fear Avoidance Model, pain catastrophizing is defined as an attentional bias toward the negative aspects of pain-related stimuli (Vlaeyen, et al., 1995a). Individuals high in levels of pain catastrophizing are more likely to notice movement-related sensations and to interpret these sensations as threatening (Vlaeyen, et al., 1995a; Vlaeyen, et al., 1995b). While Vlaeyen's original model graphically portrayed catastrophizing as a causal precursor to fear (see Figure 4), this depiction was qualified in the text by suggesting that the reverse relationship could also be possible (Vlaeyen, et al., 1995a). In later models, however, pain catastrophizing has been consistently presented as a causal precursor to pain-related fear (Asmundson, et al., 2004; Leeuw, et al., 2007a; Vlaeyen & Linton, 2000).

In 2000, negative affectivity and anxiety sensitivity were added to the Fear Avoidance Model as constructs that overlapped with pain catastrophizing (Vlaeyen & Linton, 2000). Building on previous work by Watson and Pennebaker (Watson & Pennebaker, 1989), negative affectivity was described as a stable trait that predisposed individuals to have an overly negative view of the world and of themselves. Individuals with high levels of negative affectivity were more inclined to experience worrisome thoughts, anxiety, and self-criticism. In the context of the Fear Avoidance Model, negative affectivity was described as a moderator of fear; the model posited that high levels of negative affectivity led to hypervigilance toward threatening stimuli, which led to increased fear (Vlaeyen & Linton, 2000). The closely related construct of anxiety sensitivity was defined as a personality characteristic that predisposed individuals to respond to signs of their anxiety with increased anxiousness (i.e. fear of being fearful) (Vlaeyen & Linton, 2000). Anxiety sensitivity was characterized as a more basic form of fear, from which specific fears, such as fear of movement or re-injury, could emerge.

While Vlaeyen and his colleagues proposed a close relationship between anxiety sensitivity, negative affectivity, and pain catastrophizing, these inter-relationships were only vaguely defined. For instance, despite delineating each of

these constructs in the text of their 2000 review, only the latter two variables were included in the graphic of the model (Vlaeyen & Linton, 2000). Also, a third term entitled, *threatening illness information*, is included in the graphic, but not the text; it is unclear whether this term is intended to represent the anxiety sensitivity construct or perhaps one of its perceptual consequences. Moreover, while Vlaeyen and his colleagues described the inter-relation between these factors as overlapping, the graphic suggests that negative affectivity is a causal precursor to pain catastrophizing, which, in turn, leads to pain-related fear (Vlaeyen & Linton, 2000). In their 2004 book chapter, Keogh and Asmundson provide a more detailed portrayal of these factors (Keogh & Asmundson, 2004). Here, the variables, amongst others, are portrayed in a hierarchy: negative affectivity is the most general, followed by anxiety sensitivity, which is followed by pain catastrophizing. While this hierarchy is not explicitly addressed in other model-relevant texts, the Fear Avoidance Model can be broadly understood to portray pain catastrophizing as a cognitive precursor to fear, which is grounded in more general forms of anxiety and negative personality traits.

Fear-Related Attentional Biases and Hypervigilance

The original Fear Avoidance Model addressed fear-related attentional biases in the context of pain catastrophizing and did not include a specific hypervigilance construct (Vlaeyen, et al., 1995a). However, in response to work by Eccleston, Crombez and, later, Van Damme (Crombez, Van Damme, & Eccleston, 2005; Eccleston & Crombez, 1999) the importance of attentional biases in the context of fear and avoidance became more apparent and were incorporated in the first review of the model. Hypervigilance was defined as a bias to attend to threat-related stimuli over neutral stimuli and was understood to be directly related to elevated levels of pain-related fear (Vlaeyen & Linton, 2000).

In their 2004 chapter on the Fear Avoidance Model, Van Damme and his colleagues described the processes through which fear-related thoughts and feelings influence attentional processes (Van Damme, Crombez, Eccleston, &

Roelofs, 2004). Normal attentional biases toward pain are conceptualized as an evolutionary mechanism that facilitates survival. The degree to which pain-related stimuli interrupts one's attention is not only linked to its sensory characteristics, but also to its perceived threat-value and environmental context. For instance, pain-related stimuli that are novel and unexpected are more likely to solicit one's attention. Catastrophic thinking augments the attentional bias associated with pain-related stimuli by increasing its perceived threat value. Environmental settings with low stimulation (as can occur when individuals are work-disabled) can also further increase the attentional bias on pain by not providing competitive stimuli that is sufficiently demanding. Van Damme and his colleagues describe these factors as contributing to the hypervigilance observed amongst individuals with elevated levels of fear (Van Damme, et al., 2004).

Fear Avoidance Learning

The Fear Avoidance Model links pain-related fear to avoidance behaviour through avoidance learning. The model incorporates classical and operant conditioning paradigms with cognitive processes to explain the acquisition of fear avoidance behaviour (Vlaeyen & Linton, 2000). From a classical conditioning perspective, pain is considered an innate unconditioned stimulus to which various conditioned stimuli can become associated (Vlaeyen & Linton, 2000). For instance, if back pain first developed during a bending activity, then bending may become a conditioned stimulus for pain and is, therefore, avoided. As pain becomes chronic, operant conditioning may become more influential (Vlaeyen & Linton, 2000). With time, for example, the conditioned stimulus of bending may become closely association with pain and suffering, such that bending movements provoke a phobic response. Thus, when bending is avoided, fear and distress are also avoided, and the positive feelings that accompany escape from a perceived threat reinforce the avoidance behaviour. Cognitive factors can further augment these behavioural processes. For instance, negative expectations, attention, and fear avoidance beliefs can all increase the perceived threat value of pain-related stimuli and further reinforce avoidance behaviour.

In a recent review of the Fear Avoidance Model, Vlaeyen and Linton added further theoretical nuance to the acquisition of avoidance behaviour (Vlaeyen & Linton, 2012). First, Vlaeyen and Linton proposed that different types of pain-related stimuli influence avoidance learning. Specifically, they hypothesized that while both exteroceptive (e.g. audio or visual stimuli) and interoceptive (e.g. proprioceptive stimuli) forms of stimuli are involved in avoidance learning, unpredictable, proprioceptive stimuli are likely the most influential. Second, they highlighted different mediums through which avoidance learning may occur. Consistent with social learning theory, Vlaeyen and Linton proposed overlapping relationships between direct experience, verbal instruction, and social observation. Third, they proposed that goal conflicts might also influence the expression of avoidance behaviour. For instance, while avoidance behaviour may be reinforced through escape from a threat, it may also be punished by not achieving a personal goal, such as not returning to work or becoming more physically active.

Disuse, Depression and Disability

Disuse syndrome is a crucial component of the Fear Avoidance Model that links avoidance behaviour to disability and depression outcomes. First coined by Bortz (Bortz, 1984), disuse syndrome is a broadly conceived, adverse condition that results from prolonged inactivity. The disuse syndrome was not originally linked to pain-specific conditions – indeed, the word *pain* was not used in Bortz's seminal description (Bortz, 1984) – but rather was presented as the general health implications of an increasingly sedentary Western culture. Citing a wide range of correlational studies, Bortz argued that disuse leads to numerous adverse conditions, including decreased cardiovascular function, obesity, musculoskeletal fragility, premature aging, and depression (Bortz, 1984). In contextualizing the syndrome within the Fear Avoidance Model, Vlaeyen and his colleagues focused on the effects of disuse on musculoskeletal and cardiovascular systems (Vlaeyen, et al., 1995a; Vlaeyen & Linton, 2000). They suggested that prolonged avoidance led to muscle atrophy and reduced fitness levels, which caused increased levels of

physical disability. Vlaeyen and his colleagues also emphasized the psychological consequences of disuse, suggesting that inactivity is associated with increased depression. In turn, depression is associated with reduced levels of pain tolerance, thereby feeding back into the disabling fear avoidance cycle (Vlaeyen, et al., 1995a).

Pain Experience and Injury

The final constructs to be addressed, *pain experience* and *injury,* are two of the least developed within the fear avoidance pathway. In general, the theoretical and clinical importance of injury, or tissue damage, is downplayed within the model. Injury, presumed to cause acute tissue damage, initiates the fear avoidance pathway, but is not a factor in perpetuating the downward spiral. The implied argument is that once pain conditions become chronic, any tissue changes that may have been associated with acute pain have returned to normal (Vlaeyen, et al., 1995a).

Consistent with the approach taken by Fordyce, the model does not address the role of nociceptive or physiological processes in the pain experience (Vlaeyen, et al., 1995a). Similarly, pain intensity is regarded as a weak factor in perpetuating disability. For example, in Vlaeyen and Linton's first review of the model they highlight literature supporting the conclusion that fear is more disabling than pain itself (Vlaeyen & Linton, 2000). When pain experience is addressed within model-relevant texts, emphasis is often placed on the perception and meaning of pain, rather than its intensity or sensory qualities (Vlaeyen, et al., 1995a; Vlaeyen & Linton, 2000).

Clinical Applications of the Fear Avoidance Model

The primary clinical application of the Fear Avoidance Model has focused on measuring and targeting pain-related fear and its model-relevant correlates. Vlaeyen and Linton called for a three-pronged approach to address pain-related fear in clinical settings (Vlaeyen & Linton, 2000). First, brief self-report questionnaires, such as the Tampa Scale for Kinesiophobia or the Fear Avoidance

Beliefs Questionnaire, are recommended for screening patients for elevated levels of pain-related fear. Second, they advise that patients with elevated levels of fear should be reassured that pain is not dangerous, educated about the disabling, fear avoidance cycle, and instructed to resume all normal physical activities. Third, graded exposure interventions are proposed for reducing levels of pain-related fear.

Originally developed as a behavioural treatment for non-pain-related phobias, graded exposure interventions have been used within the context of the Fear Avoidance Model to both reduce levels of fear and to progressively increase participation in fear-related physical activities (Leeuw, et al., 2007a). Exposure is conducted in a step-wise fashion (Vlaeyen & Linton, 2000). First, patients are asked to generate a ranked list of fear-related physical activities, in ascending order from least to most feared activity. Next, patients are asked to describe the anticipated consequences of performing the least feared activity. After performing this activity, patients are again asked to rate the level of fear associated with the physical activity and to reflect on its actual pain-related consequences. This process is repeated until levels of fear associated with the activity performed are decreased, after which exposure to the next activity on the ranked list is initiated. Progressive exposure to feared activities is designed to explicitly challenge patients' pessimistic expectations about the consequences of physical activity, thereby decreasing levels of fear and increasing levels of activity (Vlaeyen & Linton, 2000).

In addition to treatments that target pain-related fear, developers of the Fear Avoidance Model suggest that more generalized cognitive and behavioural interventions may also help reduce levels of pain-related psychosocial risk factors and disability (Leeuw, et al., 2007a). For instance, cognitive-behavioural treatments have been recommended to target levels of catastrophic thinking and depressed mood (Leeuw, et al., 2007a). Interventions such as thought monitoring and cognitive restructuring have been suggested to help patients identify and correct maladaptive thinking about pain (Asmundson, et al., 2004). Other behavioural interventions, such as graded activity have also been recommended as

a general means of reducing levels of pain-related disability (Leeuw, et al., 2007a). While developers of the model broadly address the importance of a variety of cognitive and behavioral treatments, they argue that graded exposure interventions are the most effective means of reducing pain-related fear, and thereby disability (Leeuw, et al., 2007a).

Empirical Evaluations of the Fear Avoidance Model

Since its original development nearly 20 years ago, different aspects of the Fear Avoidance Model have been evaluated via empirical research. During roughly the same time period that the model was first introduced, key psychosocial questionnaires were developed. For instance, the creation and validation of the Tampa Scale of Kinesiophobia (Miller, Kori, & Todd, 1991) and the Pain Catastrophizing Scale (Sullivan, Bishop, & Pivik, 1995) in the 1990s facilitated the quantification of the model-relevant fear of movement and pain catastrophizing constructs, respectively. A range of validated self-report and objective measures has also been used to quantify the pain, hypervigilance, avoidance, disuse, depression and disability constructs (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). In this manner, measures have been developed for each of the major theoretical constructs included in the fear avoidance pathway.

A compelling body of research supports links between model-relevant constructs. For instance, pain-related fear, pain catastrophizing, and depression have been significantly related to avoidance behaviour, disability, and pain severity (Leeuw, et al., 2007a). Many studies have also shown that pre-treatment levels of pain catastrophizing, fear, and depression prospectively predict post-treatment levels of pain severity and pain-related disability (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). Several studies have revealed that these relationships are maintained even after controlling for pre-treatment levels of pain intensity (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). Taken together this research lends support for the importance of the predictive factors addressed within the Fear Avoidance Model.

Despite this broad support, however, empirical evaluations of the specific inter-relationships proposed within the Model remain in their infancy. The Fear Avoidance Model suggests that model-relevant variables relate to one another in a sequential fashion: pain catastrophizing leads to fear and avoidance, which in-turn lead to disuse, depression, and disability, which feed back into the pain experience. While many studies have reported significant correlations between model-relevant variables, few have tested whether multiple model-relevant variables inter-relate in a predicted fashion. Moreover, studies that have evaluated such relationships have been predominantly cross-sectional and have often failed to simultaneously test competing theoretical relationships (Kudel, Edwards, & Moric, 2005; Pincus, Vogel, Burton, Santos, & Field, 2006). This gap in the literature of the Fear Avoidance Model may have significant theoretical and clinical implications. For instance, a lack of prospective support for the fear avoidance pathway may call into question the validity of the putative disabling cycle that is central to the model and its theory-driven, clinical interventions.

Objectives of the Present Thesis

The purpose of the present thesis was to evaluate the prospective relationships proposed by the Fear Avoidance Model, and to assess alternate relationships among model-relevant variables. To meet these objectives three broad investigations were conducted: prospective sequential relationships were evaluated; the role of pain-related fear as a prospective predictor of multiple pain-related outcomes was tested; and, competing prospective relationships that were not proposed by the Fear Avoidance Model were evaluated. These analyses were conducted in three related studies, which are presented below. Discussions of study-specific results are addressed in each of the three manuscripts, while the broad theoretical implications of the combined findings are addressed in a general discussion.

Figures

Figure 1. Descartes' portrayal of physical response to tissue injury

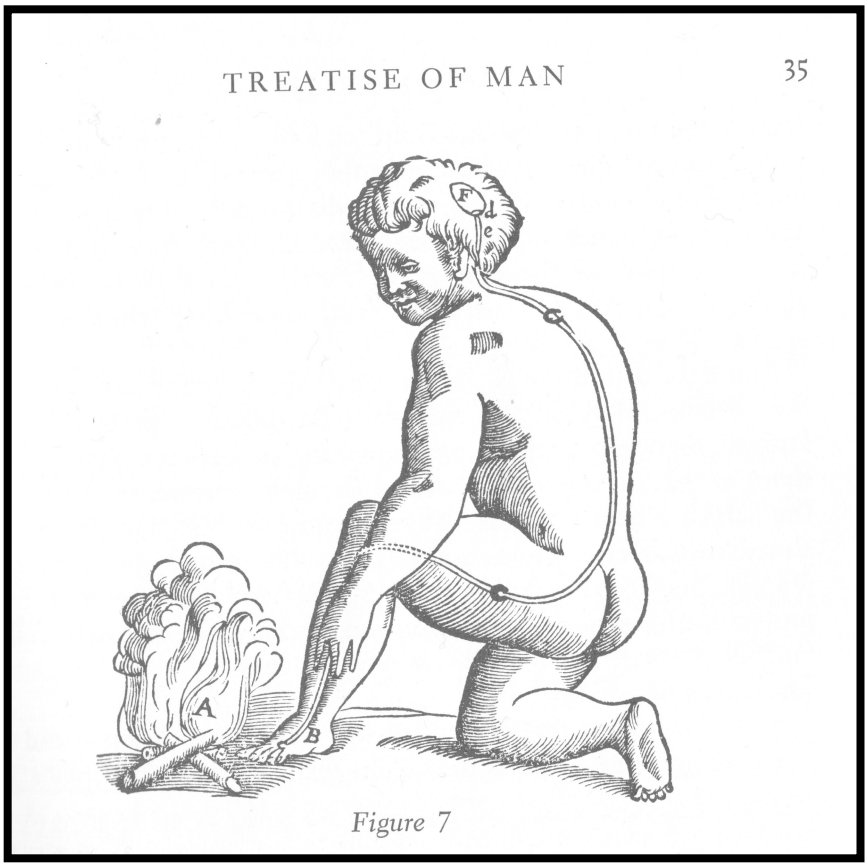


Figure 2. Descartes' portrayal of pain-related processing in the brain

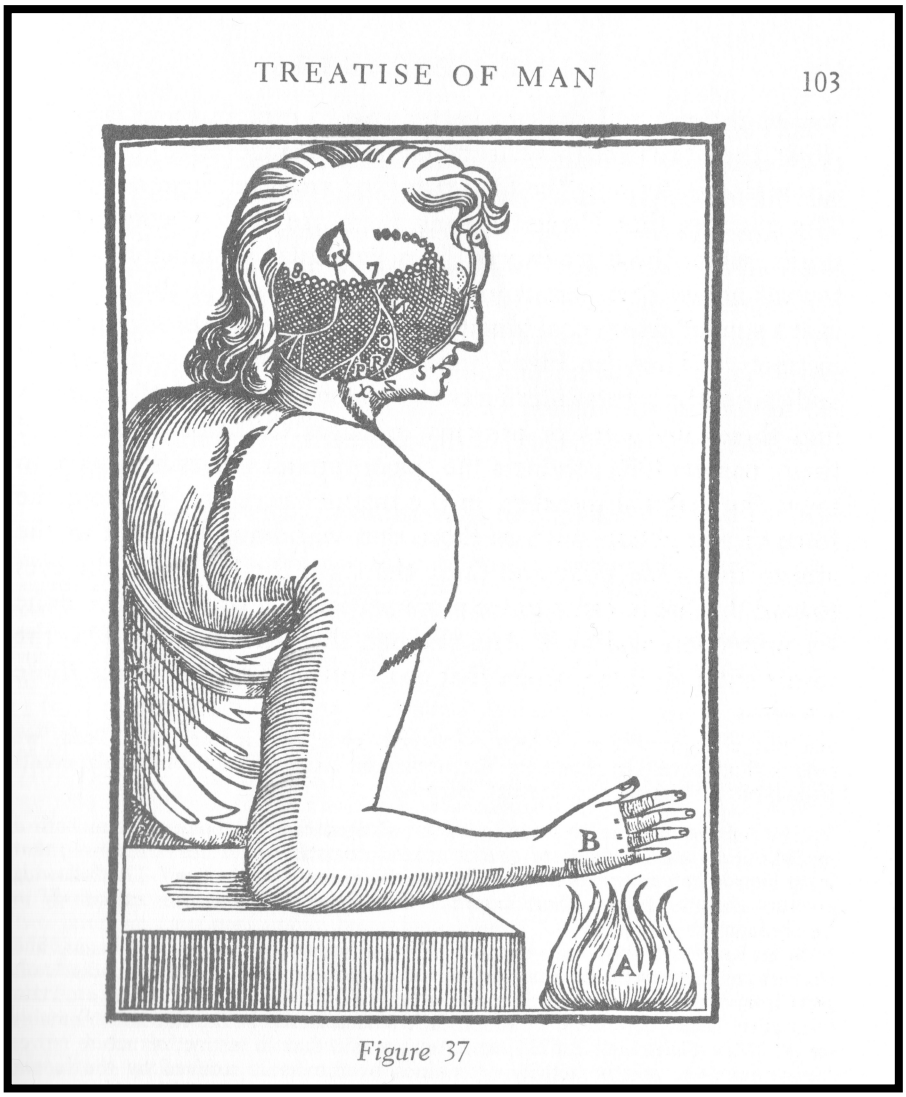


Figure 3. Melzack and Wall's Gate Control Theory of Pain

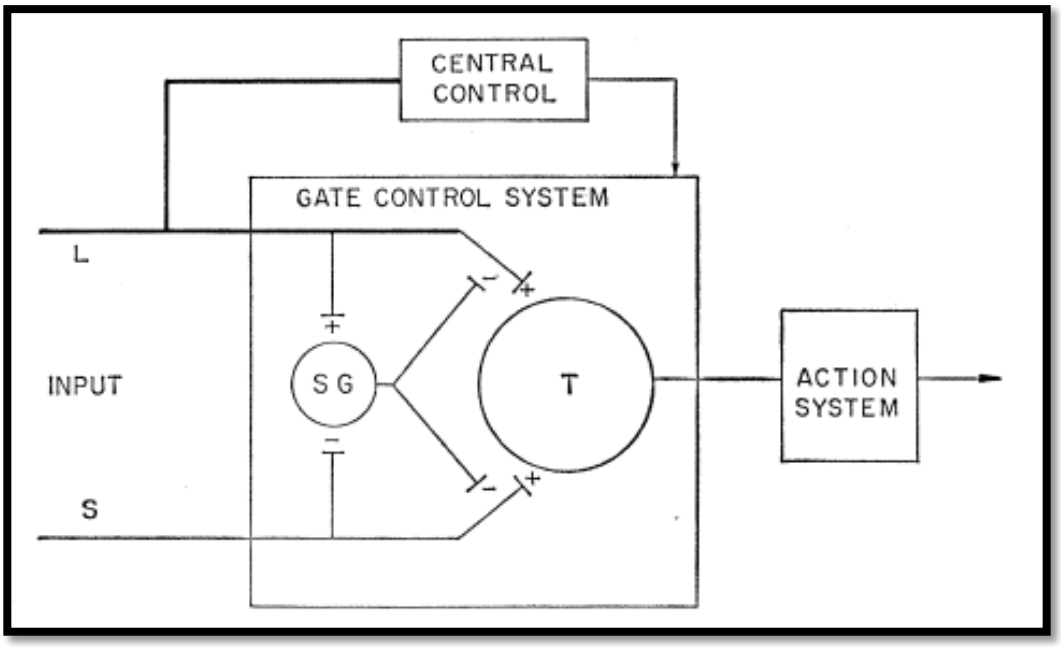


Figure 4. Cognitive Behavioral Model of Fear of Movement/(Re)Injury (Vlaeyen et al., 1995)

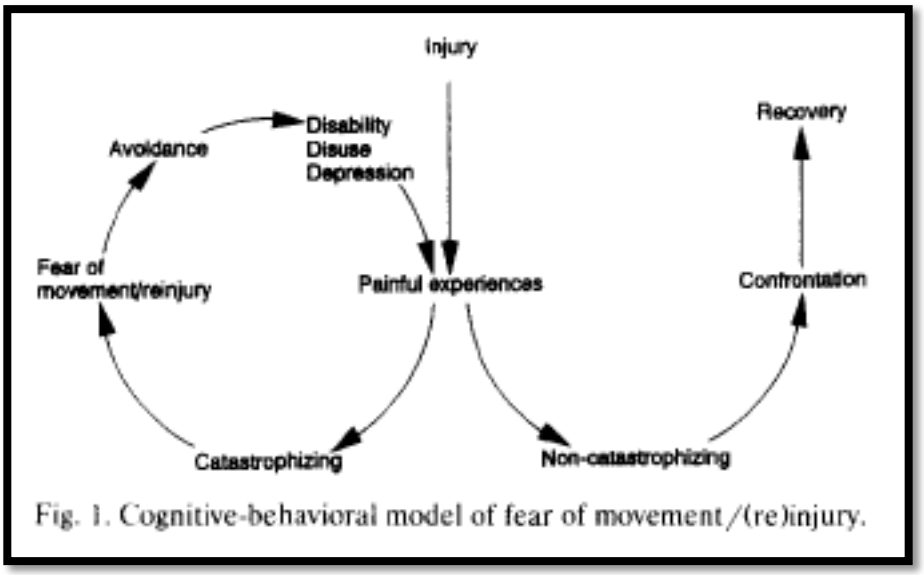


Fig. 1. Cognitive-behavioral model of fear of movement/(re)injury.

Figure 5. The Fear Avoidance Model (Vlaeyen and Linton, 2000)

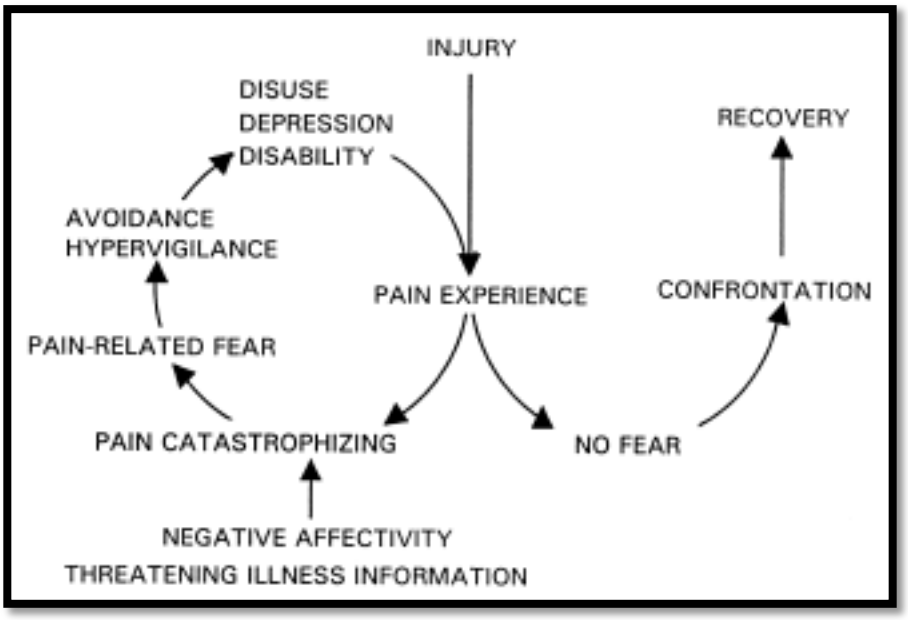


Figure 6. The Fear-Anxiety-Avoidance Model of Chronic Pain (Asmundson et al., 2004)

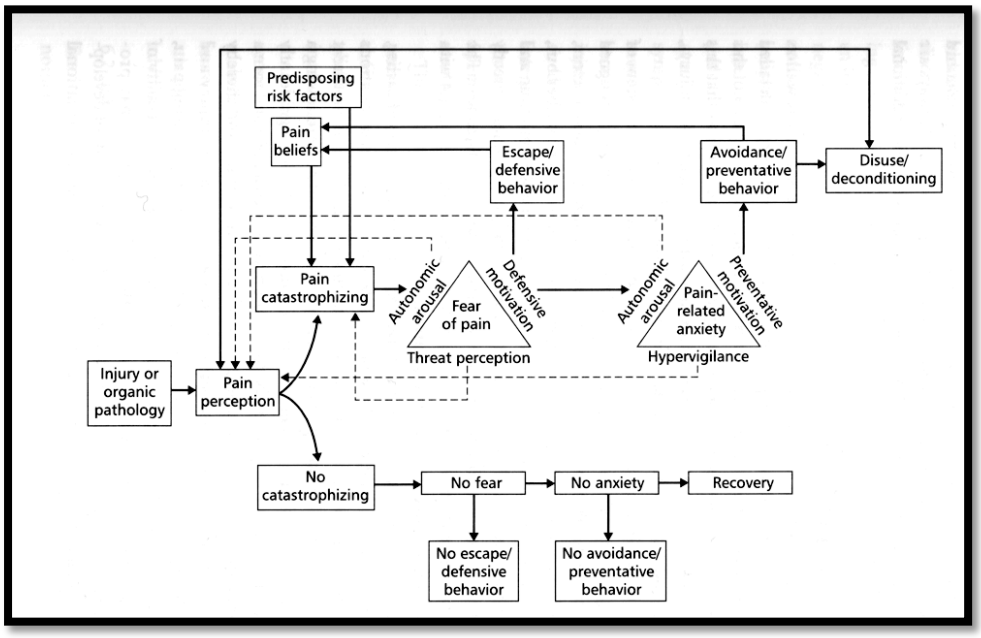
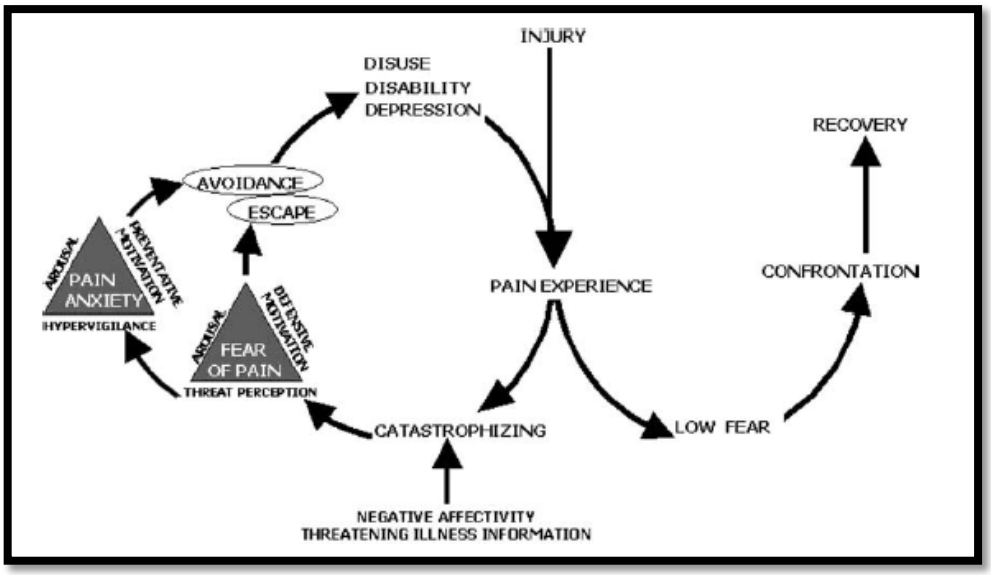


Figure 7. The Fear Avoidance Model of Chronic Pain (Leeuw et al., 2007)



CHAPTER 2: A PROSPECTIVE SEQUENTIAL ANALYSIS OF THE FEAR AVOIDANCE MODEL OF PAIN

Wideman TH, Adams H, Sullivan MJL. *Pain* 2009; 145(1): 45 -51.[5]

Abstract

The primary purpose of this study was to analyze the sequential relationships proposed by the Fear Avoidance Model of pain. Specifically, this study evaluated whether early change in catastrophizing predicted late change in fear of movement, and whether these factors influenced post-treatment return to work. Secondary analyses tested relationships between (1) early change in catastrophizing, late change in depression, and disability; and (2) early change in catastrophizing, late change in pain severity, and disability. Analyses were conducted on a sample of 121 individuals (82 men and 32 women) with a work-related musculoskeletal injury, and high baseline catastrophizing and fear of movement scores. Participants were enrolled in a 10-week community-based disability management intervention, and completed measures of catastrophizing, fear of movement, depression and pain severity at pre, mid and post-treatment. Return to work was assessed four weeks following termination of the intervention. Contrary to predictions, results from correlational analyses revealed non-significant relationships among indices of early change in catastrophizing and late changes in fear of movement, depression and pain severity. Multiple logistic regression analyses revealed that early change in catastrophizing, late changes in fear of movement and late change in pain severity were significant predictors of return to work, while late changes in depression were not. These findings highlight the importance of reductions in psychosocial risk factors in augmenting return to work outcomes. Implications for the fear avoidance model and future research are discussed.

Keywords: pain, fear of movement, catastrophizing, work disability, sequential analysis

Introduction

Work disability, caused by musculoskeletal injury, accounts for an estimated 20 billion dollars in direct annual expenditures to the USA's workers-compensation program (Baldwin, 2004). While the majority of injured workers show satisfactory recovery, up to 20% will suffer from prolonged pain and disability. Past research involving individuals with chronic pain has highlighted the importance of variables such as catastrophizing, fear of movement and depression in predicting prolonged work disability (Gheldof, Vinck, Vlaeyen, Hidding, & Crombez, 2005; Severeijns, Vlaeyen, van den Hout, & Picavet, 2004; Vowles, Gross, & Sorrell, 2004; Waddell, 2004).

The fear avoidance model of pain addresses the process by which catastrophizing and fear of movement influence disability (Leeuw, et al., 2007a; Vlaeyen, et al., 1995a; Vlaeyen & Linton, 2000). The model posits that individuals who exaggerate the threat value of pain stimuli (i.e. catastrophize) are more likely to develop fear of movement, which in turn will contribute to activity avoidance, disability and depression. Numerous investigations have reported findings consistent with the predictions of the fear avoidance model pain. For example, cross-sectional studies have reported significant correlations between measures of catastrophizing and fear of movement (Cook, Brawer, & Vowles, 2006; Goubert, Crombez, & Van Damme, 2004; Vlaeyen, et al., 1995a). Research has also shown that high scores on measures of catastrophizing and fear of movement are associated with self-reported inactivity and work-disability (Boersma & Linton, 2005a; Linton & Buer, 1995; McCracken, Faber, & Janeck, 1998; Peters, Vlaeyen, & Weber, 2005). In a recent cross-sectional study, fear of movement was shown to mediate the relationship between catastrophizing and self-reported disability (Nieto, Miró, & Huguet, 2009).

Prospective studies have also provided support for the fear avoidance model. High scores on measures of catastrophizing and fear of movement have been associated with increased risk of developing chronic pain and disability (Boersma & Linton, 2005b; Burton, Tillotson, Main, & Hollis, 1995). Past research suggests that predictor variables from the fear avoidance model make a

moderate, yet meaningful contribution to the prediction of pain and pain-related disability (Leeuw, et al., 2007a; Waddell, 2004). Research has also shown that treatment-related reductions in catastrophizing and fear predict reductions in self-reported disability (Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, 2002; Woods & Asmundson, 2008), and improved return to work outcomes (Staal, et al., 2008; Sullivan, Adams, Rhodenizer, & Stanish, 2006b; Sullivan & Stanish, 2003).

Recent reviews suggest strong support for various predictions of the fear avoidance model. Surprisingly, there is a lack of research addressing the sequential components of the model. While several studies have addressed recovery predictions of the model, most of this research has used a pre to post-treatment design. However, at least a three-wave panel design is needed to assess the sequential predictions of the fear avoidance model. Research addressing the sequential aspects of treatment-related changes in psychosocial factors is important not only from a theoretical perspective, but from a clinical perspective as well. Knowledge gained from this research, might provide the empirical foundation for the development of interventions designed to promote recovery from musculoskeletal injury.

The purpose of this study was to analyze the sequential relationships proposed by the fear avoidance model. A sample of work-injured individuals completed measures of catastrophizing, fear of movement, depression and pain severity at three time points throughout the course of a return to work intervention. We hypothesized that early change in catastrophizing would be associated with late changes in fear, which in turn would predict improved return to work outcomes. Secondary analyses addressed the relationships between changes in catastrophizing and fear of movement, and changes in depression and pain intensity.

Methods

Participants

The study sample was drawn from data collected on 401 work-disabled individuals who had been referred to a community-based disability reduction program (i.e. The Pain-Disability Prevention Program; PDP (Sullivan & Stanish, 2003)). For the purposes of this study, only individuals with initially high scores on a measure of pain catastrophizing and fear of movement, and complete data at all assessments points (pre-, mid-, post- treatment) were retained for analysis. The final study sample consisted of 121 individuals (82 men and 32 women) who were off work as a result of a work-related soft tissue injury (sample selection is described below). All participants were claimants of the Workers' Compensation Board of Nova Scotia, Canada.

Procedure

Intervention. Injured workers were considered for referral to a PDP trained psychologist when they were absent from work for more than 6 weeks, and medical evaluation revealed no evidence of organic pathology. The PDP Program was being piloted by the Workers' Compensation Board of Nova Scotia as a population health intervention aimed at preventing the development of chronic pain following occupational injury. All psychologists had previously attended a skills training workshop to familiarize them with the techniques of the intervention program. The psychologists who provided the PDP Program were independent practitioners in various communities of Nova Scotia, Canada. As part of this research initiative, psychologists were asked to forward copies of their assessment results to our research centre. The results of the psychological assessments were then linked to the WCB administrative database. In this manner, it was possible to examine the prospective relation between changes in psychological measures and return to work outcomes.

The PDP Program is a standardized 10-week intervention that uses structured activity scheduling strategies and graded activity involvement to target risk factors such as fear of movement/re-injury and perceived disability (Sullivan & Stanish, 2003). Thought monitoring and cognitive restructuring strategies are used to target catastrophic thinking and depression. Self-report measures of pain

severity, pain catastrophizing, fear of movement/re-injury, and depression were administered before implementation of the PDP Program, at its mid-point, and upon its conclusion at 10-weeks.

The PDP Program was designed such that the intervention could be terminated prior to 10 weeks if the client was ready to return to work. For many individuals, particularly those who were referred within weeks of injury, 10 weeks of intervention was not required to achieve successful work re-entry. Individuals who returned to work prior to completing all 10 weeks of the intervention had incomplete data on one or more of the assessment points, and are not included in the study sample. Clients who did not complete all 10 weeks of the intervention but returned to work were not be considered treatment dropouts. Only clients who did not complete the 10 weeks of the program and did not return to work were considered to be dropouts. By these criteria, 30 (7.5%) clients dropped out of treatment.

Approach to sample selection. Of the initial sample (N = 401), 162 (37.8%) participants had incomplete data for at least one assessment period. As the planned analyses for this study required that individuals have complete data for all measures these cases were not selected for the study sample. Of the remaining participants, 118 did not have high initial levels of catastrophizing or fear of movement (i.e. initial scores were equal to or less than the 50th percentile; initial score on the Pain Catastrophizing Scale (PCS) less than 21; initial score on the Tampa Scale for Kinesiophobia (TSK) less than 40). Thus, the final study sample consisted of 121 participants who had complete data and high initial catastrophizing and fear of movement scores. Figure 1 shows a breakdown of the described sample selection process.

Measures

Pain severity. The McGill Pain Questionnaire (MPQ) (Melzack, 1975) was used to measure current pain severity. Participants were asked to endorse adjectives that described their pain experience. The Pain Rating Index of the MPQ

is a weighted sum of all adjectives endorsed. Previous research has found the MPQ to be a reliable measure of pain severity (Turk, Rudy, & Salovey, 1985).

Pain catastrophizing. The PCS (Sullivan, et al., 1995) was used to assess pain-related thoughts and feelings. Previous research has shown that this 13 item, self-report questionnaire has good internal reliability (alpha = 0.87) (Sullivan, et al., 1995) and is a significant predictor of many pain-related outcomes (Sullivan, et al., 2001).

Fear of movement. The TSK (Roelofs, Goubert, Peters, Vlaeyen, & Crombez, 2004; Swinkels-Meewisse, Swinkels, Verbeek, Vlaeyen, & Oostendorp, 2003) was used to evaluate participants' fear of movement and re-injury. This self-report scale consists of 17 statements that address worries and concerns associated with performing physical activity. The TSK has been shown to be internally reliable (alpha = .77) (Vlaeyen, et al., 1995a) and associated with various measures of avoidance and disability (Leeuw, et al., 2007a).

Depression. The Beck Depression Inventory II (BDI-II) (Beck, Steer, & Brown, 1996) was used to assess severity of depressive symptoms. The BDI-II consists of 21 statements that describe various symptoms of depression. Participants were asked to endorse statements that best represented their feelings over the past two-weeks. The BDI-II has been shown to be a valid and reliable measure of depression for individuals with musculoskeletal conditions (Bishop, Edgley, Fisher, & Sullivan, 1993).

Return to Work. Return to work was based on WCB file status at four weeks following PDP program completion. WCB claim files were closed as a result of either returning to pre-injury full-time employment or gaining alternate full-time employment. Only participants whose claim files were closed at four weeks post treatment were classified as having successfully returned to work. All other participants were coded as not returning to work.

Data Analytic Approach

Using data from the three assessments, two change indices were calculated for each process variable (i.e. catastrophizing, fear of movement, depression and

pain severity), namely early change (pre-treatment scores minus mid-treatment scores) and late change (mid-treatment scores minus post-treatment scores). Positive scores reflect improvement in treatment-related variables, while negative scores indicate a decline.

Using pre-treatment scores, correlational analyses were performed to assess cross-sectional relationships. Also, to fully address the relationships between pre-treatment scores and return to work outcomes, both univariate mean comparisons and hierarchical logistic regressions were performed. These analyses were conducted in an effort to replicate findings from previous research that used cross-sectional measures. Correlational analyses were used to evaluate both cross-sectional and longitudinal relationships among change indices. Univariate mean comparisons and hierarchical logistic regressions were used to evaluate the relationships between the change indices and return to work outcomes.

Results

Sample Characteristics

The mean age of participants in the sample was 41.9 years with a standard deviation of 7.7 years. The majority of participants' reported that their back was the site of their musculoskeletal injury (73.6%). Injury duration ranged from less than three months to greater than one year, and was uniformly distributed across this range. Prior to injury, the majority of participants were employed as laborers (47.1%), trades people (24.8%) or nurses (7.4%). Additional details regarding sample characteristics can be found in Table 1.

Table S1 in Appendix 1 shows the means, standard deviations and results of repeated measures analysis of variance (ANOVA) for all treatment-related process variables. The mean and standard deviation for initial measures of catastrophizing (mean = 34.51, standard deviation = 7.78), fear of movement (48.03, 5.74), depression (21.07, 11.40) and pain intensity (38.12, 14.38) were comparable to previous studies that have selected participants based on high scores on measurements of psychosocial risk factors (Leeuw, et al., 2008). Repeated measures ANOVA revealed significant changes between pre- to mid-

treatment levels of catastrophizing ($F_{1, 119} = 79.230$, $p < 0.01$), fear of movement ($F_{1, 119} = 18.055$, $p < 0.01$) and depression ($F_{1, 119} = 11.787$, $p < 0.01$); and significant changes between mid- to post-treatment levels of catastrophizing ($F_{1, 119} = 13.574$, $p < 0.01$) and fear of movement ($F_{1, 119} = 10.411$, $p < 0.01$).

Relations Among Pretreatment Variables

Table S2 in the Appendix 1 shows cross-sectional correlations among pre-treatment assessment variables. Consistent with previous research, pre-treatment catastrophizing was significantly correlated with pre-treatment fear of movement (Pearson coefficient = 0.454, $p < 0.01$). Pre-treatment catastrophizing and pre-treatment fear of movement were also significantly correlated with pre-treatment depression and pain severity (Pearson coefficients ranged from 0.233 to 0.547, *Ps* < 0.05).

Predicting Return to Work Outcomes From Pre-Treatment Scores

Pre-treatment means, standard deviations and effect sizes for participants that returned to work, and did not return to work are shown in Table S3 in Appendix 1. Participants' sex ($\chi^2_1 = 2.96$, $p > 0.05$), age ($p > 0.05$) and duration of injury ($p > 0.05$) were not significantly associated with the probability of return to work. Participants who did not return to work obtained significantly higher pre-treatment scores on measures of fear of movement ($F_{1, 119} = 5.550$, $p < 0.05$) and pain severity ($F_{1, 119} = 19.064$, $p < 0.01$) than did individuals who did return to work.

Logistic regression analysis was performed to examine the value of pre-treatment catastrophizing, fear of movement, depression and pain severity scores in predicting post-treatment return to work outcomes (see Table 2). Consistent with previous research, higher levels on pre-treatment pain severity were associated with a lower probability of return to work (Gheldof, et al., 2005; Godges, Anger, Zimmerman, & Delitto, 2008).

Relations Among Change Indices

Table S4 in Appendix 1 shows the results of the cross-sectional correlation analysis for all change indices. Consistent with past research, our results revealed several significant cross-sectional correlations. Early changes in catastrophizing, fear of movement and depression were all significantly inter-correlated (significant Pearson coefficients ranged from 0.372 to 0.418, $p < 0.01$). All indices of late change were significantly correlated, and a particularly strong relationship between late changes in catastrophizing and depression was found (Pearson coefficients ranged from 0.271 to 0.634, $p < 0.01$).

Table S5 in Appendix 1 shows the results of the correlational analyses for early and late change indices. Contrary to our predictions, analyses failed to reveal any significant relationships between early change in catastrophizing and late changes in fear of movement, depression or pain severity. The only significant relationships between early and late change indices were auto-correlations (i.e. early changes predicting late changes of the same factor) for fear of movement (Pearson coefficient = -0.315, $p < 0.01$) and pain severity (Pearson coefficient = -0.314, $p < 0.01$). Similar to past research (Burns, Glenn, Bruehl, Harden, & Lofland, 2003a; Burns, Kubilus, Bruehl, Harden, & Lofland, 2003b), each of these significant relationships were negatively correlated, indicating that increased early change in one of these variables was significantly related to decreased late change in the same variable.

Predicting Return to Work From Change Indices

Table S6 in Appendix 1 shows mean comparisons of age, injury duration and change variables for participants that returned to work, and those that did not return to work. Results revealed that participants who returned to work, compared to participants who did not return to work, showed greater early ($F_{1, 119} = 6.268$, $p < 0.05$) and late changes ($F_{1, 119} = 16.443$, $p < 0.01$) in catastrophizing, and greater late change in fear of movement ($F_{1, 119} = 6.690$, $p < 0.05$) and pain-severity ($F_{1, 119} = 7.992$, $p < 0.01$).

The fear avoidance model suggests that different process variables may have an additive effect in the reduction of disability. To assess these predictions

three separate hierarchical logistic regression analyses were conducted, in which process variables were entered in model-relevant groups. Results from these analyses are shown in Table 3. The first hierarchical logistic regression analysis tested our primary predictions that early change in catastrophizing and late change in fear of movement would significantly predict return to work. Early change in catastrophizing was entered in Step 1 of the analysis, and late change in fear of movement was entered in Step 2 of the analysis. Results revealed that early change in catastrophizing was a significant predictor of return to work when entered in Step 1 ($p < 0.05$) and late change in fear of movement ($p < 0.05$) was found to be a significant predictor of return to work when entered in Step 2.

Similar hierarchical logistic regression analyses were performed to assess alternate linkages between change indices and return to work outcomes. Proceeding from a cognitive-behavioural model of catastrophizing and depression, one analysis addressed whether early change in catastrophizing and late change in depression would predict return to work. Results revealed that while early change in catastrophizing was a significant predictor of return to work ($p < 0.05$), late change in depression was not. Finally, proceeding from previous research showing relations between catastrophizing and pain severity, a logistic regression was conducted addressing whether early change in catastrophizing and late change in pain intensity would predict return to work. Results revealed that both early change in catastrophizing ($p < 0.05$) and late change in pain severity ($p < 0.01$) were significant predictors of return to work.

While results from mean comparisons and model-relevant logistic regression analyses suggest that early changes in catastrophizing and late changes in catastrophizing, fear of movement and pain severity are significant treatment-related predictors of return to work, these analyses do not fully address the degree to which the explained variance of these predictors overlap. To examine this issue, a final hierarchical logistic regression analysis was performed in which all significant, univariate treatment-related variables were evaluated as predictors of return to work. Early change in catastrophizing was entered in Step 1 of this analysis, while late change in catastrophizing, fear of movement and pain severity

were entered in Step 2. As shown in Table 4, results from this analysis indicate that only early ($p < 0.05$) and late ($p < 0.01$) changes in catastrophizing contribute unique variance to the prediction of return to work, while changes in fear and pain severity were non-significant.

Discussion

The predictions of the fear avoidance model can be addressed by analyzing different relationships among the variables in our study: the cross-sectional correlations among scores; the predictive value of pre-treatment scores in determining work-disability; the cross-sectional inter-relationships among change indices; and finally the sequential relationships of change indices, and the utility of these indices in predicting disability. Each of these relationships, and their relevance to the fear avoidance model of pain, will be addressed in turn.

The fear avoidance model proposes that high levels of catastrophizing are related to elevated levels of fear of movement, depression and pain severity. Results from our cross-sectional correlational analysis of pre-treatment measures were consistent with both past research (Cook, et al., 2006; Goubert, et al., 2004; Vlaeyen, et al., 1995a), and these hypotheses. Our findings also support model hypotheses that suggest significant correlations among treatment-related reductions in catastrophizing and other fear avoidance variables. Results from our cross-sectional analyses of change indices revealed significant correlations between change in catastrophizing, fear of movement and depression. These findings indicate that the different model-relevant variables co-vary to a significant degree.

The fear avoidance model also posits that pain-related disability can be lowered by reducing catastrophic thinking. Findings showing that early and late changes in catastrophizing were significant predictors of return to work support this hypothesis and are consistent with previous research that has found reductions in catastrophizing to be significant predictors of pain-related outcomes (Smeets, Vlaeyen, Kester, & Knottnerus, 2006a; Sullivan, et al., 2006b; Sullivan, et al., 2005c).

Although within-period changes in catastrophizing and fear of movement were significantly correlated, contrary to predictions, early change in catastrophizing was not correlated with later change in fear of movement. Early change in catastrophizing also failed to predict late changes in depression and pain severity. The fear avoidance model predicts sequential relationships among model-relevant factors, however, it does not address the length of the temporal intervals during which they occur. It is possible that our lack of significant findings was caused by assessment intervals (five weeks) that were too long to measure significant relationships amongst early and late change indices. Changes in fear of movement, depression and pain severity may follow changes in catastrophizing more closely (i.e. in the first five weeks) than could be measured with our methodology. Findings that many of the cross-sectional correlations were significant would be consistent with this interpretation. Future research that uses alternate assessment interval lengths may shed further light on the proposed sequential relationships. It is also necessary to consider that the sequential parameters of inter-relations among catastrophizing, fear and disability may differ for the *development* of pain-related disability and *recovery* of pain-related disability.

Past research suggests that weekly assessment intervals might be more likely to reveal sequential relations between changes in cognitive and affective variables. Literature addressing patients' response to cognitive-behavioral therapy has shown that significant reductions in measures of depression can be achieved in the first four weeks of treatment (Ilardi & Craighead, 1994). Moreover, results from two studies that used one-week intervals to evaluate such interventions revealed that significant reductions in depression were preceded by cognitive change (Tang & DeRubeis, 1999; Tang, DeRubeis, Beberman, & Pham, 2005). Weekly assessments may also be a useful vehicle for future analyses on the sequential predictions of the fear avoidance model. Further research in this area will help determine if our findings were unique to the assessment intervals used, and whether these conclusions have broader ramifications for the treatment-related relationships proposed by the fear avoidance model.

Our findings lend qualified support for the predicted relationship between reductions in fear of movement and disability. The fear avoidance model suggests that reductions in pain-related fear predict reductions in disability. Results from our initial univariate logistic regression analysis support this hypothesis by showing that late changes in fear of movement significantly predict return to work beyond early changes in catastrophizing. However, in our final logistic regression analysis when late changes in fear of movement were made to compete with other variables, they were no longer significant predictors of return to work; only early and late changes in catastrophizing significantly predicted return to work. Consistent with results from past research, these findings suggest that changes in catastrophizing can influence disability independent of changes in fear of movement (Sullivan, et al., 2006b; Sullivan, et al., 2005c).

Previous literature that addresses the qualitative relationship between catastrophizing and fear suggest that these variables have partially overlapping constructs. The fear avoidance model conceptualizes fear as an emotional reaction to a threatening, pain-related situation, and catastrophizing as the cognitive dimension of pain-related fear (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). Pain catastrophizing, as measured by the PCS, consists of three sub-dimensions: magnification, rumination and helplessness. In a topical review, Severeijns et al. (Severeijns, Vlaeyen, & van den Hout, 2004) suggest that these sub-dimensions represent different types of cognitive appraisal of pain. Magnification and rumination dimensions constitute primary appraisal and relate to the perception of pain as a threatening experience. The helplessness dimension of catastrophizing is classified as secondary appraisal and relates to a perceived inability to cope with pain. When framed within the fear avoidance model, there appears to be only partial overlap between the cognitive components of catastrophizing and the affective components of fear. For example, the affective components of fear involve threat perception and hypervigilance and appear to overlap with the magnification and rumination dimensions of catastrophizing (i.e. primary cognitive appraisal). However, as currently described in the model, fear does not address one's ability to cope with pain. Thus, the helplessness dimension of

catastrophizing (i.e. secondary cognitive appraisal) seems to lack an affective correlate within the model. This sub-dimension may therefore be responsible for distinguishing changes in catastrophizing from changes in fear.

It is also possible that the unique relationship between changes in catastrophizing and return to work can be attributed to treatment-related reductions in the helplessness dimension of the PCS. In this manner, changes in fear of movement may occur independently of the relationship between change in helplessness and return to work. Unfortunately, as sub-scale scores were not collected in our study, these predictions cannot be directly evaluated with analyses from our data. Previous research addressing the predictive value of helplessness, however, provides support for these hypotheses. For example, Sullivan et al. (Sullivan, Lynch, & Clark, 2005b) have shown that the PCS helplessness dimension predicts pain intensity beyond the other dimensions of catastrophizing. Similarly, Burns et al. (Burns, et al., 2003b) have shown that treatment-related reductions on a perceived helplessness index significantly predict activity interference. Future research will need to further explore these relationships by examining the predictive utility of changes in the sub-dimensions of PCS and items on the TSK in determining disability.

While our findings, as well as previous research (Sullivan, et al., 2001), suggest that reductions in catastrophizing translate into improved pain-related outcomes, the most effective interventions by which these reductions might occur remain unclear. Previous research has focused on cognitive-behavioral interventions as an effective means of reducing catastrophizing (Morley, Williams, & Hussain, 2008; Thorn, et al., 2007; Turner, Holtzman, & Mancl, 2007). However, studies that have evaluated interventions that do not directly target cognitive change, such as traditional physiotherapy or other activity-based treatments, have revealed comparable reduction in catastrophizing (Smeets, et al., 2006a; Sullivan, et al., 2006b). Additionally, alternate interventions such as education and exposure have been shown to lower levels of catastrophizing (Leeuw, et al., 2008). The effectiveness of such a broad range of interventions suggests that clinical reductions in catastrophizing are not strictly bound to

cognitive interventions. These findings also highlight our limited understanding of the mechanisms by which changes in catastrophizing occur. This theoretical ambiguity can lead to costly clinical interventions, which may aim to administer all interventions that have been shown to reduce catastrophizing. To facilitate the selection of focused and cost-effective interventions, future research will need to further explore the extent to which different interventions uniquely influence changes in catastrophizing.

Some caution should be exercised in generalizing the results of this research. First, this study did not evaluate long-term return to work status. It is possible that significant predictors of return to work vary depending on when this outcome is assessed. While changes in catastrophizing may predict return to work at four weeks following treatment, different factors may be significant with longer-term follow up. Furthermore, our selection criteria may have influenced our findings. For example, we only tested fear avoidance predictions in participants with complete data on all assessments. This subsample might be considered more treatment resistant than individuals who returned to work after a few weeks of intervention. Similarly, by only selecting individuals with high pre-treatment scores on both measures of pain catastrophizing and fear of movement our sample may have represented a highly disabled population. Past research suggests that populations that are more chronic can benefit from intensive multidisciplinary interventions (Haldorsen, et al., 2002). It is possible that such interventions may have resulted in greater treatment-related reductions in psychosocial process variables. Future research will need to address whether the same relations would be observed if these types of interventions were used.

Despite these limitations, this study presents preliminary findings that do not support the sequential predictions of the fear avoidance model of pain. Findings from this study highlight the importance of reducing levels of catastrophizing for clinical interventions that aim to lower work disability, while calling attention to the need for further research that addresses the most cost-effective mechanisms by which these reductions can occur. Our results also suggest that decreasing the time intervals between assessment points might

improve the detection of sequential inter-relations among the cognitive, affective and behavioral components of recovery during treatment of musculoskeletal conditions.

Acknowledgements

The authors thank the Workers' Compensation Board of Nova Scotia for facilitating this program of research. This research was supported by grants from the Canadian Institutes of Health Research (CIHR), the Social Sciences and Humanities Research Council of Canada, and by the Physiotherapy Foundation of Canada via the CIHR Institute of Aging Scholarship in Mobility. The authors have no financial interests in the outcome of this research.

Tables

Table 1
Characteristics of the study sample (N = 121)

Characteristics	N (%)
Age – M (SD)	41.91 (7.68)
Returned to work	37 (30.6)
Gender	
Male	82 (67.8)
Female	39 (32.2)
Injury duration	
< 3 mths	25 (20.7)
3 to < 6 mths	31 (25.6)
6 to < 12 mths	32 (26.4)
≥ 12 mths	33 (27.3)
Injury site	
Back	89 (73.6)
Neck	2 (1.7)
Upper extremity	19 (15.7)
Multiple	11 (9.1)
Occupation	
Laborer	57 (47.1)
Nursing	9 (7.4)
Fishing	9 (7.4)

Table 1

Characteristics of the study sample (N = 121)

Driving	4 (3.3)
Retail	6 (5.0)
Trade	30 (24.8)
Clerical	4 (3.3)

Table 2

Hierarchical logistic regression analysis: pre-treatment predictors of return to work

Variables	$\Delta\chi^2$	Δdf	R^2	-2LL	β	OR	95% CI
Initial PCS	19.647**	4	0.212	129.349	-0.012	0.988	0.923 – 1.057
Initial TSK					-0.062	0.940	0.854 – 1.035
Initial BDI					0.007	1.007	0.959 – 1.057
Initial MPQ					-0.057	0.944	0.913 – 0.977**

Injury dur = injury duration; PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; OR = odds ratio; CI = confidence interval; *$p < 0.05$; **$p < 0.01$.

Table 3

Hierarchical logistic regression analyses: predictors of return to work

Step	Variables	$\Delta\chi^2$	Δdf	R^2	-2LL	β	OR	95% CI
1	ch1 PCS	6.050*	1	0.069	142.946	0.068	1.071	1.013 – 1.132*
2	ch2 TSK	6.599*	1	0.140	136.347	0.087	1.091	1.018 – 1.169*
2	ch2 BDI	2.890	1	0.101	140.056	0.041	1.042	0.991 – 1.094
2	ch2 MPQ	8.251	1	0.157	134.695	0.048	1.049	1.013 – 1.086**

ch1 = early change; ch2 = late change; injury dur = injury duration; PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; OR = odds ratio; CI = confidence interval; $^{*}p < 0.05$; $^{**}p < 0.01$.

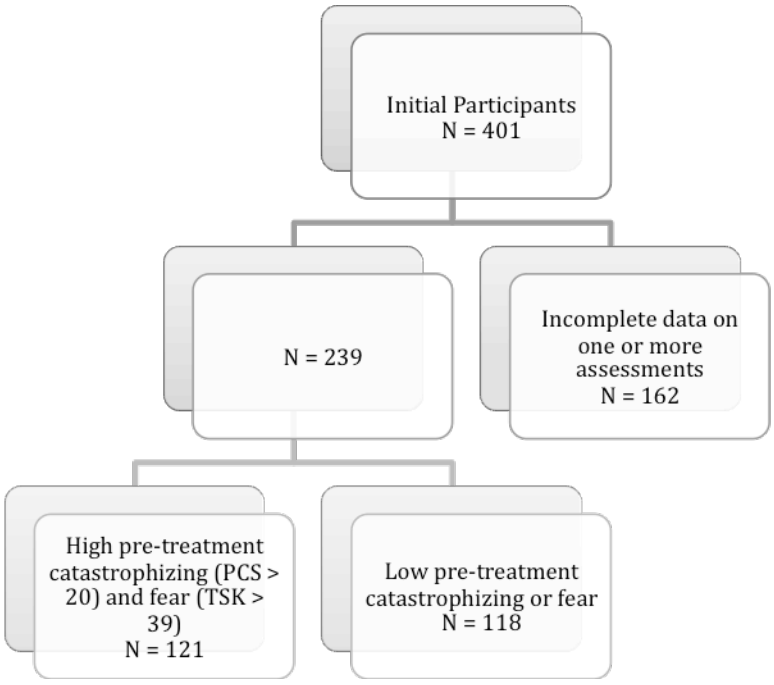
Table 4 Multiple logistic regression analyses: predictors of return to work

Step	Variables	$\Delta\chi^2$	Δdf	R^2	-2LL	β	OR	95% CI
1	ch1 PCS	6.050*	1	0.069	142.946	0.068	1.071	1.013 – 1.132*
2	ch2 PCS	19.960**	3	0.273	122.985	0.081	1.085	1.020 – 1.153**
	ch2 TSK					0.039	1.040	0.963 – 1.123
	ch2 MPQ					0.027	1.027	0.987 – 1.069

ch1 = early change; ch2 = late change; PCS = Pain catastrophizing; TSK = Fear of movement; MPQ = Pain intensity; OR = odds ratio; CI = confidence interval; *$p < 0.05$; **$p < 0.01$.

Figures

Figure 1. Sample selection flow-chart



PREFACE TO CHAPTER 3

We began our examination of the prospective relationships proposed by the Fear Avoidance Model in Study 1. Our results provided general support for the predictive value of model-relevant risk factors, but failed to support specific model predictions. The absence of support for prospective sequential relationships suggests that model-relevant psychosocial risk factors may be less inter-dependent than described by the Fear Avoidance Model. Also, our final regression model, which showed that changes in pain-related fear were not significantly linked to treatment outcome, raises questions about the central theoretical role ascribed to the fear construct. We explore and expand on both of these issues in Study 2.

In Study 2 we evaluated a key aspect of the Fear Avoidance Model, namely whether pain-related fear acts as a common psychological conduit for multiple pain-related outcomes. We also tested alternate hypotheses by examining whether model-relevant psychosocial factors act as differential predictors of pain-related outcomes, and whether *pain self-efficacy*, a factor not included in the Fear Avoidance Model, contributes any unique predictive value. When considered together, Study 1 and Study 2 provide a comprehensive assessment of the prospective inter-relationships proposed in the Fear Avoidance Model. Findings from these evaluations are therefore expected to shed important light on the level of empirical support for the fear avoidance pathway.

CHAPTER 3: DIFFERENTIAL PREDICTORS OF THE LONG-TERM LEVELS OF PAIN INTENSITY, WORK DISABILITY, HEALTHCARE USE, AND MEDICATION USE IN A SAMPLE OF WORKERS' COMPENSATION CLAIMANTS

Wideman TH & Sullivan MJL. *Pain* 2011; 152(2): 376-383.[6]

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Abstract

The Fear Avoidance Model of pain (FAM) conceptualizes pain catastrophizing as the cognitive antecedent of pain-related fear, and pain-related fear as the emotional antecedent of depression and disability. The FAM is essentially one of mediation whereby pain-related fear becomes the process by which depression or disability ensues. However, emerging literature suggests that pain catastrophizing, pain-related fear and depression might be at least partially distinct in their prediction of different pain-related outcomes. The primary purpose of the present study was to evaluate whether psychological factors in the FAM (pain catastrophizing, pain-related fear and depression) differentially predict long-term pain-related outcomes. Toward this objective, we conducted a prospective study using a cohort of 202 individuals with sub-acute, work-related musculoskeletal injuries. Participants completed a 7-week physical therapy program with a functional rehabilitation orientation. Post-treatment measures of fear of movement, pain catastrophizing, depression and pain self-efficacy were used to predict the persistence of pain symptoms, healthcare utilization, medication use and return to work at one-year follow-up. Results from hierarchical linear and logistic regression analyses revealed that pain catastrophizing and fear of movement act as differential predictors of long-term pain-related outcomes. Specifically, we found unique relationships between pain catastrophizing and long-term pain intensity, and fear of movement and long-term work disability. After controlling for pain intensity and FAM variables, pain self-efficacy was shown to be a unique predictor of medication use. Implications for the FAM and the clinical management of musculoskeletal pain conditions are discussed.

Keywords: pain, fear avoidance, catastrophizing, depression, self-efficacy, prospective analysis, work disability, healthcare utilization, medication use

Introduction

Work-related musculoskeletal injury is a major contributor to workers' compensation expenditures. In the United States alone, the annual direct costs associated with work-related musculoskeletal injuries are an estimated 20 billion dollars (Baldwin, 2004). Previous research suggests that psychological factors in the sub-acute phase of recovery are important predictors of recovery trajectories following work-related injuries (Pincus, Burton, Vogel, & Field, 2002). The Fear Avoidance Model of pain (FAM) offers a theoretical explanation for the mechanisms by which psychological factors impact on long-term health and mental health outcomes in individuals who have sustained musculoskeletal injuries (Leeuw, et al., 2007a; Vlaeyen, et al., 1995a; Vlaeyen & Linton, 2000).

The FAM proposes sequential relations between pain catastrophizing, pain-related fear, depression, disability, and pain intensity (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). The FAM conceptualizes pain catastrophizing as the cognitive antecedent of pain-related fear, and pain-related fear as the emotional antecedent of depression and disability. The model is essentially one of mediation whereby pain-related fear becomes the process by which depression or disability ensues. The FAM describes this process as recursive, such that, with time, it is expected to lead to increased pain, which then might elicit more catastrophizing and fear, thus perpetuating a downward cycle.

However, there is a basis for questioning the mediational relations posited by the FAM. For example, Gheldof and his colleagues recently showed that fear of movement prospectively predicted measures of disability, but not pain intensity (Gheldof, et al., 2010). Similarly, Wideman et al. showed that reductions in pain catastrophizing influenced pain outcomes independent of changes in fear (Wideman, Adams, & Sullivan, 2009). Findings from Sullivan et al. also suggested that reductions in catastrophizing led to decreased depression independent of fear (Sullivan, Adams, Thibault, Corbière, & Stanish, 2006a).

Emerging literature suggests that catastrophizing, fear and depression might be at least partially distinct in their prediction of different pain-related outcomes. For example, there are grounds for suggesting that catastrophizing

might impact on the persistence of pain, independent of fear, and that pain-related fear might impact on disability independent of catastrophizing (Sullivan, et al., 2009). Previous research also suggests that depression might be an independent predictor of other pain-related outcomes, such as healthcare utilization and medication use (Boersma & Linton, 2006; Braden, et al., 2009; Pincus & Newman, 2001). Together, these findings suggest that psychological predictors may have unique relationships with different pain-related outcomes.

The primary purpose of the present study was to evaluate whether the psychological factors in the FAM differentially predict long-term pain-related outcomes. Toward this objective, we conducted a prospective study using a cohort of individuals with sub-acute, work-related musculoskeletal injuries. Participants completed a seven-week physical therapy program with a functional restoration orientation. Post-treatment measures of fear of movement, pain catastrophizing and depression were used to predict the persistence of pain symptoms, return to work, healthcare utilization and medication use at one-year follow-up. The predictive utility of pain self-efficacy was also examined in light of recent findings suggesting that this variable might outperform FAM factors as a predictor of pain-related outcomes (Foster, Thomas, Bishop, Dunn, & Main, 2010). Based on previous findings, it was hypothesized that there would be unique relationships between levels of pain catastrophizing and pain intensity; fear of movement and work disability; and levels of depression and healthcare and medication use.

Methods

Study Design and Recruitment

We conducted a prospective cohort study of consecutive patients who were referred for physical therapy at one of six clinics in the province of Quebec, Canada. Patients were eligible for participation in the study if they were between 18 and 65 years old, had a work-related, musculoskeletal back or neck injury (i.e. a soft-tissue sprain or strain) that was in the sub-acute phase (i.e. three to 12 weeks since initial injury) and were receiving wage indemnity benefits from the

provincial workers' compensation board at the time of their initial physical therapy consultation. Patients were excluded from the study if there was clinical evidence of vertebral fracture, disc herniation, infectious disease, rheumatoid arthritis, ankylosing spondilitis or any medical contraindication to participating in a physical examination. Eligible and interested patients were provided with information about the study by their clinicians and were asked to sign a consent form if they agreed to participate in the study. The research program was approved by the research ethics committees of the *Centre de recherche interdisciplinaire en réadaptation du Montréal métropolitain* and the Université de Montréal.

Procedure

Treating clinicians administered self-report questionnaires at participants' initial consultations and following seven weeks of physical therapy. Participants' baseline information (age, sex, highest level of education, occupation, location of injury, time since injury and medication use) was also collected at the time of initial assessment. One year following the initial assessment, participants completed a follow-up telephone interview. Participants were compensated $25 for completing the questionnaires and $25 for completing the telephone interview.

The physical therapy intervention was designed to reduce pain and disability associated with musculoskeletal conditions. While the content of the physical therapy interventions varied at the discretion of the treating clinician, all interventions conformed to practice guidelines for early intervention of musculoskeletal problems that was consistent with the reimbursement policies of the provincial workers' compensation board. All interventions were characterized by a functional restoration orientation that emphasized mobilization and activity. Treatment consisted primarily of joint manipulation, active range of motion exercises and strengthening exercises, progressively increasing in intensity. The frequency of visits was three sessions per week. No specific data on adherence to treatment were available.

Measures

Participants completed self-report measures of pain intensity, pain catastrophizing, fear of movement, depressive symptoms and pain self-efficacy at treatment onset and upon completion of seven weeks of treatment. The follow-up interview was conducted one-year after initial assessment and evaluated participants' pain intensity, return to work status, healthcare utilization and medication use.

Pain intensity. A numeric rating scale was used to measure participants' levels of pain severity at the time of assessment. Participants were asked to rank their pain on an 11-point numeric scale that ranged from 0 (no pain) to 10 (excruciating pain). Previous research has found this scale to be a reliable measure of pain intensity (Downie, et al., 1978).

Pain catastrophizing. Pain catastrophizing was measured with the 13-item Pain Catastrophizing Scale (PCS) (Sullivan, et al., 1995). The PCS quantifies the frequency of pain-related thoughts that are related to three sub-dimensions of catastrophizing: rumination, helplessness and magnification. Total scores for the PCS range from zero to 52, higher scores indicate a greater frequency of catastrophic thoughts. Previous research has shown that the PCS has good internal reliability ($\alpha = 0.87$) (Sullivan, et al., 1995) and that it is a significant predictor of negative pain outcomes (Sullivan, et al., 2001).

Fear of movement. Fear of movement and (re-)injury was measured with the Tampa Scale of Kinesiophobia (TSK) (Roelofs, et al., 2004; Swinkels-Meewisse, et al., 2003). The TSK instructs participants to rate their level of agreement to 17 statements about pain, injury and physical activity. Total scores range from 17 to 68, where higher scores indicate greater fear of movement. The TSK has previously been shown to have good internal reliability ($\alpha = 0.77$) (Vlaeyen, et al., 1995a) and to be a significant predictor of pain-related disability (Leeuw, et al., 2007a).

Depression. The Beck Depression Inventory II (BDI) (Beck, et al., 1996) was used to quantify the severity of depressive symptoms. The BDI instructs participants to endorse statements about their experience of depressive symptoms

during the two weeks preceding assessment. Total scores range from zero to 64, where higher scores indicate greater severity of depressive symptoms. The BDI has been shown to be a valid and reliable measure of depression in individuals with musculoskeletal pain conditions (Bishop, et al., 1993) and a significant predictor of pain-related outcomes (Boersma & Linton, 2006; Pincus & Newman, 2001).

Pain self-efficacy. The Pain Self-Efficacy Questionnaire (PSEQ) (Nicholas, 2007) was used to measure participants' self-confidence in their ability to perform activities of daily living despite the discomfort caused by their pain condition. The PSEQ consists of ten items that ask participants to rate their self-confidence in different life domains on a seven-point Likert-type scale. Total scores range from zero to 60 where low scores correspond to reduced pain self-efficacy. The PSEQ has been shown to predict pain-related outcomes in a wide range of pain populations (Ayre & Tyson, 2001; Foster, et al., 2010; Kall, 2009) and has been shown to have excellent internal reliability ($\alpha = 0.92$) (Nicholas, 2007).

One-year follow-up interview. A telephone interview was used to assess participants' levels of pain intensity, return to work status, healthcare utilization and medication use one-year after initial assessment. Current pain intensity was measured with a verbal 11-point numerical scale that corresponded to the numeric pain scale used at the pre- and post-treatment assessments (i.e. verbal anchors were set as 0 for "no pain" and 10 for "excruciating pain"). Participants were asked to identify whether they were currently working full-time (yes or no), and whether they were currently using any of the following healthcare services for their pain condition (yes or no for each service): physical therapy, psychology, massage therapy or medical services. Participants were also asked whether they were using any of the following medications for their pain condition: over-the-counter non-steroidal anti-inflammatories, opioids, prescription anti-inflammatories and psychotropics. No data regarding the frequency or quantity of healthcare utilization or medication use was collected.

Data Analysis

SPSS (version 18.0) was used to conduct all data analyses. Questionnaire items were totaled in a manner that was consistent with previous use of the scales. An index of healthcare utilization was created by dichotomously coding participants' use of each health service; "1" signified that the participant was presently using the service, "0" signified that the participant was not currently using the service. Summing the coded variables derived a total healthcare utilization score that ranged from zero to four where higher scores indicate greater use of different health services. An index of follow-up medication use was created in a similar manner; scores ranged from zero (no medication use at follow-up) to four (use of over-the-counter non-steroidal anti-inflammatories, opioids, prescription anti-inflammatories and psychotropics medications at follow-up) Descriptive statistics were conducted for sex, age, pain duration, location of injury, occupation, and assessment results.

Data analysis was conducted in three steps. First, zero-order relationships between sex, age, pain duration, pre-treatment medication use, post-treatment measures and one-year follow-up outcomes (pain intensity, return to work status, healthcare utilization and medication use) were examined. Correlational analysis was used for pain intensity, healthcare utilization and medication use, while mean comparisons (i.e. t-tests) were used for return to work. Only predictors that were significant in the zero-order analyses were used in the hierarchical regression analyses examining the prospective prediction of different pain-related outcomes.

The post-treatment assessment time point was selected as a predictor because it was considered to be the most pertinent assessment time-point in predicting long-term follow-up outcomes. Pre-treatment values, on the other hand, would not have been sensitive to potential changes associated with the seven weeks of treatment, and indices of treatment-related change may have been skewed by high or low pre-treatment scores.

Hierarchical linear and logistic regression analyses were conducted to determine whether psychological variables contributed unique variance to any of the four follow-up outcomes when controlling for significant baseline variables

and post-treatment pain intensity. To evaluate the predictions of the FAM, four hierarchical analyses were performed to determine the correlates of long-term pain intensity, work disability, healthcare utilization and medication use. Step 1 was used to control for post-treatment pain intensity and any baseline variables that were significant in the zero-order analyses. In Step 2, psychological factors from the FAM were entered as predictors. Finally, pain self-efficacy was entered in the third step of the analyses in an effort to replicate recent findings that suggest that this variable prospectively predicts pain-related outcomes beyond FAM factors (Foster, et al., 2010).

A final analysis was conducted to specifically evaluate the mediational relations proposed in the FAM. Baron and Kenny suggested that mediation be tested by first establishing the relationship between the independent variable and the potential mediator variable, and the relationship between these two variables and the outcome variable; in our study this step was conducted through the correlational and zero-order prospective analyses (Baron & Kenny, 1986). Next, using a hierarchical approach, the independent variable is entered as a predictor of the outcome variable, followed by the potential mediator variable; mediation is confirmed if the independent variable looses its significance after entering the mediator variable. To test this latter requirement, four hierarchical linear and logistic regression analyses were planned to determine whether (1) fear mediated the relationship between pain-catastrophizing and follow-up pain intensity; (2) fear mediated the relationship between pain-catastrophizing and follow-up return to work status; (3) depression mediated the relationship between fear and healthcare utilization; and (4) depression mediated the relationship between fear and medication use.

Logistic regression analysis was used for all hierarchical analyses that involved the dichotomous return to work outcome. Statistics that were used to report the results of the logistic regression analysis included the log-likelihood ratio, chi-square, Nagelkerke's R^2 statistic, the odds ratio, the regression coefficient (B) and the Wald statistic (Tabachnick & Fidell, 2007). The log-likelihood ratio statistic represents the goodness of the fit between the observed

and predicted models; smaller values indicate a better model fit than larger values. The chi-square statistic evaluates whether the model fit has been significantly improved after each hierarchical step, while the Nagelkerke's R^2 statistic is a measure of the model's effect size. The odds ratio represents the probability of a certain outcome (e.g. successful return to work) when the predictor variable has increased by one unit. The odds ratio is another indicator of effect size; the effect size increases as the odds ratio moves further from a value of 1. The B statistic represents the regression coefficient for each of the predictor variables, while the Wald statistic is used to determine whether each of the predictor variables is significant.

Results

Missing Data and Sample Characteristics

235 patients agreed to participate in the study, and completed pre-treatment assessments. However, 33 (14.04%) of these individuals did not complete either the post-treatment assessment, or the one-year follow-up assessment or both. Specifically, of the participants who had incomplete data, 23 (9.8% of total sample) did not have any post-treatment or follow-up data, 4 (1.7% of total sample) had complete post-treatment data but no follow-up data, and 6 (2.5% of total sample) had no post-treatment data but had complete follow-up data. Information regarding the cause of the missing data was not available. A summary of the data available for each of these groups is presented in Table S1 within Appendix 2.

Mean comparisons between participants that had complete data (N= 202) and those that had incomplete data (N = 33) were conducted on the following variables: sex, age, pain duration and initial levels of pain intensity, pain catastrophizing, fear of movement, depression and pain self-efficacy. All comparisons were non-significant ($p > 0.05$), with the exception of pain duration. Participants with complete data had pain significantly longer (mean = 8.63 weeks, SD = 3.35) than those with incomplete data (mean = 6.64 weeks, SD = 3.02; $p < 0.05$, $t_{1, 233} = 3.216$). Despite this difference, the two groups appear to be

homogenous, suggesting that there was not a significant biasing effect by removing them from the sample. Accordingly, the 33 cases with incomplete data were not used in the study sample.

The study sample therefore consisted of 202 individuals (123 women and 79 men) who, at the time of initial consultation, were not working as a result of a sub-acute musculoskeletal injury. Participants' mean age was 36.57 years, with a standard deviation (SD) of 10.34 years. The majority of participants had a post-secondary education, had been previously employed as a laborer or nurse, and identified the back or neck as the primary location(s) of pain. Initial levels of pain intensity and psychological variables were consistent with past studies that evaluated the role of psychological factors as predictors of pain-related outcomes for individuals with musculoskeletal injuries (Ayre & Tyson, 2001; Foster, et al., 2010; Woby, Urmston, & Watson, 2007). Table 1 shows the number and distribution for demographic, pre-treatment, post-treatment and follow-up variables.

Assessment Values and Cross-Sectional Correlations

Table 2 shows cross-sectional correlations between post-treatment variables. Consistent with past research, psychological variables and levels of pain intensity had significant inter-relations (Cook, et al., 2006; Goubert, et al., 2004). Pain catastrophizing was significantly correlated with fear of movement and depression, thus satisfying the first prerequisite of mediational testing.

Zero-Order Prospective Relationships

Zero-order correlations between age, sex, pain duration, pre-treatment medication use, post-treatment variables and continuous follow-up outcomes (i.e. pain intensity, healthcare utilization and medication use) are presented in Table 3. Analyses revealed significant relationships between pre-treatment opioid use and follow-up healthcare utilization. Follow-up medication use was significantly related to sex, pain duration and pre-treatment opioid use. Post-treatment pain intensity, FAM variables and pain self-efficacy were all significantly related to

follow-up pain intensity, health care utilization and medication use (Pearson r ranged from -0.568 to 0.557, all p values < 0.01).

Means, standard deviations and effects sizes on dependent measures for participants who returned to work and who did not return to work are shown in Table 4. Separate chi-square analyses revealed that sex and pre-treatment medication use were not significantly related to return to work status ($p < 0.05$). Post-treatment pain intensity ($t_{200} = 6.302$, $p < 0.01$), pain catastrophizing ($t_{200} = 5.780$, $p < 0.01$), fear of movement ($t_{200} = 5.224$, $p < 0.01$) and depression ($t_{200} = 5.249$, $p < 0.01$) were all significantly related to the likelihood of return to work at one-year follow-up.

Hierarchical, Prospective Relationships

Hierarchical regression analyses were conducted to evaluate 1) whether FAM variables and pain self-efficacy predicted pain intensity, return to work status, healthcare utilization and medication use measured at one-year follow-up; and 2) the degree to which FAM mediational predictions were supported. Results are organized by outcome variable.

Predicting pain intensity at one-year follow-up. Table 5 shows the results of the hierarchical multiple regression analysis with follow-up pain intensity as the dependent variable. Post-treatment pain intensity was entered in Step 1 of the analysis and was shown to be a significant predictor of follow-up pain intensity ($t = 13.173$, $p < 0.01$). In Step 2 of the analysis, post-treatment pain catastrophizing, fear of movement and depression were entered. Pain catastrophizing was the only factor from the FAM that was found to be a significant predictor of pain intensity at one-year follow-up ($t = 2.119$, $p < 0.05$). In Step 3 of the analysis, post-treatment pain self-efficacy was entered, but was not a significant predictor of follow-up pain intensity ($t = -0.442$, $p > 0.05$). After the final step of the analysis, 49.8% of the variance in follow-up pain intensity was accounted for and post-treatment pain intensity ($t = 6.734$, $p < 0.01$) and pain catastrophizing ($t = 2.076$, $p < 0.05$) were the only significant predictors.

The FAM posits that fear mediates the relationship between pain

catastrophizing and pain intensity. To evaluate these predictions a three-step, multiple regression analysis was conducted in which post-treatment pain intensity was entered in Step 1, post-treatment pain catastrophizing was entered in Step 2 and post-treatment fear of movement was entered in Step 3. The results of this analysis are shown in Table S2 within Appendix 2. Fear of movement failed to contribute any unique variance to pain intensity, while pain catastrophizing was a significant predictor ($t = 2.508$, $p < 0.05$); the FAM predictions regarding pain intensity were therefore not supported.

Predicting return to work at one-year follow-up. Table 6 shows the results of the hierarchical logistic regression analysis used to determine the predictors of return to work status at one-year follow-up. Similar to the previous analysis, post-treatment pain intensity was entered in Step 1, post-treatment FAM variables were entered in Step 2, and post-treatment pain self-efficacy was entered in Step 3. Fear of movement was the only FAM factor to significantly predict return to work status at one-year follow-up ($B = 0.061$, $p < 0.05$). Pain self-efficacy failed to contribute any unique variance to return to work ($B = 0.023$, $p > 0.05$).

Table 7 shows the results from the hierarchical logistic regression analysis that was used to test the FAM predictions that fear mediates the relationship between pain catastrophizing and disability. To evaluate this hypothesis, pain catastrophizing was entered as a predictor after controlling for pain intensity. Pain catastrophizing was shown to be a significant predictor of return to work status at one-year follow-up ($B = 0.042$, $p < 0.05$). However, in Step 3 of the analysis, fear of movement was shown to predict return to work status ($B = 0.063$, $p > 0.05$), while pain catastrophizing was non-significant ($B = 0.013$, $p > 0.05$). Thus, the FAM predictions regarding fear and pain-related disability were supported.

Predicting healthcare utilization at one-year follow-up. Table S3, within Appendix 2, shows the results of the hierarchical multiple regression analysis with healthcare utilization as the dependent variable. Zero-order correlational analysis revealed a significant relationship between pre-treatment opioid use and healthcare utilization. Accordingly, this variable was entered in the first step of

the hierarchical regression and was shown to be a significant predictor ($t = 2.926$, $p < 0.01$). In Step 2, post-treatment pain intensity was found to be a significant predictor ($t = 2.671$, $p < 0.01$). In Steps 3 and 4, FAM variables and pain self-efficacy failed to achieve significance. After the final step of the analysis, 18.9% of the variance in follow-up healthcare utilization was accounted for, and pre-treatment opioid use ($t = 4.224$, $p < 0.01$) was the sole significant predictor.

To rule out the possibility that participants' use of medical services (as opposed to the utilization of health services in general) may have inflated the relationship between opioid use and healthcare utilization, the above analysis was repeated using a revised healthcare utilization variable that excluded medical services (i.e. only use of physical therapy, psychology and massage therapy were used in the revised index). The same results were reproduced with the revised index (i.e. opioid use was the only significant predictor; $t = 3.115$, $p < 0.01$). Thus, no further mediational analysis was conducted, and our predictions regarding depression and healthcare utilization were not supported.

Predicting medication use at one-year follow-up. Table 8 shows the results of the hierarchical multiple regression analysis with medication use as the dependent variable. Based on zero order correlations, sex, pain duration and pre-treatment opioid use were entered in Step 1. Sex ($t = -2.092$, $p < 0.05$) and pre-treatment opioid use ($t = 14.496$, $p < 0.05$) were both significant predictors of follow-up medication use. Pain intensity, entered in Step 2 of the analysis, was also shown to be a significant predictor ($t = 3.659$, $p < 0.05$). None of the FAM variables entered in Step 3, however, attained significance. Pain self-efficacy was entered in Step 4 of the model and was found to be a significant predictor of follow-up medication use ($t = -2.375$, $p < 0.05$). In the final model, 59.3% of the variance in follow-up medication use was explained and pain self-efficacy was the sole significant psychosocial predictor. Our predictions regarding levels of depression and medication use were therefore not supported, and no further mediational analysis was conducted.

Discussion

Our findings add to the growing body of research suggesting that psychological factors, measured in the sub-acute or early chronic period following musculoskeletal injury, are predictors of long-term pain-related outcomes (Pincus, et al., 2002; Storheim, Brox, Holm, & Bø, 2005). Consistent with previous research, we have found significant zero-order prospective relationships between psychological factors and pain intensity, return to work status, healthcare utilization and medication use (Boersma & Linton, 2006; Gheldof, et al., 2010; Pincus, et al., 2002; Wideman, et al., 2009). Our findings extend previous research by showing that pain catastrophizing, fear of movement and pain self-efficacy act as differential predictors of long-term pain-related outcomes. Specifically, our results suggest unique relationships between pain catastrophizing and long-term pain intensity; fear of movement and long-term work disability; and between pain self-efficacy and long-term medication use. To the best of our knowledge, this was the first prospective study to compare all of the psychological factors from the FAM in their ability to differentially predict four model-relevant outcomes.

While our findings provide general support for the importance of the psychological variables in the FAM, they only provide partial support for the specific FAM predictions that were tested. The FAM suggests that pain-related fear is the common psychological conduit through which several pain-related outcomes occur (Leeuw, et al., 2007a; Vlaeyen & Linton, 2000). However, results from our hierarchical analyses show that while fear of movement was a unique predictor of work disability, it did not contribute unique variance to pain intensity, healthcare utilization or medication use. Consistent with recent research, and with our predictions, these findings suggest that fear may have a more influential role in determining levels of pain-related disability, than in predicting other model-relevant outcomes (Gheldof, et al., 2010; Sullivan, et al., 2009).

Our results suggest that pain catastrophizing may play a central role in determining both pain and pain-related disability. Results from our analyses show that pain catastrophizing was uniquely related to long-term pain intensity. The impact of catastrophizing on pain outcomes has been discussed from both

psychological and neurophysiological perspectives (Quartana, Campbell, & Edwards, 2009; Sullivan, et al., 2001). For example it has been suggested that catastrophizing might exert its impact on pain through appraisal or coping processes (Sullivan, et al., 2001). It has also been suggested that catastrophizing might impact more directly on pain via its influence on descending pain modulation (Quartana, et al., 2009). A growing body of evidence suggests that elevated levels of pain catastrophizing are related to changes in activation of a number of cortical areas involved in the processing of nociceptive input and to dysfunction of the endogenous-opioid system (Campbell & Edwards, 2009; Goodin, et al., 2009; Quartana, et al., 2009; Seminowicz & Davis, 2006). Emerging research suggests that catastrophizing might be a complex variable that impacts on the pain experience through distinct psychological and neurophysiological pathways. To characterize catastrophizing as only a cognitive antecedent of fear of movement might represent an oversimplification.

Results from our analyses are not consistent with recent findings that pain self-efficacy supersedes the ability of FAM variables to predict levels of disability. Recently, Foster et al. conducted a large prospective study in which the predictive value of pain self-efficacy was compared to other psychological factors, including pain catastrophizing, fear of movement and depression (Foster, et al., 2010). Results from their analysis showed that pain self-efficacy, but not FAM variables, predicted pain-related disability. Based on these findings the authors suggest that FAM variables are rendered redundant when compared to the predictive utility of pain self-efficacy. In contrast to this study, however, our results show that pain self-efficacy only contributed unique variance to follow-up medication use, and that pain catastrophizing, fear of movement and pain self-efficacy have distinct relations with different pain-related outcomes.

One reason for this discrepancy may be attributed to the fact that Foster et al. used a self-report questionnaire to measure disability, while we assessed return to work status. In keeping with our findings, past research that has evaluated self-efficacy as a predictor of work disability or objective measures of function suggest a weaker, and less consistent, link between these factors (Lacaille, Sheps,

Spinelli, Chalmers, & Esdaile, 2004; Schiphorst Preuper, et al., 2008; Watson, Booker, & Maas, 1997). Differences in study populations may have also contributed to these differences. For example, Foster et al. used a study sample of primary care patients, while our sample consisted of workers' compensation claimants. Another possible cause for the divergence between our results is that in the present analyses, we controlled for pain intensity, whereas Foster et al. only controlled for the duration and location of pain. Results from our zero-order analyses revealed that, in comparison to all other factors, post-treatment pain intensity was the strongest predictor of follow-up pain symptoms and work disability, while pain duration only demonstrated a weak correlation with follow-up medication use and was not significantly related to any other outcomes. In light of these findings, it is possible that had Foster and her colleagues controlled for pain intensity in their study, the predictive value of pain self-efficacy may have been reduced. Together, these results highlight the importance of considering different pain-related outcomes and controlling for pain intensity when assessing the redundancy of different psychological predictors.

Our finding that pain self-efficacy is an important predictor of medication use, however, is consistent with past research. Nicholas (Nicholas, 2007), for example, reports that levels of pain self-efficacy are correlated with pain medication use at a four-year, post-treatment follow-up. The strong relationship between pain self-efficacy and long-term medication use may be explained by an item in this scale that specifically addresses patients' perceived ability to cope with their pain without using medication. None of the other FAM measures that were used in our study explicitly addressed medication use.

However, it is also important to consider that in early factor analytic studies, that measures of self-efficacy and pain catastrophizing have loaded on the same factor (Sullivan, et al., 2001). These data suggest that catastrophizing and self-efficacy are very closely related and might even represent different poles of the same underlying dimension. With a high degree of shared variance, it is unlikely that, in any study, both will emerge as significant predictors of outcomes. Whether self-efficacy or catastrophizing wins a predictive race might simply be

the result of chance variations in the distribution of scores; which is likely to differ from one study to the next. Further research is required to elucidate the processes contributing to the overlap between measures of pain catastrophizing and pain self-efficacy and to shed further light on the unique importance of these two predictors.

Results from our study also have important implications for the clinical management of pain conditions. Our findings suggest that individuals with elevated levels of catastrophizing, fear of movement and low levels of self-efficacy following a course of rehabilitation for sub-acute musculoskeletal pain are at risk of developing long-term health complications. Previous research has identified the sub-acute and early chronic phases as critical periods for interventions to prevent the development of prolonged pain conditions (Karjalainen, et al., 2008; Schultz, et al., 2008; Sullivan, Feuerstein, Gatchel, Linton, & Pransky, 2005a). Our findings that pain catastrophizing was linked to both levels of pain and pain-related disability, suggest that reductions in this variable may be the most efficient method of preventing negative long-term outcomes. Previous research suggests that a wide range of interventions can lead to reductions in pain catastrophizing (Morley, et al., 2008; Moseley, Nicholas, & Hodges, 2004; Smeets, et al., 2006a; Sullivan, et al., 2006b). However, the most cost-effective means of reducing pain catastrophizing remains to be identified. Further research is therefore needed to systematically compare the efficacy of different interventions that target pain catastrophizing. Future research will also need to explore whether interventions that reduce patients' levels of pain catastrophizing are also effective in increasing levels of pain self-efficacy.

Some caution should be exercised in generalizing findings from this research. First, readers are reminded that our study design does not permit any conclusions regarding causality. Also, return to work, healthcare utilization and medication use are outcomes that are influenced by numerous social factors that were not considered in our analysis. For example, it is possible that in controlling for economic factors or access to health services, the psychological variables included in our study would not have been significant predictors of these

outcomes. Furthermore, the physical therapy interventions used in our study were not standardized. While the physical therapy treatment was considered to be comparable across participants, it is possible that differences between clinicians may have influenced our results.

Despite these limitations, this was the first prospective study to compare all of the psychological factors from the FAM in their ability to differentially predict four model-relevant outcomes; accordingly, it contributes several novel findings and theoretical implications to this area of research. Our findings suggest that psychological factors in the FAM have differential relations with long-term, pain-related outcomes. Specifically, we found direct relationships between pain catastrophizing and pain intensity, fear of movement and work disability, and between pain self-efficacy and medication use. Through our mediational analyses, we also found that levels of fear of movement mediate the relationship between pain catastrophizing and disability, but not the relationship between pain catastrophizing and pain intensity. Our findings lend partial support for FAM predictions and suggest that psychological factors have unique predictive utility in determining long-term pain-related outcomes.

Acknowledgements

This research was supported by funds from the Canadian Institutes for Health Research, Fonds de la recherche en santé du Québec (FRSQ), the Institut de recherche Robert-Sauvé en santé et en securité du travail (IRSST) and by the Physiotherapy Foundation of Canada through the Ann Collins Whitmore Memorial Scholarship. The authors have no financial interest in the results of this research.

Tables

Table 1

Characteristics of the study sample (N = 202).

Characteristics	N (%) or Mean (SD)
Education	
Less than high school	35 (17.3)
High school	58 (28.7)
Trade school	40 (19.8)
College	47 (23.3)
University	22 (10.9)
Occupation	
Laborer	65 (32.2)
Nursing	48 (23.8)
Clerical	39 (19.3)
Trade	21 (10.4)
Driving	13 (6.4)
Sales	13 (6.4)
Other	3 (1.5)
Injury site (categories are not mutually exclusive)	
Back	187 (92.6)
Neck	162 (80.2)
Upper extremity	115 (56.9)
Lower extremity	52 (25.7)
Pre-treatment assessment variables	
Pain intensity	5.1 (1.8)
PCS	21.5 (10.7)
TSK	42.8 (8.1)
BDI	15.1 (9.4)
PSEQ	34.4 (11.8)
Post-treatment assessment variables	
Pain intensity	3.8 (2.1)
PCS	12.9 (11.1)
TSK	37.5 (8.8)

BDI		10.8 (9.1)
PSEQ		42.7 (13.3)
One-year follow-up variables		
Pain intensity		3.6 (2.3)
Returned-to-work		139 (68.8)
Healthcare utilization		1.31 (0.5)
Number of services used		
	0	123 (60.9)
	1	49 (24.3)
	2	17 (8.4)
	3	5 (2.5)
	4	8 (4)
Types of services used		
	Physical therapy	59 (29.2)
	Medical treatment	31 (15.3)
	Psychology	26 (12.9)
	Massage therapy	14 (6.9)
Medication use		0.9 (1.0)
Number of medications used		
	0	88 (43.6)
	1	64 (31.7)
	2	38 (18.8)
	3	6 (3)
	4	6 (3)
Types of medications used		
	OTC NSAIDs	90 (44.6)
	Opioids	49 (24.3)
	Anti-inflammatories	19 (9.4)
	Psychotropics	24 (11.9)

SD = Standard deviation; PCS = Pain catastrophizing scale; TSK = Tampa scale of kinesiophobia; BDI = Beck depression Inventory; PSEQ = Pain self-efficacy questionnaire; OTC NSAID = Over-The-Counter Non Steroidal Anti-Inflammatory Drugs

Table 2

Correlations between post-treatment variables.

Variables	PT pain intensity	PT PCS	PT TSK	PT BDI
PT PCS	0.612**			
PT TSK	0.376**	0.637**		
PT BDI	0.508**	0.650**	0.461**	
PT PSEQ	-0.722**	-0.671**	-0.595**	-0.624**

PCS = Pain catastrophizing scale; TSK = Tampa scale of kinesiophobia; BDI = Beck depression Inventory; PSEQ = Pain self-efficacy questionnaire; PT = Post-treatment; $^{**}p < 0.01$ (2-tailed).

Table 3

Correlations between demographic, post-treatment and one-year follow-up variables.

Variables	FU Pain Intensity	FU Healthcare Utilization	FU Medication Use
Age	0.033	-0.054	-0.114
Sex	-0.024	-0.109	-0.184**
Pain duration	0.128	0.007	0.186**
Pre-treatment OTC NSAID Use	-0.002	0.022	0.045
Pre-treatment opioid use	0.081	0.203**	0.163*
Pre-treatment anti-inflammatory Use	-0.097	0.006	0.067
PT pain intensity	0.682**	0.258**	0.362**
PT PCS	0.557**	0.289**	0.375**
PT TSK	0.376**	0.191**	0.217**
PT BDI	0.442**	0.306**	0.396**
PT PSEQ	-0.568**	-0.236**	-0.412**

OTC NSAID = Over-The-Counter Non Steroidal Anti-Inflammatory drugs; PCS = Pain catastrophizing scale; TSK = Tampa scale of kinesiophobia; BDI = Beck depression Inventory; PSEQ = Pain self-efficacy questionnaire; PT = Post-treatment; FU = Follow-up; $^{**}p < 0.01$ (2-tailed).

Table 4

Means, standard deviations and effect sizes of age, pain duration and post-treatment variables for participants that did, and did not, return to work.

Variables	Returned-to-work M (SD) N = 139	Did not return to work M (SD) N = 63	t statistic (Cohen's d)
Age	36.57 (10.343)	36.81 (9.382)	0.158 (0.024)
Pain duration	8.324 (3.397)	9.317 (3.161)	1.967 (0.303)
PT pain intensity	3.190 (1.859)	5.040 (2.095)	6.302 (0.934)**
PT PCS	10.129 (9.266)	19.190 (12.351)	5.780 (0.830)**
PT TSK	35.482 (8.613)	42.048 (7.467)	5.224 (0.815)**
PT BDI	8.705 (7.555)	15.524 (10.437)	5.249 (0.748)**
PT PSEQ	45.878 (11.568)	35.825 (14.239)	-5.313 (-0.775)**

PCS = Pain catastrophizing scale; TSK = Tampa Scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; PT = Post-treatment; $^{**}p < 0.01$.

Table 5

Hierarchical linear regression analysis: predictors of pain intensity at one-year follow-up.

	Variables	t	Beta	R^2	R^2 Change	F change
Step 1	PT pain intensity					
		13.173**	0.682	0.465	0.465	173.527**
Step 2						
	PT PCS	2.119*	0.177			
	PT TSK	0.692	0.046			
	PT BDI	0.456	0.031	0.498	0.033	4.337**
Step 3						
	PT PSEQ	-0.442	-0.040	0.498	0.001	0.195

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table 6

Hierarchical logistic regression analysis: predictors of return to work at one-year follow-up

	Variables	$\Delta\chi^2$	Δdf	R^2	-2LL	B	Wald	OR	95% CI
Step 1									
	PT pain intensity	35.046**	1	0.224	215.676	0.470	27.814**	1.599	1.343 – 1.904
Step 2									
	PT PCS					0.000	0.000	1.000	0.954 – 1.048
	PT TSK					0.061	5.587*	1.063	1.010 – 1.118
	PT BDI	13.324**	3	0.300	202.352	0.032	1.848	1.033	0.986 – 1.082
Step 3									
	PT PSEQ	1.049	1	0.305	201.303	0.023	1.032	1.023	0.949 – 1.068

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; -2LL = Log Likelihood Ratio; B = regression coefficient; OR = Odds Ratio; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table 7

Hierarchical logistic regression analysis: evaluation of fear of movement as a mediator between pain catastrophizing and return to work at one-year follow-up.

	Variables	$\Delta\chi^2$	Δdf	R^2	-2LL	B	Wald	OR	95% CI
Step 1									
	PT pain intensity	35.046**	1	0.224	215.676	0.470	27.814**	1.599	1.343 – 1.904
Step 2									
	PT PCS	5.215*	1	0.254	210.461	0.042	5.091*	1.042	1.005 – 1.081
Step 3									
	PT PCS	–	–	–	–	0.013	0.337	1.013	0.970 – 1.057
	PT TSK	6.262*	1	0.289	204.198	0.063	6.094*	1.066	1.013 – 1.121

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; -2LL = Log Likelihood Ratio; B = regression coefficient; OR = Odds Ratio; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table 8

Hierarchical linear regression analysis: predictors of medication use at one-year follow-up.

	Variables	t	Beta	R^2	R^2 Change	F change
Step 1	Sex	-2.092*	-0.103			
	Pain Duration	1.021	0.051			
	Pre-treatment opioid use	14.496**	0.708	0.542	0.542	78.151**
Step 2						
	PT pain intensity	3.659**	0.178	0.571	0.029	13.391**
Step 3						
	PT PCS	1.169	0.091			
	PT TSK	0.200	0.013			
	PT BDI	0.653	0.043	0.581	0.010	1.481
Step 4						
	PT PSEQ	-2.375*	-0.198	0.593	0.012	5.639*

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; $^{*}p < 0.05$; $^{**}p < 0.01$.

PREFACE TO CHAPTER 4

Findings from Study 2 suggest that, contrary to model predictions, pain-related fear is not a common psychological conduit for multiple pain-related outcomes. Consistent with findings from Study 1, these results also suggest that model-relevant psychosocial factors function more independently than predicted by the Fear Avoidance Model. When considered together, results from Study 1 and Study 2 raise doubts about the validity of the inter-relationships proposed within the Fear Avoidance Model; these questions are explored in the general discussion.

Results from these studies also suggest that model-relevant psychosocial risk factors may interact through alternate relationships, which have not been proposed by the Fear Avoidance Model. One alternative to the proposed sequential and mediational relationships of the Fear Avoidance Model is that different model-relevant risk factors relate to one another in a cumulative fashion. For instance, contrary to model predictions but consistent with our previous results, pain catastrophizing, fear of movement, and depression may tap independent risk constructs. If this were the case, then individuals with elevated scores on multiple psychosocial risk factors would have a worse prognosis for recovery than individuals with elevated scores on only one model-relevant risk factor. Study 3 explored these hypothesized relationships within a sample of patients with musculoskeletal pain. When contextualized with the previous results, support for such cumulative relationships would have further negative implications for our assessment of the Fear Avoidance Model.

CHAPTER 4: DEVELOPMENT OF A CUMULATIVE PSYCHOSOCIAL FACTOR INDEX FOR PROBLEMATIC RECOVERY FOLLOWING WORK-RELATED MUSCULOSKELETAL INJURIES

Wideman TH & Sullivan MJL. Physical Therapy 2012; 92: 58-68.[7]

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Abstract

Psychosocial variables such as fear of movement, depression, and pain catastrophizing have been shown to be important prognostic factors for a wide range of pain-related outcomes. The potential for a cumulative relationship between different elevated psychosocial factors and problematic recovery following physical therapy has not been fully explored. This prospective cohort study aimed to determine whether the level of risk for problematic recovery following work-related injuries is associated with the number of elevated psychosocial factors. 202 individuals with sub-acute, work-related musculoskeletal injuries completed a seven-week physical therapy intervention and participated in testing at treatment onset and one-year later. An index of psychosocial risk was created from measures of fear of movement, depression and pain catastrophizing. This index was used to predict the likelihood of experiencing problematic recovery in reference to pain intensity and return to work status at one-year follow-up. Logistic regression analysis revealed that the number of prognostic factors was a significant predictor of persistent pain and work disability at one-year follow-up. Chi-square analysis revealed that the risk for problematic recovery increased for patients with elevated levels on at least one psychosocial factor, and was highest when patients had elevated scores on all three psychosocial factors. The number of elevated psychosocial factors present in the sub-acute phase has a cumulative effect on the level of risk for problematic recovery one-year later. This research suggests that a cumulative prognostic factor index could be used in clinical settings to improve prognostic accuracy and to facilitate clinical decision-making.

Introduction

Work-related musculoskeletal injuries are a leading cause of prolonged pain and occupational disability. Clinical practice guidelines commonly recommend activity-based interventions, such as physical therapy, for individuals with sub-acute musculoskeletal pain conditions (Airaksinen, et al., 2006; Chou & Huffman, 2007; Koes, et al., 2010; Sanders, Harden, & Vicente, 2005). While most individuals with these conditions make a full recovery, an estimated 10 to 20% will go on to develop chronic pain and disability (Baldwin, 2004). This relatively small but significant group accounts for the large majority of the workers' compensation expenditures that are associated with these conditions (Baldwin, 2004). Previous research suggests that psychosocial factors measured in the early stages of recovery can help predict patients' prognoses for long-term rehabilitation (Boersma & Linton, 2005b; Fritz & George, 2002; Fritz, George, & Delitto, 2001; George, Fritz, & Childs, 2008).

Factors such as fear of movement, depression, and pain catastrophizing have been shown to be important predictors of a wide range of rehabilitation and work-related outcomes. For example, previous research has shown that these psychosocial factors prospectively predict measures of pain severity, physical function, and return to work status, even after controlling for baseline levels of pain (Bair, Robinson, Katon, & Kroenke, 2003; Leeuw, et al., 2007a; Quartana, et al., 2009; Sullivan, Reesor, Mikail, & Fisher, 1992). Related research has shown that these prognostic factors are modifiable, and that their treatment-related reduction is associated with improved rehabilitation outcomes (Burns, et al., 2003b; George, Fritz, & McNeil, 2006; Sullivan, et al., 2006b; Vlaeyen, et al., 2002; Wideman, et al., 2009). In response to these findings, there have been calls in the literature to address psychosocial factors in physical therapy practice (Calley, Jackson, Collins, & George, 2010; George, et al., 2011; George, et al., 2008; Haggman, Maher, & Refshauge, 2004; Sullivan, et al., 2006b).

Despite the established clinical importance of different psychosocial factors, there is a lack of research that facilitates the clinical interpretation of these measures by physical therapists. One challenge is that previous research exploring

the clinical implications of psychosocial factors has typically reported results in the form of regression coefficients. While high in statistical and theoretical utility, regression coefficients are relatively low in clinical utility. For instance, previous reports that have used regression coefficients alone have not provided adequate information about the clinical implications of patients with a varying number of elevated scores on different psychosocial factors. For the practicing clinician, it therefore remains a challenge to understand the prognostic implications of patients who have elevated scores on more than one psychosocial factor.

To date, there is only limited research exploring the cumulative relationship among different psychosocial factors. Recent findings suggest that different psychosocial factors might have an additive effect on patients' prognoses for problematic outcomes (Linton, et al., 2011). For instance, a recent study suggests that physical therapy patients with high scores on measures of both pain catastrophizing and depression are more likely to have a problematic recovery than individuals with elevated scores on just one measure (Bergbom, Boersma, Overmeer, & Linton, 2011). This research suggests that information regarding the *number* of elevated psychosocial factors may help physical therapists better determine their patients' prognoses for recovery. This line of research, however, remains in its infancy. For instance, previous research exploring the cumulative effect of different psychosocial factors among physical therapy patients has yet to consider the effects of fear of movement. There is a compelling body of research in the field of physical therapy linking pain-related fear to a wide variety of clinical outcomes (George, Dover, & Fillingim, 2007; Lentz, Barabas, Day, Bishop, & George, 2009; Swinkels-Meewisse, et al., 2006; Thomas & France, 2007). Also, the Fear Avoidance Model (FAM) of pain suggests that, in addition to pain catastrophizing and depression, fear of movement is an important determinant of prolonged pain and pain-related disability (Leeuw, et al., 2007a); further research exploring the cumulative relationship among these three psychosocial factors may help guide physical therapists in the clinical management of patients suffering from pain conditions (Bergbom, et al., 2011).

The purpose of this study was to determine whether the risk for problematic recovery following work-related injuries varies as a function of the number of elevated psychosocial factors. To address this question we conducted a prospective, cohort study using a sample of individuals with sub-acute, work-related musculoskeletal injuries. Study participants completed a seven-week physical therapy intervention and participated in testing at treatment onset and one-year later. An index of cumulative prognostic factors was created from previously validated measures of fear of movement, depression and pain catastrophizing. This index was used to predict the likelihood of experiencing problematic recovery in reference to patients' pain intensity and return to work status at one-year follow-up. We hypothesized that patients with higher numbers of elevated psychosocial factors would be more likely to experience problematic outcomes at one-year follow-up.

Methods

Study Design

A prospective cohort study was conducted in which a convenience sample of patients was recruited from six physical therapy clinics across the province of Quebec, Canada.

Inclusion Criteria

Patients were eligible for the study if they were between the ages of 18 and 65 years, and had sustained a work-related, soft-tissue injury to their back or neck. At the time of initial consultation, all patients were in the sub-acute phase of recovery (i.e. three to 12 weeks since injury), and were receiving wage indemnity benefits from the provincial workers' compensation board. Patients were not eligible for the study if they had been diagnosed with a vertebral fracture, disc herniation, ankylosing spondilitis, infectious disease or any medical condition that did not permit a physical evaluation. This research program was approved by the ethics review boards of the *Centre de recherche interdisciplinaire en réadaptation du Montréal métropolitain* and the *Université de Montréal*.

Procedure

Interested and eligible patients were first asked to sign an informed consent form. Self-report questionnaires were administered at the onset of physical therapy and after completing seven weeks of treatment. Baseline and demographic information was also collected at treatment onset. A follow-up telephone interview was conducted one year following treatment onset.

Physical therapy interventions were aimed at reducing pain and disability associated with soft-tissue injuries. Specific interventions varied at the discretion of the treating therapist, however, treatments were consistent with clinical practice guidelines for functional restoration after a sub-acute musculoskeletal injury and the reimbursement guidelines of the provincial workers' compensation board (Airaksinen, et al., 2006; Chou, et al., 2007; Panel, 2001). Treatment therefore focused on early mobilization and physical activity, and primarily consisted of range of motion, joint manipulation, and progressive strength exercises. Physical therapy sessions were scheduled three days per week.

Measures

Participants completed self-report measures of pain intensity, pain catastrophizing, fear of movement and depressive symptoms. Participants also provided baseline information relating to their age, sex, pre-injury occupation, highest level of education, location of injury, time since injury, and use of medication for their pain condition. The one-year follow-up interview evaluated participants' levels of pain intensity and return to work status.

Pain intensity. Numeric rating scales were used to quantify participants' levels of pain severity. Participants were asked to rate their pain intensity on an 11-point scale with end-point anchors of 0 (no pain) and 10 (excruciating pain). Past research suggests that such scales are reliable and valid measures of pain intensity (Downie, et al., 1978).

Pain catastrophizing. The Pain Catastrophizing Scale (PCS) was used to quantify participants' levels of catastrophic thoughts. The PCS is a 13-item self-

report questionnaire that includes items to measure each of the three sub-dimensions of pain catastrophizing (rumination, magnification and helplessness). Higher scores on the PCS indicate greater levels of catastrophic thinking; past research has used a cut-off score of 20 to identify patients with elevated scores of pain catastrophizing (Adams, Ellis, Stanish, & Sullivan, 2007; Wideman, et al., 2009). Previous research has found the PCS to have good reliability and validity, and that elevated scores indicate risk for poor pain-related outcomes (Sullivan, et al., 1995; Sullivan, et al., 2001).

Fear of movement. Fear of movement and re-injury was measured via the Tampa Scale of Kinesiophobia (TSK) (Roelofs, et al., 2004; Swinkels-Meewisse, et al., 2003). The TSK is a self-report questionnaire that consists of 17 items that address beliefs relating to pain, movement and injury. Participants are asked to rate their level of agreement for each of the items; higher scores indicate greater levels of fear of movement. Past research has found the TSK to have good reliability and to be an important predictor of pain-related disability (Leeuw, et al., 2007a). Previous research has used a cut-off score of 39 to identify individuals with elevated levels of fear of movement (Adams, et al., 2007; Wideman, et al., 2009).

Depression. The Beck Depression Inventory II (BDI) was used to measure the severity of participants' depressive symptoms (Beck, et al., 1996). Elevated BDI scores are an indication of more severe depressive symptoms, and have been shown to be an important predictor of negative pain-related outcomes (Boersma & Linton, 2006; Pincus & Newman, 2001). Previous research has identified a cut-off score of 13 to identify patients with clinically meaningful symptoms of depression (Harris & D'Eon, 2008). Past research in the field of pain suggests that the BDI is a reliable and valid measure of depressive symptoms (Bishop, et al., 1993).

One-year follow-up interview. One year after initial assessment, a telephone interview was used to assess participants' levels of pain intensity and return to work status. Pain intensity was measured using an 11-point numeric rating scale that had the same anchors as the previously described measure (i.e. 0

for “no pain”, 10 for “excruciating pain”). Return to work status was based on whether patients had, or had not, returned to full-time employment (yes/no).[8]

Data Analysis

Scores on the PCS, TSK and BDI were split on previously established cut-off scores (i.e. 20, 39, and 13 respectively), and participants were coded as either having, or not having, elevated scores on each scale (Adams, et al., 2007; Harris & D'Eon, 2008; Wideman, et al., 2009). Using this data, a variable named *number of prognostic factors* was created; *prognostic factor* in this study is used to indicate an elevated score on a psychosocial measure. This variable ranged from zero (i.e. below, or equal to, the cut-off for all scales) to three (i.e. above the cut-off for all scales).

The Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) advisory board has recommended a 30% reduction as cut-off criteria for clinically meaningful change in pain intensity (Dworkin, et al., 2008). Percent reductions in pain intensity were calculated from initial assessment to one-year follow-up and, using a 30% reduction as a cut-off, participants were coded as experiencing either a *reduction in pain* or *no reduction in pain*.

Using SPSS version 18.0, data analysis was conducted in three steps. First, zero-order mean comparisons were used to determine the relationships between pre-treatment variables and one-year follow-up outcomes. Next, hierarchical logistic regression analysis was used to determine whether number of prognostic factors was a significant predictor of each outcome while controlling for all other factors that were significant in the zero-order analysis. Finally, chi-square analysis was conducted to determine the relationship between the number of prognostic factors and the rates of problematic recovery.

Results

[8] In the province in which testing occurred, employers are required by law to provide injured workers with the opportunity to return to their pre-injury occupation. As a result, return-to-work status in this study can be construed as a measure of participants’ ability to *return* to their previous employment, rather than their ability to *find* new employment.

Missing Data

235 individuals agreed to participate in the study and completed the pre-treatment assessment. Of this group, however, 33 (14.0%) did not have complete follow-up data. Mean comparisons were conducted to determine whether individuals with complete and incomplete data differed with respect to their sex, age, pain duration, and their pre-treatment levels of medication use, pain intensity, pain catastrophizing, fear of movement or depression. Results showed a significant between-group difference in pain duration; individuals with complete data had an average pain duration of 8.63 weeks (Standard Deviation (SD) = 3.35 weeks), while individuals with incomplete data averaged 6.64 (SD = 3.02) weeks since their injury. Aside from this difference the groups appeared homogenous; the 33 individuals with incomplete data were therefore not included in the study sample.

Previous findings suggest that psychosocial prognostic factors are not strongly related to self-reported location of pain (George, et al., 2011; George, Fritz, & Erhard, 2001). To ensure that this was the case in our sample and that patients with back and/or neck pain were homogenous, we conducted an analysis that compared baseline and pre-treatment variables for individuals with (N = 187) and without (N = 15) back pain; all comparisons were non-significant ($p < 0.05$). The study sample (N = 202) was therefore considered to be homogenous with respect to self-reported location of pain.

Sample Characteristics

The study sample consisted of 202 individuals (79 men and 123 women), with a mean age of 36.57 years (SD = 10.34 years). Table 1 presents participants' level of education; pre-injury occupation; location of injury; and the distribution on their pre-treatment and follow-up assessments. To summarize, the majority of participants completed some form of post-secondary education and, prior to their injury, were working as either a laborer or nurse. The majority of participants identified their back as the primary location of pain. One-year following initial

assessment 106 (52.5%) did not experience a meaningful reduction in their pain intensity, and 63 (31.2%) participants had not returned-to-work.

Correlation analyses between participants' pre-treatment scores on the psychosocial factors are presented in Table 2. Consistent with previous findings (Cook, et al., 2006; Goubert, et al., 2004; Vlaeyen & Linton, 2000), our results revealed significant inter-correlations between all factors that are in the modest to moderate range ($p < 0.001$; Pearson coefficients ranged from .288 to .570).

Mean comparisons between scores on psychosocial prognostic factors and the follow-up outcomes are presented in Table 3. Pre-treatment levels of pain catastrophizing and depression were significantly related to both of the outcomes ($p < 0.05$), while relationships with levels of fear of movement were marginally significant.

Predicting Reductions in Pain Intensity at One-Year Follow-Up

First, zero-order analyses were conducted to determine whether age, sex, pain duration, pre-treatment medication use, pre-treatment pain intensity and number of prognostic factors were significantly related to follow-up reductions in pain intensity. Results revealed that the number of prognostic factors ($F = 11.279$, $\eta^2 = 0.053$, $p = 0.001$) was the only variable to have a significant relationship with reduction in pain intensity. As a result, no hierarchical testing was required.

In order to ensure that our categorization of patients' reduction in pain intensity (i.e. using a 30% reduction cut-score) had not influenced the predictive value of the number of prognostic factors, we repeated this analysis using two continuous dependent variables: pain intensity at one-year follow-up and the percent reduction in pain intensity from pre-treatment to one-year follow-up. Similar to our initial findings both analyses showed that after controlling for other baseline factors, that the number of prognostic factors was a significant predictor of pain-related measures at follow-up ($p < 0.05$).

Next, chi-square analysis was conducted to determine whether the likelihood of failing to achieve a meaningful reduction in pain intensity increased with the number of prognostic factors; results confirmed this relationship ($\chi_3^2 =$

14.947, $p = 0.002$). The likelihood of failing to achieve a reduction in pain for participants with zero, one, two and three prognostic factors was 23.5%, 55.6%, 54.7% and 63.9%, respectively.

Table 4 presents a summary of how participants with different numbers of prognostic factors are distributed across both outcomes; Figure 1 presents this data within a graph. Contrast comparisons revealed significant differences in the likelihood of pain reduction between individuals with zero prognostic factors and those with one prognostic factor ($\chi_1^2 = 8.722$, p = 0.003, Phi = -0.315); with zero prognostic factors and those two prognostic factors ($\chi_1^2 = 8.243$, p = 0.004, Phi = -0.308); and between individuals with zero prognostic factors and those with three prognostic factors ($\chi_1^2 = 14.258$, $p < 0.001$, Phi = -0.387). Results of contrast comparisons for pain reduction and return to work status are presented in Table 5.

Predicting Return to Work Status at One-Year Follow-Up

Zero-order analysis revealed significant findings for pre-treatment pain intensity (F = 4.532, $\eta^2 = 0.022$, p = 0.034) and number of prognostic factors (F = 13.245, $\eta^2 = 0.062$, $p < 0.001$). Hierarchical logistic regression analysis revealed that the number of prognostic factors predicted return to work status after controlling for level of pain intensity (Wald = 9.308, OR = 1.630 p = 0.034). Chi-square analysis showed that the likelihood of failing to return to work increased with the number of prognostic factors ($\chi_3^2 = 14.076$, p = 0.003). The rate of work disability for participants with zero, one, two and three prognostic factors was 11.8%, 27.8%, 28.3% and 47.5%, respectively. Contrast comparisons revealed significant differences for individuals with zero prognostic factors and those with three prognostic factors ($\chi_1^2 = 12.326$, $p < 0.001$, Phi = 0.360); individuals with one prognostic factor and those with three prognostic factors ($\chi_1^2 = 4.736$, p = 0.030, Phi = 0.203); and between individuals with two prognostic factors and those with three prognostic factors ($\chi_1^2 = 4.429$, p = 0.035, Phi = 0.197).

Secondary Analysis to Explore Specific Combinations of Two Prognostic Factors

The above analysis suggests that participants' likelihood of problematic recovery is related to their number of prognostic factors (i.e. 0, 1, 2 or 3). However, it is also possible that for participants with two prognostic factors that the specific combination of prognostic factors contributed to their recovery (Linton, et al., 2011). To explore these relationships within our sample, we conducted a secondary analysis to determine whether elevated scores on different combinations of two factors related to problematic recovery. First, we created a sub-sample of individuals with elevated scores on two psychosocial factors (N = 53). We then grouped these individuals into three categories, which represented each of the possible interactions between prognostic factors, namely: 1) high catastrophizing and high fear; 2) high depression and high catastrophizing; 3) high fear and high depression. Next, chi-square analyses were conducted to determine whether there were significant relationships between the different combinations and problematic recovery on the follow-up indices. Results failed to reveal any significant differences ($p < 0.05$).

Discussion

The purpose of this study was to explore the relationship between the number of elevated scores on FAM-relevant factors and the likelihood of problematic recovery following physical therapy. Our results suggest that elevated scores on the Cumulative Prognostic Factor Index (CPFI) were associated with an increased risk of problematic recovery. These results build on previous cumulative psychosocial research (Linton, et al., 2011) by showing a similar phenomenon within a work-disabled population and by addressing the three primary psychosocial factors of the FAM. Our findings also suggest that use of the CPFI in the early stages of recovery may help identify patients' at risk for problematic recovery and facilitate decision-making regarding clinical management.

Our results suggest that different scores on the CPFI are associated with different profiles of recovery. *Profile of recovery*, in this instance, refers to patients' performance at one-year follow-up on the two dependent variables (i.e.

pain reduction *and* work disability). When considered from this perspective, a score of zero on the CPFI represents the most favorable profile of recovery following physical therapy. Our results suggest that the profile of recovery changes (in reference to a CPFI of zero) with certain incremental increases on the index. For instance, when compared to a CPFI of zero, a CPFI of one or two is associated with a significant increase in the likelihood of experiencing unimproved pain symptoms at one-year follow-up. A CPFI of three further contributes to the likelihood of problematic recovery by being associated with both an elevated likelihood of experiencing prolonged pain *and* an increased risk of experiencing long-term work disability. Our findings therefore suggest that the risk associated with problematic recovery increases with CPFI scores above zero, and that levels of risk are most elevated with a CPFI score of three.

The manner by which risk for problematic recovery increases in our sample suggests that reductions in pain intensity and return to work status have at least partially distinct relationships with the number of elevated psychosocial factors. Our findings suggest that individuals with elevated scores on one or two prognostic factors are significantly more likely to report persistent pain at one-year follow-up (when compared to those with no elevated scores), but are not significantly more likely to experience work disability at the same time-point; only patients with CPFI scores of three have an increased risk of experiencing work disability. These findings suggest that while levels of pain intensity may be sensitive to the presence of just one prognostic factor, measures of work disability are more resilient, only being influenced by elevated scores on several psychosocial factors. It is possible that rates of work disability are only significantly increased when patients' psychosocial distress has become sufficiently complex. Work disability is a behavioral/social outcome that is influenced by a wide variety of different psychosocial mechanisms (e.g. negative expectancies, avoidant behavior, motivational deficits) (Sullivan, et al., 2005a). It is possible that patients that are experiencing distress in only a few psychosocial domains (i.e. patients with lower CPFI scores) are able to successfully overcome these return to work barriers over the course of physical therapy, while patients

experiencing distress in a wider range of psychosocial domains are not. The relationship between different psychosocial factors and levels of pain severity, on the other hand, has been suggested to occur through common physiological pathways (e.g. descending modulation of pain; (Edwards, Calahan, Mensing, Smith, & Haythornthwaite, 2011; Price, 2000). This overlap may mean that more complex forms of psychosocial distress have less of a dramatic impact on the severity of pain intensity. The reader is reminded, however, that this interpretation remains speculative and that despite the reported statistical differences between specific CPFI scores, both outcomes followed a general trend of increasing with the number of elevated prognostic factors (as shown in Figure 1).

While the FAM proposes sequential relations between pain catastrophizing, fear of movement and depressive symptoms, it does not provide specific predictions regarding the potential cumulative relationships among these factors. Recent findings have suggested the prognostic importance of *specific combinations* of model-relevant factors (Linton, et al., 2011). Our findings, however, failed to show any differences between individuals with various combinations of two prognostic factors, but did show significant differences between individuals with varying CPFI scores. Our results therefore suggest that the *number,* rather than specific combinations*,* of elevated FAM factors may be more closely related to prognosis. Readers should be cautioned in the interpretation of these findings, however, as it is possible that this secondary analysis was underpowered. Assuming that our findings are valid, one explanation for the discrepancy with previous findings is that related analyses had measured two, rather than three, FAM-relevant factors; with only two factors under analysis it is not possible to flush out the contributions between the number of elevated factors versus the contribution of their interaction. Future research will need to use larger sample sizes to further explore the cumulative relationships among FAM-relevant factors to determine whether expansion of the model is warranted.

Our findings also relate to recent clinical research exploring the use of brief psychosocial screening tools. Previous research suggests that scores on *The Orebro Musculoskeletal Pain Screening Questionnaire* and the *Subgroups for*

Targeted Treatment (STarT) Back Screening Tool can predict pain-related recovery (Fritz, Beneciuk, & George, 2011; Hill, et al., 2008; Hill, Dunn, Main, & Hay, 2010; Hockings, McAuley, & Maher, 2008; Hurley, et al., 2000). These scales share several similarities with the CPFI. For instance, each of these tools aims to provide a generalized measure of different forms of pain-related psychosocial distress that can be used to within clinical settings. Despite their prognostic value and similarities, however, there may be important differences in the clinical information that they can provide. One important difference may be the underlying psychosocial constructs that each of the scales were designed to represent. As described above, the CPFI is not a new scale, but rather a novel index of scores on validated, FAM-relevant scales. As a result, the relatively large body of FAM literature can be used to guide the clinical interpretation of CPFI scores. For instance, previous research suggests that the psychosocial factors in the FAM are modifiable by a wide range of clinical interventions (Morley, et al., 2008; Moseley, et al., 2004; Smeets, et al., 2006a). It has also been suggested that lighter FAM-based interventions suffice for individuals with lower profiles of psychosocial risk, while more intensive psychosocial treatments are required for those with higher profiles of risk (Haldorsen, et al., 2002). Together this research suggests that the CPFI may be useful in guiding the use of different FAM-based interventions. For example, it is possible that individuals with CPFI scores of one or two may benefit from targeted psychosocial interventions that can be easily integrated within traditional physical therapy; interventions such as graded activity, graded exposure and education have been used in this manner (George, Fritz, Bialosky, & Donald, 2003; Sullivan & Wideman, 2011; Wideman & Sullivan, 2011). While individuals with CPFI scores of three may require, in addition to traditional physical therapy, more intensive psychosocial interventions and/or referral to additional health professionals; standardized ten-week interventions have been delivered by physical therapists and psychologists in this context (Sullivan, et al., 2006b; Sullivan & Stanish, 2003; Thorn, et al., 2007).

In comparison, the clinical information that physical therapists can garner from streamlined screening tools may be less clear. For example, the Orebro

Questionnaire was originally constructed without reference to a guiding theoretical framework (Linton & Halldén, 1998) and the broad psychosocial constructs that are addressed in this questionnaire have yet to be validated in reference to established psychosocial scales. Previous research also suggests ambiguity in the number, and name, of factors underlying this scale (Grotle, Vøllestad, & Brox, 2006; Linton & Halldén, 1998; Westman, Linton, Ohrvik, Wahlén, & Leppert, 2008). Without a clear sense of the psychosocial factors or theoretical framework that are relevant to the Orebro Questionnaire it may be difficult for physical therapists to use patients' scores on this scale to guide their clinical practice.

The STarT Back Tool, on the other hand, was designed to address specific psychosocial factors that have been shown to be modifiable. STarT Back scores are intended to help primary care physicians decide if their patients with acute back pain should be referred for additional healthcare services (e.g. physical therapy and/or psychology); use of the CPFI in clinical settings may prove complementary to this screening tool. For example, the concise nature of the STarT Back Tool may be ideal for a brief physician consult, while the CPFI, based on more comprehensive psychosocial scales, may be better suited for rehabilitation settings, particularly for patients who have been screened into the high-risk category in the initial consultation. While psychosocial screening tools have clear advantages with respect to responder burden, it is not clear whether there is additional clinically-meaningful information that can be gained by using more comprehensive measures of psychosocial factors; recent findings showing that the STarT Back Tool did not perform as expected when administered in physical therapy settings (Fritz, et al., 2011) suggest that more information may be required. Future research is needed to determine the most appropriate clinical contexts for using streamlined screening tools versus their more comprehensive counterparts.

Several limitations influence the application of our findings. For instance, our study did not include a specific measure of physical function. While successful return to work may infer a certain level of physical performance (e.g.

tolerating a regular work schedule), it does not address participants' specific physical impairments or abilities. A specific measure of physical function would have provided a more comprehensive profile of recovery. Also, while physical therapy treatment was guided by practice guidelines, we did not have any information on the specific interventions that were administered; it is possible that variance in the treatment interventions may have contributed to patients' outcomes. Furthermore, no information was collected regarding treatment attendance or participation. Finally, the manner in which we created the CPFI was based on the *a priori* assumption that each of the three psychosocial factors had equal prognostic weights. It is possible that the CPFI would have had greater prognostic accuracy if a prediction model had been created in which exact weights for each factor were used. While our simplified approach may facilitate the integration of the CPFI into clinical practice, there is likely a loss of information regarding the specificity between different factors and problematic recovery. As the integration of psychosocial factors into physical therapy practice becomes more prevalent (and nuanced), research that explores the use of more sophisticated clinical prediction models will likely be warranted.

Despite these limitations, this research sheds light on the relatively novel area of cumulative psychosocial risk. Our results suggest that patients' prognoses for problematic recovery is related to the number of elevated psychosocial factors. More precisely, our findings therefore suggest that the risk associated with problematic recovery increases with CPFI scores above zero, and that levels of risk are most severe with elevated scores on all three psychosocial factors. This research suggests that the CPFI could be used in clinical settings to better evaluate prognosis and to facilitate decisions regarding clinical management.

Acknowledgements

The primary author was supported by funds from the Canadian Institutes for Health Research (CIHR), the Institut de recherche Robert-Sauvé en santé et en securité du travail (IRSST) and by the Physiotherapy Foundation of Canada

through the Dominion Physiotherapy Research Scholarship. The authors have no financial interest in the results of this research.

Tables

Table 1

Characteristics of the study sample (N = 202).

Characteristics	N (%) or Mean (SD)
Education	
Less than high school	35 (17.3)
High school	58 (28.7)
Trade school	40 (19.8)
College	47 (23.3)
University	22 (10.9)
Occupation	
Laborer	65 (32.2)
Nursing	48 (23.8)
Clerical	39 (19.3)
Trade	21 (10.4)
Driving	13 (6.4)
Sales	13 (6.4)
Other	3 (1.5)
Injury site (categories are not mutually exclusive)	
Back	187 (92.6)
Neck	162 (80.2)
Upper extremity	115 (56.9)
Lower extremity	52 (25.7)
Pre-treatment medication use	
OTC NSAIDs	96 (47.5)
Opioids	36 (17.8)
Anti-inflammatories	12 (5.9)
Pre-treatment assessment variables	
Pain intensity	5.1 (1.8)
PCS	21.5 (10.7)
TSK	42.8 (8.1)
BDI	15.1 (9.4)

Pre-treatment psychosocial prognostic factor index	1.7 (1.1)
Number of prognostic factors	
0	34 (16.8)
1	54 (26.7)
2	53 (26.2)
3	61 (30.2)
One-year follow-up variables	
Pain intensity	3.6 (2.3)
Less than 30% reduction in pain intensity	106 (52.5%)
Did not return to work	63 (31.2)

SD = Standard deviation; PCS = Pain catastrophizing scale; TSK = Tampa scale of kinesiophobia; BDI = Beck depression Inventory; OTC NSAID = Over-The-Counter Non Steroidal Anti-Inflammatory Drugs

Table 2

Inter-Correlations Between Pre-Treatment Psychosocial Factors (N = 202).

	TSK	BDI
PCS	.570*	.503*
TSK	–	.288*

PCS = Pain catastrophizing scale; TSK = Tampa scale of kinesiophobia; BDI = Beck depression Inventory; * $p < 0.001$

Table 3

Means, standard deviations and effect sizes of pre-treatment psychosocial factors for participants that did, and did not, (a) experience a meaningful reduction in pain intensity, and (b) return to work at one-year follow up.

		Positive Outcome	Negative Outcome		
Variables		M (SD)	M (SD)		
	a)	Meaningful reduction in pain N = 96	No meaningful reduction in pain N = 106	F statistic (η^2)	P-value
PCS		19.25 (10.58)	23.61 (10.40)	8.720 (0.042)	0.004*
TSK		41.61 (8.69)	43.80 (7.48)	3.693 (0.018)	0.056
BDI		13.68 (9.30)	16.35 (9.30)	4.129 (0.020)	0.043*
	b)	Returned to work N = 139	Did not return to work N = 63		
PCS		19.88 (10.75)	25.21 (9.65)	11.33 (0.054)	0.001*
TSK		42.05 (8.32)	44.33 (7.52)	3.46 (0.017)	0.064
BDI		13.15 (7.53)	19.33 (11.57)	20.55 (0.093)	<0.001*

PCS = Pain catastrophizing scale; TSK = Tampa Scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; PT = Post-treatment; $^{*}p < 0.05$.

Table 4

Distribution of participants across CPFI scores that experienced a problematic outcome at one-year follow-up

CPFI Scores	Participants Without Meaningful Reduction in Pain Intensity N (%)	Participants That Did Not Return to Work N (%)
0 (N = 34)	8 (23.5%)	4 (11.8)
1 (N = 54)	30 (55.6)	15 (27.8)
2 (N = 53)	29 (54.7)	15 (28.3)
3 (N = 61)	39 (63.9)	29 (47.5)

Table 5

Contrast comparisons of the likelihood of outcome between each number of elevated psychosocial factors.

Prognostic factor comparisons by outcome	Chi-square Value (Effect size – Phi statistic)	P-value
Reduction in pain		
0 versus 1 prognostic factor	8.722 (-0.315)	0.003*
0 versus 2 prognostic factors	8.243 (-0.308)	0.004*
0 versus 3 prognostic factors	14.258 (-0.387)	<0.001*
1 versus 2 prognostic factor(s)	0.008 (0.008)	0.931
1 versus 3 prognostic factor(s)	0.838 (-0.085)	0.360
2 versus 3 prognostic factors	1.001 (-0.094)	0.317
Return to work		
0 versus 1 prognostic factor	3.160 (0.190)	0.075
0 versus 2 prognostic factors	3.318 (0.195)	0.069
0 versus 3 prognostic factors	12.326 (0.360)	<0.001*
1 versus 2 prognostic factor(s)	0.04 (0.006)	0.952
1 versus 3 prognostic factor(s)	4.736 (0.203)	0.030*
2 versus 3 prognostic factors	4.429 (0.197)	0.035*

* = $p < 0.05$

Figures

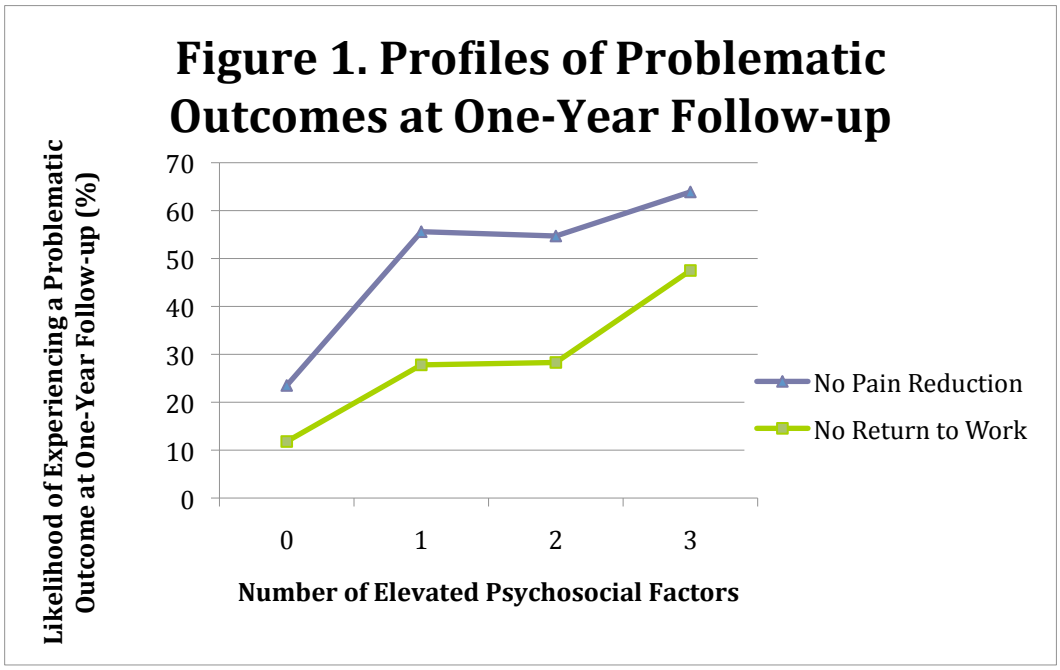
Figure 1. Profiles of Problematic Outcomes at One-Year Follow-up
Likelihood of Experiencing a Problematic Outcome at One-Year Follow-up (%)
70
60
50
40
30
20
10
0
0
1
2
3
Number of Elevated Psychosocial Factors
No Pain Reduction
No Return to Work

Chapter 5: General Discussion

Summary of Model-Relevant Findings and Their Context Within Related Research

Findings from these three studies relate to several postulates of the Fear Avoidance Model. First, each of these studies lends broad support for the importance of the psychosocial constructs addressed within the Fear Avoidance Model. For instance, each study showed that model-relevant measures were linked to a range of pain-related outcomes. Considered together, these studies revealed that measures of pain catastrophizing, fear of movement, and depression were each linked, at least at the zero-order level, to pain intensity, disability, medication use, and healthcare utilization. Findings from these studies also lend some support for the inter-relationships proposed in the Fear Avoidance Model. For instance, each of the studies found that all model-relevant measures were cross-sectionally correlated. Study 2 also showed that when catastrophizing and fear were measured at the same time-point, fear mediated the relationship between catastrophizing and disability.

Despite this support, however, findings from these studies contradicted several key postulates of the Fear Avoidance Model. Study 1 showed that the prospective sequential relationships proposed by the Fear Avoidance Model were not supported. Study 2 showed that fear was not a common psychological conduit for different pain-related outcomes. Studies 2 and 3 both supported alternate inter-relationships, which were not predicted by the Fear Avoidance Model. Study 2 revealed that catastrophizing and fear act as differential predictors of pain and disability, respectively. Study 2 also showed that pain self-efficacy, a factor not addressed in the Fear Avoidance Model, is a unique predictor of pain-related behaviour. Study 3 revealed that model-relevant variables have a cumulative effect on pain and disability. Together these findings suggest that the specific inter-relationships proposed within the Fear Avoidance Model may not accurately portray the experiences of people living with musculoskeletal pain conditions.

Recent research suggests a similar lack of empirical support for other aspects of the Fear Avoidance Model. For instance, after reviewing the literature relating to the Fear Avoidance Model, Pincus and her colleagues concluded that there is little support for the causal relationships proposed in the model (Pincus, Smeets, Simmonds, & Sullivan, 2010; Pincus, et al., 2006). Her most recent review also highlighted the limited efficacy of model-driven, clinical interventions (Pincus, et al., 2010). Other researchers have suggested that the proposed disuse syndrome, the theoretical link between fear avoidance and disability, has poor empirical validity. For example, several researchers have concluded that despite numerous cross-sectional and longitudinal studies there has been no evidence to suggest that decreases in cardiovascular fitness, muscular strength or functional endurance lead to the development of chronic pain and disability (Smeets, et al., 2006b; Smeets & Wittink, 2007; Verbunt, Seelen, & Vlaeyen, 2004; Verbunt, Smeets, & Wittink, 2010). Moreover, Smeets and his colleagues revealed that for a subgroup of individuals with chronic musculoskeletal pain who *did* show impaired levels of aerobic fitness, that these impairments were not associated with levels of pain-related fear (Smeets, van Geel, & Verbunt, 2009). Considered within the context of our negative findings, this lack of empirical support for the core mechanism that drives the Fear Avoidance Model prompts broad concerns about the empirical validity of the model.

In the discussion for each of the three studies presented, findings were contextualized within the Fear Avoidance Model as it has been traditionally presented within the literature. It is possible, however, that the observed lack of empirical support for fundamental aspects of the Fear Avoidance Model (both in our studies and in related research) stems from shortcomings within the theoretical model itself. In the discussion that follows we explore the validity of various theoretical assumptions that are made within the model, but have largely been overlooked within previous reviews. Our discussion is focused on three themes, namely: pain-related phobia as a central and influential construct among individuals with musculoskeletal pain; the co-presentation of both chronic pain

and pain-related disability; and the independence of model-relevant constructs from pain-related biological processes. Following this discussion we explore implications and future directions for models of pain-related disability.

Pain-Related Phobia as a Central Construct Among Individuals With Pain: Is Fear Avoidance the Only Pathway to Disability?

A central assumption made by the Fear Avoidance Model is that characteristics of phobic disorders are applicable and influential among individuals living with chronic musculoskeletal pain. As outlined in the introduction, pain-related fear (or kinesiophobia) is conceptualized within the Fear Avoidance Model as an excessive and irrational fear of movement that drives avoidance and precipitates disability. Graded exposure interventions, the recommended treatment for patients with elevated levels of fear, were originally developed for, and are commonly used among, individuals suffering from phobic disorders. Interestingly, despite the model's strong emphasis on pain-related phobia, the relevance of this concept for individuals living with pain conditions has largely been taken for granted. In the text below we question this conjecture by comparing key characteristics of phobic disorders with the typical presentation of individuals with musculoskeletal pain conditions.

Phobia is a form of anxiety disorder that is characterized by excessive and irrational fear, and persistent behaviour to avoid feared stimuli. The Diagnostic and Statistical Manual (DSM) of Mental Disorders provides the following diagnostic criteria for specific phobic disorders:

1. "Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation
2. Exposure to the phobic stimulus almost invariably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed panic attack.
3. The person recognizes that the fear is excessive or unreasonable.
4. The phobic situation(s) is avoided or else is endured with intense anxiety or distress.

5. The avoidance, anxious anticipation or distress in the feared situation(s) interferes significantly with the person's normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.
6. In individuals under the age of 18, the duration is at least 6 months.
7. The anxiety, panic attack, or phobic avoidance associated with the specific object or situation are not better accounted for by another mental disorder." (p. 449 – 450; (APA, 2000))

While many of the themes addressed in the DSM criteria have been broadly integrated within the Fear Avoidance Model, they are not commonly observed among individuals with musculoskeletal pain conditions. Pain researchers familiar with the diagnostic criteria of phobic conditions have observed such discrepancies within their clinical practice. For instance, C. de C. Williams & McCracken (C. de C. Williams & McCracken, 2004) report that patients with elevated levels of pain-related fear rarely, if ever, view their fear as excessive or unreasonable (DSM criteria number 3). They also highlight that such patients commonly fail to show signs of distress when confronted with *feared* stimuli (DSM criteria numbers 1, 2, and 4). Based on their clinical experiences, these researchers have concluded that "fear of pain and damage do not resemble the classic phobia" (p. 302; (C. de C. Williams & McCracken, 2004)). Findings from empirical research support this conclusion. For instance, a recent study compared brain activation patterns amongst individuals with elevated levels of pain-related fear to individuals with spider phobias (Barke, Baudewig, Schmidt-Samoa, Dechent, & Kröner-Herwig, 2012). As expected, when confronted with images of spiders, participants with arachnophobia showed activation in areas of the brain commonly associated with fear (e.g. the limbic system). However, such activation patterns were absent when participants with elevated levels of pain-related fear were exposed to equivalent feared stimuli (Barke, et al., 2012). Related research has also shown that individuals with elevated levels of pain-related fear fail to show classic physiological signs of distress, such as elevated heart rate, skin conductance or muscle activation, when performing feared

movements (Vlaeyen, et al., 1995a; Vlaeyen, et al., 1999). Drawing from a similar population, other research has failed to associate implicit negative attitudes or a startle response with fear-related stimuli (Goubert, Crombez, Hermans, & Vanderstraeten, 2003; Kronshage, Kroener-Herwig, & Pfingsten, 2001; Leeuw, Peters, Wiers, & Vlaeyen, 2007b). Contrary to the Fear Avoidance Model's characterization of fear, this research suggests that phobia-related distress plays a limited role in the lives of people with musculoskeletal pain conditions.

As a potential consequence of this limited distress, elevated levels of pain-related fear do not appear to disrupt daily function to the level that would be expected among individuals suffering from a phobic disorder (DSM criteria number 5). For example, previous research shows that levels of pain-related fear fail to influence the daily physical activity levels, metabolic rates, or functional capabilities (e.g. ability to walk, climb stairs or reach) of individuals living with pain conditions (Bousema, Verbunt, Seelen, Vlaeyen, & Knottnerus, 2007; Verbunt, et al., 2001). Considered together, this research suggests that most of the diagnostic criteria associated with phobic conditions (i.e. DSM criteria numbers 1 through 5) fail to apply to individuals living with pain and elevated levels of pain-related fear.

Despite limited relevance for a pain-related phobia construct, previous research suggests that *measures* of pain-related fear hold predictive utility. These seemingly paradoxical findings may be explained by a discrepancy between the model's conceptualization and the empirical measurement of pain-related fear. While self-report measures of pain-related fear have consistently been shown to have high levels of reliability, their construct validity has been questioned. For instance, after reviewing the measurement properties of five model-relevant questionnaires, Lundberg and colleagues concluded that a unifying conceptual construct of pain-related fear was lacking (Lundberg, Grimby-Ekman, Verbunt, & Simmonds, 2011). Indeed, while the Fear Avoidance Model suggests that pain-related fear consists of physiological, behavioural and cognitive dimensions, items within self-report measures commonly focus on cognitive processes, such as beliefs and expectations related to pain, injury, and physical activity. Items that

address negative affect (e.g. feeling tense or nervous) or distress-related behaviour (e.g. sweating, blushing, shaking), which are common among self-report measures of phobic disorders (Letamendi, Chavira, & Stein, 2009), are absent from gold standard measures of pain-related fear (e.g. the Tampa Scale of Kinesiophobia or the Fear Avoidance Beliefs Questionnaire). This discrepancy between the phobia-based theoretical construct and the cognition-oriented measurement tools may help account for some of the inconsistencies in previous research. For instance, measures of pain-related fear have been well established as strong predictors of pain outcomes. This is likely a reflection of the prognostic importance of pain-related cognitive processes and the measurement emphasis that is placed on such processes. On the other hand, graded exposure interventions have been associated with limited efficacy, particularly with respect to their ability to influence levels of pain-related disability (Pincus, et al., 2010). This limited efficacy may be a result of targeting phobia-related processes that, for the most part, are not applicable to the lives of individuals with musculoskeletal pain conditions. In this manner, empirical evidence relating to the Fear Avoidance Model may lend support for measures of "pain-related fear", while failing to support the theoretical constructs of pain-related phobia and avoidance.

The Fear Avoidance Model has been proposed as a broad, heuristic theory of pain-related disability (Vlaeyen, Crombez, & Linton, 2009). However, through its central emphasis on phobia-driven avoidance, the model essentially describes only one possible pathway to negative pain-related outcomes. It is possible that this narrow focus severely limits the explanatory scope of the model. Competing cognitive and behavioural theories suggest that this may be the case. For instance, Fordyce suggested three primary mechanisms that could cause pain-related disability, only one of which focused on avoidance behaviour. Moreover, Fordyce recognized many potential causes of avoidance behaviour, not just fear (Fordyce, Shelton, & Dundore, 1982). Other theories have proposed, as alternatives to fear avoidance mechanisms, that pain-related disability can result from overuse activities or lack of motivation (Hasenbring & Verbunt, 2010; Pincus, et al., 2010). Each of these models highlights at least some empirical support for these

alternative pathways. In light of both the limited pertinence of a pain-related phobia construct and viable alternative pathways, it is unlikely that a single fear avoidance mechanism accounts for the process by which most individuals develop pain-related disability.

The Co-Presentation of Chronic Pain and Pain-Related Disability: What is the Role of Positive Psychosocial Factors?

A further theoretical assumption made within the Fear Avoidance Model is the concurrent manifestation of chronic pain and prolonged pain-related disability. The model proposes two polar trajectories that lead to either elevated levels of both pain and disability, or recovery from both pain and disability; the presence, or absence, of psychosocial risk factors determines the path charted. Thus, the Fear Avoidance Model suggests that individuals cannot live with chronic pain without also experiencing increased psychosocial burden and prolonged disability. Empirical findings, however, suggests otherwise. For example, a large, national survey across the United States revealed that more than 13% of individuals who live with chronic and severe pain do not report significant levels of fear avoidance beliefs, catastrophic thinking or pain-related disability (Karoly & Ruehlman, 2006). While the Fear Avoidance Model is unable to account for this cohort, recent research exploring resiliency factors may help explain their existence.

Resiliency research focuses on how individuals can successfully adapt to adverse stimuli or situations, such as prolonged and persistent pain (Sturgeon & Zautra, 2010). Resiliency literature has its roots in developmental research, exploring the mechanisms by which children overcome early-life trauma (Reich, Zautra, & Hall, 2010). Resiliency research, however, has recently been applied to the field of pain to help explain the regulatory processes that govern pain-related distress and disability. Resiliency has been conceptually described as an ability to recover from, or adapt to, adversity and stress (Karoly & Ruehlman, 2006). In the context of pain research, resiliency has been operationalized as experiencing high levels of pain without high levels of emotional distress or pain interference

(Karoly & Ruehlman, 2006). A wide-range of pain-related factors has been conceptually linked to the resiliency construct, for example: *dispositional optimism* is defined as the capacity to maintain positive expectations in the face of adversity; *purpose of life* is the extent to which one feels purpose and meaning; *acceptance* is characterized as one's willingness to experience pain and discomfort in the course of pursuing meaningful activities (Reich, et al., 2010; Sturgeon & Zautra, 2010).

Recent research suggests that resiliency factors are linked to constructs associated with the Fear Avoidance Model. For instance, elevated levels of resiliency factors have been associated with increased pain tolerance (Smith, et al., 2009), lower depressive symptoms (Smith & Zautra, 2004), and reductions in pain catastrophizing (Ong, Zautra, & Reid, 2010). Cross-sectional and longitudinal research findings also suggest that resiliency factors buffer the relationship between pain and negative affect; when exposed to pain stimuli, individuals with elevated levels of resiliency are less likely to experience decreased mood (Strand, et al., 2006; Zautra, Johnson, & Davis, 2005).

Recent evidence suggests that resiliency factors and risk factors represent two discrete domains that have unique predictive value. For instance, a study of people with chronic arthritis found that 39% of the sample either had low levels of both resiliency and risk factors or high levels of both factors, suggesting that positive and negative factors can vary independently (Smith & Zautra, 2008). This research also revealed that resiliency factors had unique prospective relationships with positive outcomes, while risk factors demonstrated unique relationships with problematic outcomes (Smith & Zautra, 2008). Similar relationships have been supported in samples of patients with fibromyalgia (Furlong, Zautra, Puente, López-López, & Valero, 2010; Zautra, et al., 2005). Considered together, this research suggests that addressing both resiliency factors and psychosocial risk constructs will facilitate the prediction of a wider range of both positive and negative outcomes. Consideration of both factors may also help explain how individuals can live with chronic pain without concurrently experiencing pain-

related disability or, conversely, how individuals can fail to show signs of pain-related disability while experiencing elevated levels of psychosocial risk factors.

The Independence of Model-Relevant Constructs From Pain-Related Biological Processes: How do Biopsychosocial Findings Relate to a Cognitive-Behavioural Model?

As outlined in the general introduction, the Fear Avoidance Model proposes limited roles for sensory and physiological aspects of pain. While the Fear Avoidance Model argues for physiological changes in relation to fear and avoidance (e.g. cardiovascular and muscular deconditioning), it fails to address any pain-related tissue changes that may occur following initial injury. The limited weight given to biological factors within the Fear Avoidance Model can be traced back to Fordyce's behavioural approaches to pain-related disability. Arguing that the Gate Control Theory did not provide an adequate explanation of chronic pain conditions, Fordyce developed an explanatory model that was not dependent of biological factors. This early theoretical fissure can be understood to have produced two divergent lines of study. On the one hand, the Fear Avoidance Model has resulted in a great deal of research supporting the role of psychosocial risk factors in the development and maintenance of pain-related outcomes. On the other hand, the Gate Control Theory has inspired an increasingly compelling body of research that addresses the role of physiological processes in the persistence of chronic pain.

In several instances, however, findings from this latter line of research conflict with the postulates of the Fear Avoidance Model. For instance, contrary to the early predictions of Fear Avoidance Model researchers, pain intensity has been frequently shown to be a significant predictor of both avoidance behaviour and pain-related disability, even after controlling for model-relevant psychosocial factors (Leeuw, et al., 2007a). Also, results from brain-imaging research suggest strong links between various chronic, non-specific pain conditions (e.g. fibromyalgia, non-specific back pain) and pain-related tissue changes (Apkarian, Baliki, & Geha, 2009; Apkarian, Hashmi, & Baliki, 2011; Jensen, 2010). For

example, compared to healthy controls, individuals with musculoskeletal pain conditions have been shown to have significantly less grey matter in areas of the brain that are related to the sensory, emotional and cognitive aspects of pain (Jensen, 2010). These tissue changes have also been linked to pain duration and shown to revert back to normal when pain resolves, suggesting that they coincide with the persistence of pain-related symptoms (Apkarian, et al., 2009; Jensen, 2010; Seminowicz, et al., 2011). Other research suggests that pain-related physiological changes at the level of the dorsal horn are involved in the development of chronic pain (Eide, 2000; Meeus & Nijs, 2007). This research suggests that repetitive, non-harmful, pain stimuli can trigger an increase in the size of neuronal receptive fields and a decrease in stimulation thresholds (Eide, 2000). Under these circumstances the neurological system is primed to amplify pain-related signals and to interpret normal sensory stimulation as pain, thus initiating and maintaining pain chronicity (Meeus & Nijs, 2007). Previous research has linked these processes to several chronic musculoskeletal pain conditions, and to increased levels of pain-related disability (George, Wittmer, Fillingim, & Robinson, 2006; Staud, et al., 2003). In contrast to the postulates of the Fear Avoidance Model, these findings suggest that even when measurable signs of tissue damage are absent, that pain-related physiological changes contribute to the development and persistence of disabling musculoskeletal pain conditions.

Inconsistencies between these two lines of research may be a reflection of the Fear Avoidance Model's dichotomous characterization of pain. Similar to Fordyce's description of respondent and operative pain behaviour, the Fear Avoidance Model presumes that *either* organic or non-organic factors underlie pain-related outcomes. While the Fear Avoidance Model suggests that biological factors may initiate acute pain and pain-related disability, it proposes that only psychosocial factors perpetuate these states. At odds with this understanding of pain, is a growing body of research that challenges such exaggerated delineations between biological and psychosocial factors. For instance, psychosocial factors have been shown to have genetic links, to be involved in inflammatory and

endocrine processes, and to be related to the structure and function of the central nervous system; many of these relationships have been supported in various pain conditions and at different temporal intervals within the acute to chronic pain continuum (Campbell & Edwards, 2009; Edwards, et al., 2011; Quartana, et al., 2009). Thus, contrary to model-relevant predictions, this research suggests that a close, bi-directional relationship between biological and psychosocial processes exists throughout both the development and persistence of pain conditions.

Implications for Advancing Models of Pain-Related Disability

Descartes' legacy of a fractured portrayal of pain has largely been resolved through current biopsychosocial conceptualizations. His influence, however, is still apparent within the leading model of pain-related disability. Since its development, the Fear Avoidance Model has had an instrumental role in integrating cognitive-behavioural factors within the literature addressing pain-related disability. However, when considered within the broader context of contemporary pain research, the model fails to address a wide range of physiological findings. Moreover, the model's ability to explain the development of pain-related disability appears to be restricted by its central emphasis on pain-related phobia. This defining focus also limits its potential for meaningful revision. Thus, while the Fear Avoidance Model has been a crucial theoretical stepping-stone in the historical advancement of pain and disability research, it may be time to consider how future models of pain-related disability can evolve to inspire new avenues of research.

One avenue for advancing future theory is to focus on the development of new models that have a broader capacity to explain pain-related disability. While presenting such a model is beyond the scope of this discussion, we can glean several recommendations through our empirical and theoretical analyses. First, new models of pain-related disability will need to permit multiple pathways and mechanisms to different pain-related outcomes. Second, such models should incorporate psychosocial risk factors addressed within the Fear Avoidance Model. While such factors may fail to relate in accordance with the Fear Avoidance

Model, their prognostic value has been well established. One caveat to this recommendation is that the construct of *pain-related fear/kinesiophobia* should be re-named to better reflect its empirical measures. Also, inter-relations should focus on cumulative, rather than sequential, relationships. Third, in addition to psychosocial risk factors, positive resiliency factors should be considered. Future research will need to explore whether positive and negative factors have a net cumulative effect on pain-related outcomes or whether they are better suited to differentiate indices of recovery and disability. Fourth, evolving models of pain-related disability will need to propose bi-directional relations between both psychosocial and physiological factors. Framing such models within a biopsychosocial perspective will facilitate the reconciliation of divergent lines of research and increase congruency with contemporary models of pain.

Integrating these recommendations into new models of pain-related disability may also be associated with important benefits for clinical practice. Addressing a broad range of psychosocial and physiological factors is expected to improve prognostic accuracy and inspire a wide range of theory-driven interventions. For instance, previous research shows that positive psychosocial factors are modifiable and that various clinical interventions can be used to increase levels of resiliency (Reich, et al., 2010; Sturgeon & Zautra, 2010). New psychosocial interventions that facilitate the development of positive factors may serve to complement existing risk factor targeted treatments. Similarly, there may be advantages to combining treatments that target both physiological and psychosocial factors. Research addressing the specific effects of such combination therapies is still in its infancy and may be accelerated through the advent of new biopsychosocial models of pain-related disability.

Summary

The three manuscripts presented in this thesis lend little support for several aspects of the Fear Avoidance Model. Specifically, our findings failed to support model-relevant sequential relations and the proposed central role of pain-related fear. Contrary to model predictions, our findings also lend support for

psychosocial factors having differential relationships with pain-related outcomes and interacting in a cumulative fashion. When considered in the context of other empirical findings these results suggest a lack of support for the Fear Avoidance Model's central means of explaining pain-related disability. Several theoretical assumptions appear to undermine the Fear Avoidance Model's heuristic value. For instance, the central emphasis on pain-related phobia and avoidance may limit the model's validity and generalizability; the implied co-presentation of chronic pain and disability make it difficult to explain the observed variance in these two states; and the failure to integrate pain-related physiological mechanisms is at odds with a large body of biopsychosocial research. In light of the empirical and theoretical evidence, it is recommended that future research consider how new models of pain-related disability can begin to evolve. New models will need to permit inter-relationships among resiliency factors, psychosocial risk factors and biological processes, and be capable of explaining multiple mechanisms for the development of and recovery from pain-related disability. Models that address these points are expected to have a meaningful and positive impact on the lives of individuals suffering from musculoskeletal pain conditions.

APPENDIX 1

Table S1. Pre, mid and post-treatment values for process variables

Variables	Pre	Mid	Post	Pre to Mid change	Mid to Post change
	Mean (SD)	Mean (SD)	Mean (SD)	F statistic (Partial η^2)	F statistic (Partial η^2)
PCS	34.51 (7.78)	28.68 (9.46)	25.75 (11.41)	79.230** (.398)	13.574** (.102)
TSK	48.03 (5.74)	45.52 (7.03)	43.69 (7.83)	18.055** (.131)	10.411** (.080)
BDI	21.07 (11.40)	18.43 (10.14)	17.15 (11.31)	11.787** (.089)	2.450 (.020)
MPQ	38.12 (14.38)	38.14 (14.90)	36.84 (17.40)	0.000 (.000)	1.039 (.009)

PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table S2

Correlations between pre-treatment scores

Variables	1	2	3
1. Pre-treatment PCS	–		
2. Pre-treatment TSK	0.454**	–	
3. Pre-treatment BDI	0.547**	0.356**	–
4. Pre-treatment MPQ	0.233*	0.268**	0.454**

PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; $^{**}p < 0.01$ (2-tailed); $^{*}p < 0.05$ (2-tailed).

Table S3

Pre-treatment means, standard deviations and effect sizes for participants that returned to work, and did not return to work

Change Indices	Returned to work M (SD) N = 37	Did not return to work M (SD) N = 84	F statistic (Partial η^2)
Age	41.35 (8.19)	42.15 (7.50)	0.279 (0.002)
Injury duration	29.76 (31.75)	61.63 (113.59)	2.805 (0.023)
Pre-treatment PCS	32.84 (6.54)	35.25 (8.20)	2.496 (0.021)
Pre-treatment TSK	46.22 (5.36)	48.83 (5.74)	5.55 (0.045)*
Pre-treatment BDI	18.11 (9.32)	22.37 (12.03)	3.665 (0.030)
Pre-treatment MPQ	30.11 (12.85)	41.65 (13.63)	19.064 (0.138)**

PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; $^{*}p < 0.05$; $^{**}p < 0.01$.

Table S4

Cross-sectional correlations for early and late change indices

Variables

	A. ch2 PCS	B. ch2 TSK	C. ch2 BDI	D. ch2 MPQ
1. ch1 PCS	–	0.384**	0.634**	0.392**
2. ch1 TSK	0.372**	–	0.327**	0.271**
3. ch1 BDI	0.418**	0.376**	–	0.324**
4. ch1 MPQ	0.068	0.107	-0.056	–

Note. Cross-sectional correlations between early change indices can be found below the diagonal, while cross-sectional correlations between late change indices are located above the diagonal.

ch1 = early change; ch2 = late change; PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; $^{**}p < 0.01$ (two-tailed)

Table S5

Longitudinal correlations between early and late change indices

Variables	ch1 PCS	ch1 TSK	ch1 BDI	ch1 MPQ
ch2 PCS	-0.050	0.042	-0.085	-0.055
ch2 TSK	0.093	-0.315**	-0.131	-0.020
ch2 BDI	0.016	0.068	-0.163	-0.053
ch2 MPQ	0.074	-0.053	0.081	-0.314**

ch1 = early change; ch2 = late change; PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; $^{**}p < 0.01$ (two-tailed).

Table S6

Means, standard deviations and effect sizes for participants who retuned to work (RTW), and did not return to work (No RTW)

	RTW	No RTW	
Variables	M (SD) N = 37	M (SD) N = 84	F statistic (Partial η^2)
age	41.35 (8.19)	42.15 (7.48)	0.279 (0.002)
injury dur	29.76 (31.74)	61.63 (113.59)	2.805 (0.023)
ch1 PCS	8.24 (7.04)	4.76 (7.04)	6.268 (0.050)*
ch2 PCS	7.51 (8.17)	0.92 (8.28)	16.443 (0.121)**
ch1 TSK	2.86 (6.60)	2.36 (6.49)	0.155 (0.001)
ch2 TSK	4.00 (5.81)	0.88 (6.24)	6.690 (0.053)*
ch1 BDI	4.62 (7.76)	1.76 (8.63)	2.993 (0.025)
ch2 BDI	3.32 (6.83)	0.38 (9.71)	2.787 (0.023)
ch1 MPQ	0.51 (10.30)	-0.25 (12.27)	0.109 (0.001)
ch2 MPQ	6.57 (10.38)	-1.02 (14.79)	7.992 (0.063)**

injury dur = injury duration; ch1 = early change; ch2 = late change; PCS = Pain catastrophizing; TSK = Fear of movement; BDI = Depression; MPQ = Pain intensity; * $p < 0.05$; ** $p < 0.01$.

APPENDIX 2

Table S1

Characteristics of participants with incomplete data

	N (%) or Mean (SD)			
	Participants with incomplete data (N = 33)	Participants with incomplete T3 and follow-up data (N = 23)	Participants with complete T3 data, but no follow-up data (N = 4)	Participants with complete follow-up data, but incomplete T3 data (N = 6)
Sex				
Male	9 (27.3%)	5 (21.7%)	1 (25%)	3 (50%)
Female	24 (72.7%)	18 (78.3%)	3 (75%)	3 (50%)
Age	37.55 (10.27)	37.48 (10.00)	40.50 (15.20)	35.83 (9.24)
Pain duration	6.6 (3.02)	7.04 (2.98)	5.25 (3.20)	6.00 (3.29)
Pre-treatment assessment values				
Pain	5.47 (1.70)	5.63 (1.81)	5.5 (2.08)	4.83 (0.98)
PCS	22.67 (12.37)	26 (12.63)	16.50 (6.81)	14.00 (8.88)
TSK	42.97 (6.78)	43.61 (7.93)	42.00 (3.65)	41.17 (1.72)
BDI	15.45 (9.68)	18.09 (9.31)	11.00 (11.89)	8.33 (4.97)
PSEQ	32.51 (13.99)	29.30 (14.33)	36.25 (3.20)	42.33 (12.97)
PT assessment values	–	–		–
Pain			5.5 (1.00)	
PCS			13.00 (11.16)	
TSK			37.75 (10.40)	
BDI			21.50 (25.99)	
PSEQ			39.50 (7.50)	
Follow-up values	–	–		
Pain				1.83 (1.33)
Healthcare utilization				0.00 (0.00)
Return to work				Yes: 5 (83.3%) No: 1 (16.7%)
Medication use				0.33 (0.52)

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire;

Table S2

Hierarchical linear regression analysis: evaluation of fear of movement as a mediator between pain catastrophizing and pain intensity at one-year follow-up.

	Variables	t	Beta	R^2	R^2 Change	F change
Step 1	PT pain intensity					
		13.173**	0.682	0.465	0.465	173.527**
Step 2						
	PT PCS	3.513**	0.224	0.496	0.031	12.342**
Step 3						
	PT PCS	2.508*	0.192	–	–	–
	PT TSK	0.736	0.048	0.497	0.001	0.541

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; $^{**}p < 0.01$; $*p < 0.05$.

Table S3

Hierarchical linear regression analysis: predictors of healthcare utilization at one-year follow-up.

	Variables	t	Beta	R^2	R^2 Change	F change
Step 1						
	Pre-treatment opioid use	2.926**	0.203	0.138	0.138	31.920**
Step 2						
	PT pain intensity	2.671**	0.178	0.167	0.030	7.134**
Step 3						
	PT PCS	0.670	0.072			
	PT TSK	0.057	0.005			
	PT BDI	1.322	0.117	0.186	0.018	1.454
Step 4						
	PT PSEQ	0.918	0.105	0.189	0.004	0.843

PT = Post-treatment; PCS = Pain catastrophizing scale; TSK = Tampa scale of Kinesiophobia; BDI = Beck depression inventory; PSEQ = Pain self-efficacy questionnaire; $^{*}p < 0.05$; $^{**}p < 0.01$.

REFERENCES

Adams, H., Ellis, T., Stanish, W. D., & Sullivan, M. J. L. (2007). Psychosocial factors related to return to work following rehabilitation of whiplash injuries. *Journal of occupational rehabilitation, 17*(2), 305-315.

Airaksinen, O., Brox, J. I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., et al. (2006). Chapter 4. European guidelines for the management of chronic nonspecific low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 15 Suppl 2*, S192-300.

APA (2000). *Diagnostic and statistical manual of mental disorders (4th edition)*. Arlington: American Psychiatric Publishing.

Apkarian, A. V., Baliki, M. N., & Geha, P. Y. (2009). Towards a theory of chronic pain. *Progress in Neurobiology, 87*(2), 81-97.

Apkarian, A. V., Hashmi, J. A., & Baliki, M. N. (2011). Pain and the brain: specificity and plasticity of the brain in clinical chronic pain. *Pain, 152*(3 Suppl), S49-64.

Asmundson, G., Vlaeyen, J., & Crombez, G. (2004). *Understanding and treating fear of pain*. New York: Oxford University Press.

Audi, R. (Ed.). (1995). *The Cambridge Dictionary of Philosophy*. New York: Cambridge University Press.

Ayre, M., & Tyson, G. A. (2001). The role of self-efficacy and fear-avoidance beliefs in the prediction of disability. *Australian Psychologist, 36*(3), 250-253.

Bair, M. J., Robinson, R. L., Katon, W., & Kroenke, K. (2003). Depression and pain comorbidity: a literature review. *Archives of Internal Medicine, 163*(20), 2433-2445.

Baldwin, M. L. (2004). Reducing the costs of work-related musculoskeletal disorders: targeting strategies to chronic disability cases. *Journal of*

electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology, 14(1), 33-41.

Barke, A., Baudewig, J., Schmidt-Samoa, C., Dechent, P., & Kröner-Herwig, B. (2012). Neural correlates of fear of movement in high and low fear-avoidant chronic low back pain patients: An event-related fMRI study. *Pain, 153*(3), 540-552.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of personality and social psychology, 51*(6), 1173-1182.

Beck, A., Steer, R., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory - II*.Unpublished manuscript, San Antonio, TX.

Bergbom, S., Boersma, K., Overmeer, T., & Linton, S. J. (2011). Relationship Among Pain Catastrophizing, Depressed Mood, and Outcomes Across Physical Therapy Treatments. *Physical therapy, 91*(5), 754-764.

Bishop, S., Edgley, K., Fisher, R., & Sullivan, M. (1993). Screening for depression in chronic low back pain with the Beck Depression Inventory. *Canadian Journal of Rehabilitation, 7*, 143-148.

Boersma, K., & Linton, S. J. (2005a). How does persistent pain develop? An analysis of the relationship between psychological variables, pain and function across stages of chronicity. *Behaviour research and therapy, 43*(11), 1495-1507.

Boersma, K., & Linton, S. J. (2005b). Screening to identify patients at risk: profiles of psychological risk factors for early intervention. *The Clinical journal of pain, 21*(1), 38-43; discussion 69-72.

Boersma, K., & Linton, S. J. (2006). Psychological processes underlying the development of a chronic pain problem: a prospective study of the relationship between profiles of psychological variables in the fear-avoidance model and disability. *The Clinical journal of pain, 22*(2), 160-166.

Bortz, W. M. (1984). The disuse syndrome. *The Western Journal of Medicine, 141*(5), 691-694.

Bousema, E. J., Verbunt, J. A., Seelen, H. A. M., Vlaeyen, J. W. S., & Knottnerus, J. A. (2007). Disuse and physical deconditioning in the first year after the onset of back pain. *Pain, 130*(3), 279-286.

Braden, J. B., Sullivan, M. D., Ray, G. T., Saunders, K., Merrill, J., Silverberg, M. J., et al. (2009). Trends in long-term opioid therapy for noncancer pain among persons with a history of depression. *General Hospital Psychiatry, 31*(6), 564-570.

Burns, J. W., Glenn, B., Bruehl, S., Harden, R. N., & Lofland, K. (2003a). Cognitive factors influence outcome following multidisciplinary chronic pain treatment: a replication and extension of a cross-lagged panel analysis. *Behaviour research and therapy, 41*(10), 1163-1182.

Burns, J. W., Kubilus, A., Bruehl, S., Harden, R. N., & Lofland, K. (2003b). Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. *Journal of consulting and clinical psychology, 71*(1), 81-91.

Burton, A. K., Tillotson, K. M., Main, C. J., & Hollis, S. (1995). Psychosocial predictors of outcome in acute and subchronic low back trouble. *Spine, 20*(6), 722-728.

C. de C. Williams, A., & McCracken, L. (2004). Cognitive-behavioral therapy for chronic pain: An overview with specific reference to fear and avoidance. In G. J. Asmundson, J. Vlaeyen & G. Crombez (Eds.), *Understanding and treating fear of pain*. Toronto: Oxford University Press.

Calley, D. Q., Jackson, S., Collins, H., & George, S. Z. (2010). Identifying patient fear-avoidance beliefs by physical therapists managing patients with low back pain. *The Journal of orthopaedic and sports physical therapy, 40*(12), 774-783.

Campbell, C. M., & Edwards, R. R. (2009). Mind-body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts. *Translational Research, 153*(3), 97-101.

Chou, R., & Huffman, L. H. (2007). Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Annals of Internal Medicine, 147*(7), 492-504.

Chou, R., Qaseem, A., Snow, V., Casey, D., Cross, J. T., Shekelle, P., et al. (2007). Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. *Annals of Internal Medicine, 147*(7), 478-491.

Cook, A. J., Brawer, P. A., & Vowles, K. E. (2006). The fear-avoidance model of chronic pain: validation and age analysis using structural equation modeling. *Pain, 121*(3), 195-206.

Crombez, G., Van Damme, S., & Eccleston, C. (2005). Hypervigilance to pain: an experimental and clinical analysis. *Pain, 116*(1-2), 4-7.

Crombez, G., Vlaeyen, J. W., Heuts, P. H., & Lysens, R. (1999). Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. *Pain, 80*(1-2), 329-339.

Descartes, R. (1644/2008). Principles of Philosophy. In J. Veitch (Ed.), *The Barnes & Noble Library of Essential Reading*. New York: Barnes & Noble, Inc.

Descartes, R. (1649/1989). The Passions of the Soul. In S. Voss (Ed.), *The Passions of the Soul*. Indianapolis: Hackett Publishing Company.

Descartes, R. (1664/1972). Treatise of Man. In T. S. Hall (Ed.), *Harvard Monographs in the History of Science*. Cambridge, MA: Harvard University Press.

Downie, W. W., Leatham, P. A., Rhind, V. M., Wright, V., Branco, J. A., & Anderson, J. A. (1978). Studies with pain rating scales. *Annals of the Rheumatic Diseases, 37*(4), 378-381.

Duncan, G. (2000). Mind-body dualism and the biopsychosocial model of pain: what did Descartes really say? *Journal of Medicine and Philosophy, 25*(4), 485-513.

Dworkin, R. H., Turk, D. C., Wyrwich, K. W., Beaton, D., Cleeland, C. S., Farrar, J. T., et al. (2008). Interpreting the clinical importance of treatment outcomes in chronic pain clinical trials: IMMPACT recommendations. *J Pain, 9*(2), 105-121.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: a cognitive-affective model of the interruptive function of pain. *Psychological bulletin, 125*(3), 356-366.

Edwards, R. R., Calahan, C., Mensing, G., Smith, M., & Haythornthwaite, J. A. (2011). Pain, catastrophizing, and depression in the rheumatic diseases. *Nature Reviews Rheumatology, 7*(4), 216-224.

Eide, P. K. (2000). Wind-up and the NMDA receptor complex from a clinical perspective. *European journal of pain (London, England), 4*(1), 5-15.

Fordyce, W. E. (1976). *Behavioral Methods for Chronic Pain and Illness*. Saint Louis: The C. V. Mosby Company.

Fordyce, W. E. (1984). Behavioural science and chronic pain. *Postgraduate Medical Journal, 60*(710), 865-868.

Fordyce, W. E., Roberts, A. H., & Sternbach, R. A. (1985). The behavioral management of chronic pain: a response to critics. *Pain, 22*(2), 113-125.

Fordyce, W. E., Shelton, J. L., & Dundore, D. E. (1982). The modification of avoidance learning pain behaviors. *Journal of behavioral medicine, 5*(4), 405-414.

Foster, N. E., Thomas, E., Bishop, A., Dunn, K. M., & Main, C. J. (2010). Distinctiveness of psychological obstacles to recovery in low back pain patients in primary care. *Pain, 148*(3), 398-406.

Fritz, J. M., Beneciuk, J. M., & George, S. Z. (2011). Relationship Between Categorization With the STarT Back Screening Tool and Prognosis for People Receiving Physical Therapy for Low Back Pain. *Physical therapy, 91*(5), 722-732.

Fritz, J. M., & George, S. Z. (2002). Identifying psychosocial variables in patients with acute work-related low back pain: the importance of fear-avoidance beliefs. *Physical therapy, 82*(10), 973-983.

Fritz, J. M., George, S. Z., & Delitto, A. (2001). The role of fear-avoidance beliefs in acute low back pain: relationships with current and future disability and work status. *Pain, 94*(1), 7-15.

Furlong, L. V., Zautra, A., Puente, C. P., López-López, A., & Valero, P. B. (2010). Cognitive-affective assets and vulnerabilities: two factors influencing adaptation to fibromyalgia. *Psychology & Health, 25*(2), 197-212.

George, S. Z., Coronado, R. A., Beneciuk, J. M., Valencia, C., Werneke, M. W., & Hart, D. L. (2011). Depressive Symptoms, Anatomical Region, and Clinical Outcomes for Patients Seeking Outpatient Physical Therapy for Musculoskeletal Pain. *Physical therapy, 91*(3), 358 - 372.

George, S. Z., Dover, G. C., & Fillingim, R. B. (2007). Fear of pain influences outcomes after exercise-induced delayed onset muscle soreness at the shoulder. *The Clinical journal of pain, 23*(1), 76-84.

George, S. Z., Fritz, J. M., Bialosky, J. E., & Donald, D. A. (2003). The effect of a fear-avoidance-based physical therapy intervention for patients with acute low back pain: results of a randomized clinical trial. *Spine, 28*(23), 2551-2560.

George, S. Z., Fritz, J. M., & Childs, J. D. (2008). Investigation of elevated fear-avoidance beliefs for patients with low back pain: a secondary analysis involving patients enrolled in physical therapy clinical trials. *The Journal of orthopaedic and sports physical therapy, 38*(2), 50-58.

George, S. Z., Fritz, J. M., & Erhard, R. E. (2001). A comparison of fear-avoidance beliefs in patients with lumbar spine pain and cervical spine pain. *Spine, 26*(19), 2139-2145.

George, S. Z., Fritz, J. M., & McNeil, D. W. (2006). Fear-avoidance beliefs as measured by the fear-avoidance beliefs questionnaire: change in fear-avoidance beliefs questionnaire is predictive of change in self-report of disability and pain intensity for patients with acute low back pain. *The Clinical journal of pain, 22*(2), 197-203.

George, S. Z., Wittmer, V. T., Fillingim, R. B., & Robinson, M. E. (2006). Fear-avoidance beliefs and temporal summation of evoked thermal pain influence self-report of disability in patients with chronic low back pain. *Journal of occupational rehabilitation, 16*(1), 95-108.

Gheldof, E. L. M., Crombez, G., Van den Bussche, E., Vinck, J., Van Nieuwenhuyse, A., Moens, G., et al. (2010). Pain-related fear predicts disability, but not pain severity: A path analytic approach of the fear-avoidance model. *European journal of pain (London, England), 14*, 870.e871 - 870.e879.

Gheldof, E. L. M., Vinck, J., Vlaeyen, J. W. S., Hidding, A., & Crombez, G. (2005). The differential role of pain, work characteristics and pain-related fear in explaining back pain and sick leave in occupational settings. *Pain, 113*(1-2), 71-81.

Godges, J. J., Anger, M. A., Zimmerman, G., & Delitto, A. (2008). Effects of education on return-to-work status for people with fear-avoidance beliefs and acute low back pain. *Physical therapy, 88*(2), 231-239.

Goetzel, R. Z., Hawkins, K., Ozminkowski, R. J., & Wang, S. (2003). The health and productivity cost burden of the "top 10" physical and mental health conditions affecting six large U.S. employers in 1999. *Journal of Occupational and Environmental Medicine, 45*(1), 5-14.

Goodin, B. R., McGuire, L., Allshouse, M., Stapleton, L., Haythornthwaite, J. A., Burns, N., et al. (2009). Associations between catastrophizing and endogenous pain-inhibitory processes: sex differences. *The journal of pain : official journal of the American Pain Society, 10*(2), 180-190.

Goubert, L., Crombez, G., Hermans, D., & Vanderstraeten, G. (2003). Implicit attitude towards pictures of back-stressing activities in pain-free subjects and patients with low back pain: an affective priming study. *European journal of pain (London, England), 7*(1), 33-42.

Goubert, L., Crombez, G., & Van Damme, S. (2004). The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. *Pain, 107*(3), 234-241.

Grotle, M., Vøllestad, N. K., & Brox, J. I. (2006). Screening for yellow flags in first-time acute low back pain: reliability and validity of a Norwegian version of the Acute Low Back Pain Screening Questionnaire. *The Clinical journal of pain, 22*(5), 458-467.

Haggman, S., Maher, C. G., & Refshauge, K. M. (2004). Screening for symptoms of depression by physical therapists managing low back pain. *Physical therapy, 84*(12), 1157-1166.

Haldorsen, E. M. H., Grasdal, A. L., Skouen, J. S., Risa, A. E., Kronholm, K., & Ursin, H. (2002). Is there a right treatment for a particular patient group? Comparison of ordinary treatment, light multidisciplinary treatment, and extensive multidisciplinary treatment for long-term sick-listed employees with musculoskeletal pain. *Pain, 95*(1-2), 49-63.

Harris, C. A., & D'Eon, J. L. (2008). Psychometric properties of the Beck Depression Inventory--second edition (BDI-II) in individuals with chronic pain. *Pain, 137*(3), 609-622.

Hasenbring, M. I., & Verbunt, J. A. (2010). Fear-avoidance and endurance-related responses to pain: new models of behavior and their consequences for clinical practice. *The Clinical journal of pain, 26*(9), 747-753.

Hill, J. C., Dunn, K. M., Lewis, M., Mullis, R., Main, C. J., Foster, N. E., et al. (2008). A primary care back pain screening tool: identifying patient subgroups for initial treatment. *Arthritis & Rheumatism, 59*(5), 632-641.

Hill, J. C., Dunn, K. M., Main, C. J., & Hay, E. M. (2010). Subgrouping low back pain: a comparison of the STarT Back Tool with the Orebro Musculoskeletal Pain Screening Questionnaire. *European journal of pain (London, England), 14*(1), 83-89.

Hockings, R. L., McAuley, J. H., & Maher, C. G. (2008). A systematic review of the predictive ability of the Orebro Musculoskeletal Pain Questionnaire. *Spine, 33*(15), E494-500.

Hurley, D. A., Dusoir, T. E., McDonough, S. M., Moore, A. P., Linton, S. J., & Baxter, G. D. (2000). Biopsychosocial screening questionnaire for patients

with low back pain: preliminary report of utility in physiotherapy practice in Northern Ireland. *The Clinical journal of pain, 16*(3), 214-228.

Ilardi, S., & Craighead, W. (1994). The Role of Nonspecific Factors in Cognitive-Behavior Therapy for Depression. *Clinical Psychology: Science and Practice, 1*, 138-156.

Jensen, M. (2010). A Neuropsychological Model of Pain: Research and Clinical Implications. *The journal of pain : official journal of the American Pain Society, 11*(1), 2-12.

Kall, L. B. (2009). Psychological determinants of quality of life in patients with whiplash associated disorders-a prospective study. *Disability and rehabilitation, 31*(3), 227-236.

Karjalainen, K., Malmivaara, A., van Tulder, M., Roine, R., Jauhiainen, M., Hurri, H., et al. (2008). Multidisciplinary biopsychosocial rehabilitation for subacute low back pain among working age adults. *The Cochrane Collaboration*(4).

Karoly, P., & Ruehlman, L. S. (2006). Psychological "resilience" and its correlates in chronic pain: findings from a national community sample. *Pain, 123*(1-2), 90-97.

Kenny, A. (Ed.). (1997). *The Oxford Illustrated History of Western Philosophy*. New York: Oxford University Press.

Keogh, E., & Asmundson, G. J. (2004). Negative affectivity, catastrophizing, and anxiety sensitivity. In G. J. Asmundson, J. Vlaeyen & G. Crombez (Eds.), *Understanding and treating fear of pain*. Toronto: Oxford University Press.

Koes, B. W., van Tulder, M., Lin, C.-W. C., Macedo, L. G., McAuley, J., & Maher, C. (2010). An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 19*(12), 2075-2094.

Kronshage, U., Kroener-Herwig, B., & Pfingsten, M. (2001). Kinesiophobia in chronic low back pain patients—does the startle paradigm support the hypothesis? *International journal of behavioral medicine, 8*(4), 304-318.

Kudel, I., Edwards, R. R., & Moric, M. (2005). The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. A comment on Goubert et al. (2004). *Pain, 115*(1-2), 214-216.

Lacaille, D., Sheps, S., Spinelli, J. J., Chalmers, A., & Esdaile, J. M. (2004). Identification of modifiable work-related factors that influence the risk of work disability in rheumatoid arthritis. *Arthritis & Rheumatism, 51*(5), 843-852.

Lang, P. J. (1968). Fear reduction and fear behavior: Problems in treating a construct. In J. M. Shlien (Ed.), *Research in psychotherapy*. Washington, DC: American Psychological Association.

Langley, P. C. (2011). The prevalence, correlates and treatment of pain in the European Union. *Current Medical Research & Opinion, 27*(2), 463-480.

Leeuw, M., Goossens, M. E., van Breukelen, G. J., de Jong, J. R., Heuts, P. H., Smeets, R. J., et al. (2008). Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. *Pain, 138*(1), 192-207.

Leeuw, M., Goossens, M. E. J. B., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. S. (2007a). The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *Journal of behavioral medicine, 30*(1), 77-94.

Leeuw, M., Peters, M. L., Wiers, R. W., & Vlaeyen, J. W. S. (2007b). Measuring fear of movement/(re)injury in chronic low back pain using implicit measures. *Cognitive Behaviour Therapy, 36*(1), 52-64.

Lentz, T. A., Barabas, J. A., Day, T., Bishop, M. D., & George, S. Z. (2009). The relationship of pain intensity, physical impairment, and pain-related fear to function in patients with shoulder pathology. *The Journal of orthopaedic and sports physical therapy, 39*(4), 270-277.

Letamendi, A. M., Chavira, D. A., & Stein, M. B. (2009). Issues in the assessment of social phobia: a review. *Israel Journal of Psychiatry & Related Sciences, 46*(1), 13-24.

Lethem, J., Slade, P. D., Troup, J. D., & Bentley, G. (1983). Outline of a Fear-Avoidance Model of exaggerated pain perception--I. *Behaviour research and therapy, 21*(4), 401-408.

Linton, S. J., & Buer, N. (1995). Working despite pain: factors associated with work attendance versus dysfunction. *International journal of behavioral medicine, 2*(3), 252-262.

Linton, S. J., & Halldén, K. (1998). Can we screen for problematic back pain? A screening questionnaire for predicting outcome in acute and subacute back pain. *The Clinical journal of pain, 14*(3), 209-215.

Linton, S. J., Nicholas, M. K., Macdonald, S., Boersma, K., Bergbom, S., Maher, C., et al. (2011). The role of depression and catastrophizing in musculoskeletal pain. *European journal of pain (London, England), 15*(4), 416-422.

Lundberg, M., Grimby-Ekman, A., Verbunt, J., & Simmonds, M. J. (2011). Pain-related fear: a critical review of the related measures. *Pain Research and Treatment, 2011*, 494196.

McCracken, L. M., Faber, S. D., & Janeck, A. S. (1998). Pain-related anxiety predicts non-specific physical complaints in persons with chronic pain. *Behaviour research and therapy, 36*(6), 621-630.

Meeus, M., & Nijs, J. (2007). Central sensitization: a biopsychosocial explanation for chronic widespread pain in patients with fibromyalgia and chronic fatigue syndrome. *Clinical Rheumatology, 26*(4), 465-473.

Melzack, R. (1975). The McGill Pain Questionnaire: major properties and scoring methods. *Pain, 1*(3), 277-299.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: a new theory. *Science, 150*(699), 971-979.

Melzack, R., & Wall, P. D. (1996). *The Challenge of Pain* (Updated second edition ed.). Toronto: Penguin Group.

Miller, R. P., Kori, S. H., & Todd, D. D. (1991). The Tampa Scale: A Measure of Kinisophobia. *The Clinical journal of pain, 7*(1), 51-52.

Morley, S., Williams, A., & Hussain, S. (2008). Estimating the clinical effectiveness of cognitive behavioural therapy in the clinic: evaluation of a CBT informed pain management programme. *Pain, 137*(3), 670-680.

Moseley, G. L., Nicholas, M. K., & Hodges, P. W. (2004). A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *The Clinical journal of pain, 20*(5), 324-330.

Nicholas, M. K. (2007). The pain self-efficacy questionnaire: Taking pain into account. *European journal of pain (London, England), 11*(2), 153-163.

Nieto, R., Miró, J., & Huguet, A. (2009). The fear-avoidance model in whiplash injuries. *European journal of pain (London, England), 13*(5), 518-523.

Norton, P., & Asmundson, G. (2003). Amending the fear-avoidance model of chronci pain: What is the role of physiological arousal? *Behavior Therapy, 34*, 17-30.

Ong, A. D., Zautra, A. J., & Reid, M. C. (2010). Psychological resilience predicts decreases in pain catastrophizing through positive emotions. *Psychology & Aging, 25*(3), 516-523.

Panel, P. (2001). Philadelphia Panel evidence-based clinical practice guidelines on selected rehabilitation interventions for low back pain. *Physical therapy, 81*(10), 1641-1674.

Peters, M. L., Vlaeyen, J. W. S., & Weber, W. E. J. (2005). The joint contribution of physical pathology, pain-related fear and catastrophizing to chronic back pain disability. *Pain, 113*(1-2), 45-50.

Philips, H. C. (1987). Avoidance behaviour and its role in sustaining chronic pain. *Behaviour research and therapy, 25*(4), 273-279.

Pincus, T., Burton, A. K., Vogel, S., & Field, A. P. (2002). A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine, 27*(5), E109-120.

Pincus, T., & Newman, S. (2001). Recall bias, pain, depression and cost in back pain patients. *British Journal of Clinical Psychology, 40*(Pt 2), 143-156.

Pincus, T., Smeets, R. J. E. M., Simmonds, M. J., & Sullivan, M. J. L. (2010). The fear avoidance model disentangled: improving the clinical utility of the fear avoidance model. *The Clinical journal of pain, 26*(9), 739-746.

Pincus, T., Vogel, S., Burton, A. K., Santos, R., & Field, A. P. (2006). Fear avoidance and prognosis in back pain: a systematic review and synthesis of current evidence. *Arthritis Rheum, 54*(12), 3999-4010.

Price, D. D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science, 288*(5472), 1769-1772.

Quartana, P. J., Campbell, C. M., & Edwards, R. R. (2009). Pain catastrophizing: a critical review. *Expert Review of Neurotherapeutics, 9*(5), 745-758.

Reich, J. W., Zautra, A., & Hall, J. S. (Eds.). (2010). *Handbook of Adult Resilience*. New York: The Guilford Press.

Rey, R. (1995). *The History of Pain*. Cambridge, MA: Harvard University Press.

Roelofs, J., Goubert, L., Peters, M. L., Vlaeyen, J. W., & Crombez, G. (2004). The Tampa Scale for Kinesiophobia: further examination of psychometric properties in patients with chronic low back pain and fibromyalgia. *European journal of pain (London, England), 8*(5), 495-502.

Sanders, S. H., Harden, R. N., & Vicente, P. J. (2005). Evidence-based clinical practice guidelines for interdisciplinary rehabilitation of chronic nonmalignant pain syndrome patients. *Pain practice : the official journal of World Institute of Pain, 5*(4), 303-315.

Schiphorst Preuper, H. R., Reneman, M. F., Boonstra, A. M., Dijkstra, P. U., Versteegen, G. J., Geertzen, J. H., et al. (2008). Relationship between psychological factors and performance-based and self-reported disability in chronic low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 17*(11), 1448-1456.

Schultz, I. Z., Crook, J., Berkowitz, J., Milner, R., Meloche, G. R., & Lewis, M. L. (2008). A prospective study of the effectiveness of early intervention

with high-risk back-injured workers--a pilot study. *Journal of occupational rehabilitation, 18*(2), 140-151.

Seminowicz, D. A., & Davis, K. D. (2006). Cortical responses to pain in healthy individuals depends on pain catastrophizing. *Pain, 120*(3), 297-306.

Seminowicz, D. A., Wideman, T. H., Naso, L., Hatami-Khoroushahi, Z., Fallatah, S., Ware, M. A., et al. (2011). Effective treatment of chronic low back pain in humans reverses abnormal brain anatomy and function. *The Journal of Neuroscience, 31*(20), 7540-7550.

Severeijns, R., Vlaeyen, J. W. S., & van den Hout, M. A. (2004). Do we need a communal coping model of pain catastrophizing? An alternative explanation. *Pain, 111*(3), 226-229.

Severeijns, R., Vlaeyen, J. W. S., van den Hout, M. A., & Picavet, H. S. J. (2004). Pain catastrophizing is associated with health indices in musculoskeletal pain: a cross-sectional study in the Dutch community. *Health psychology : official journal of the Division of Health Psychology, American Psychological Association, 23*(1), 49-57.

Smeets, R. J., van Geel, K. D., & Verbunt, J. A. (2009). Is the fear avoidance model associated with the reduced level of aerobic fitness in patients with chronic low back pain? *Archives of physical medicine and rehabilitation, 90*(1), 109-117.

Smeets, R. J. E. M., Vlaeyen, J. W. S., Kester, A. D. M., & Knottnerus, J. A. (2006a). Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. *The journal of pain : official journal of the American Pain Society, 7*(4), 261-271.

Smeets, R. J. E. M., Wade, D., Hidding, A., Van Leeuwen, P. J. C. M., Vlaeyen, J. W. S., & Knottnerus, J. A. (2006b). The association of physical deconditioning and chronic low back pain: a hypothesis-oriented systematic review. *Disability and rehabilitation, 28*(11), 673-693.

Smeets, R. J. E. M., & Wittink, H. (2007). The deconditioning paradigm for chronic low back pain unmasked? *Pain, 130*(3), 201-202.

Smith, B. W., Tooley, E. M., Montague, E. Q., Robinson, A. E., Cosper, C. J., & Mullins, P. G. (2009). The role of resilience and purpose in life in habituation to heat and cold pain. *The journal of pain : official journal of the American Pain Society, 10*(5), 493-500.

Smith, B. W., & Zautra, A. J. (2004). The role of purpose in life in recovery from knee surgery. *International journal of behavioral medicine, 11*(4), 197-202.

Smith, B. W., & Zautra, A. J. (2008). Vulnerability and resilience in women with arthritis: test of a two-factor model. *Journal of consulting and clinical psychology, 76*(5), 799-810.

Staal, J. B., Hlobil, H., Köke, A. J. A., Twisk, J. W. R., Smid, T., & van Mechelen, W. (2008). Graded activity for workers with low back pain: who benefits most and how does it work? *Arthritis & Rheumatism, 59*(5), 642-649.

Staud, R., Cannon, R. C., Mauderli, A. P., Robinson, M. E., Price, D. D., & Vierck, C. J. (2003). Temporal summation of pain from mechanical stimulation of muscle tissue in normal controls and subjects with fibromyalgia syndrome. *Pain, 102*(1-2), 87-95.

Storheim, K., Brox, J. I., Holm, I., & Bø, K. (2005). Predictors of return to work in patients sick listed for sub-acute low back pain: a 12-month follow-up study. *Journal of Rehabilitation Medicine, 37*(6), 365-371.

Strand, E. B., Zautra, A. J., Thoresen, M., Ødegård, S., Uhlig, T., & Finset, A. (2006). Positive affect as a factor of resilience in the pain-negative affect relationship in patients with rheumatoid arthritis. *Journal of psychosomatic research, 60*(5), 477-484.

Sturgeon, J. A., & Zautra, A. J. (2010). Resilience: a new paradigm for adaptation to chronic pain. *Currrent Pain and Headache Reports, 14*(2), 105-112.

Sullivan, M., Bishop, S., & Pivik, J. (1995). The Pain Catastrophizing Scale: Development and Validation. *Psychological Assessment, 7*(4), 524 - 532.

Sullivan, M., Tanzer, M., Stanish, W., Fallaha, M., Keefe, F. J., Simmonds, M., et al. (2009). Psychological determinants of problematic outcomes following Total Knee Arthroplasty. *Pain, 143*(1-2), 123-129.

Sullivan, M., & Wideman, T. (2011). Pain catastrophizing and fear of movement: detection and intervention. In M. E. Lynch, K. D. Craig & P. W. H. Peng (Eds.), *Clinical pain management : a practical guide* (pp. 207 - 214). Chichester, West Sussex, UK ; Hoboken, NJ: Blackwell Pub.

Sullivan, M. J., Adams, H., Thibault, P., Corbière, M., & Stanish, W. D. (2006a). Initial depression severity and the trajectory of recovery following cognitive-behavioral intervention for work disability. *Journal of occupational rehabilitation, 16*(1), 63-74.

Sullivan, M. J., Feuerstein, M., Gatchel, R., Linton, S. J., & Pransky, G. (2005a). Integrating psychosocial and behavioral interventions to achieve optimal rehabilitation outcomes. *Journal of occupational rehabilitation, 15*(4), 475-489.

Sullivan, M. J., Reesor, K., Mikail, S., & Fisher, R. (1992). The treatment of depression in chronic low back pain: review and recommendations. *Pain, 50*(1), 5-13.

Sullivan, M. J., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., et al. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *The Clinical journal of pain, 17*(1), 52-64.

Sullivan, M. J. L., Adams, H., Rhodenizer, T., & Stanish, W. D. (2006b). A psychosocial risk factor--targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Physical therapy, 86*(1), 8-18.

Sullivan, M. J. L., Lynch, M. E., & Clark, A. J. (2005b). Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. *Pain, 113*(3), 310-315.

Sullivan, M. J. L., & Stanish, W. D. (2003). Psychologically based occupational rehabilitation: the Pain-Disability Prevention Program. *The Clinical journal of pain, 19*(2), 97-104.

Sullivan, M. J. L., Ward, L. C., Tripp, D., French, D. J., Adams, H., & Stanish, W. D. (2005c). Secondary prevention of work disability: community-based psychosocial intervention for musculoskeletal disorders. *Journal of occupational rehabilitation, 15*(3), 377-392.

Swinkels-Meewisse, E. J. C. M., Swinkels, R. A. H. M., Verbeek, A. L. M., Vlaeyen, J. W. S., & Oostendorp, R. A. B. (2003). Psychometric properties of the Tampa Scale for kinesiophobia and the fear-avoidance beliefs questionnaire in acute low back pain. *Manual therapy, 8*(1), 29-36.

Swinkels-Meewisse, I. E. J., Roelofs, J., Schouten, E. G. W., Verbeek, A. L. M., Oostendorp, R. A. B., & Vlaeyen, J. W. S. (2006). Fear of movement/(re)injury predicting chronic disabling low back pain: a prospective inception cohort study. *Spine, 31*(6), 658-664.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Pearson/Allyn & Bacon.

Tang, T. Z., & DeRubeis, R. J. (1999). Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *Journal of consulting and clinical psychology, 67*(6), 894-904.

Tang, T. Z., DeRubeis, R. J., Beberman, R., & Pham, T. (2005). Cognitive changes, critical sessions, and sudden gains in cognitive-behavioral therapy for depression. *Journal of consulting and clinical psychology, 73*(1), 168-172.

Thomas, J. S., & France, C. R. (2007). Pain-related fear is associated with avoidance of spinal motion during recovery from low back pain. *Spine, 32*(16), E460-466.

Thorn, B. E., Pence, L. B., Ward, L. C., Kilgo, G., Clements, K. L., Cross, T. H., et al. (2007). A randomized clinical trial of targeted cognitive behavioral treatment to reduce catastrophizing in chronic headache sufferers. *The journal of pain : official journal of the American Pain Society, 8*(12), 938-949.

Turk, D. C., Rudy, T. E., & Salovey, P. (1985). The McGill Pain Questionnaire reconsidered: confirming the factor structure and examining appropriate uses. *Pain, 21*(4), 385-397.

Turner, J. A., Holtzman, S., & Mancl, L. (2007). Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain. *Pain, 127*(3), 276-286.

Van Damme, S., Crombez, G., Eccleston, C., & Roelofs, J. (2004). The role of hypervigilance in the experience of pain. In G. J. Asmundson, J. Vlaeyen & G. Crombez (Eds.), *Understanding and treating fear of pain*. Toronto: Oxford University Press.

Verbunt, J., Seelen, H. A., & Vlaeyen, J. (2004). Disuse and physical deconditioning in chronic low back pain. In G. J. Asmundson, J. Vlaeyen & G. Crombez (Eds.), *Understanding and treating fear of pain*. Toronto: Oxford University Press.

Verbunt, J. A., Smeets, R. J., & Wittink, H. M. (2010). Cause or effect? Deconditioning and chronic low back pain. *Pain, 149*(3), 428-430.

Verbunt, J. A., Westerterp, K. R., van der Heijden, G. J., Seelen, H. A., Vlaeyen, J. W., & Knottnerus, J. A. (2001). Physical activity in daily life in patients with chronic low back pain. *Archives of physical medicine and rehabilitation, 82*(6), 726-730.

Vlaeyen, J., Crombez, G., & Linton, S. (2009). The fear-avoidance model of pain: We are not there yet. Comment on Wideman et al. "A prospective sequential analysis of the fear-avoidance model of pain" [Pain, 2009] and Nicholas "First things first: reduction in catastrophizing before fear of movement". *Pain, 146*, 222-223.

Vlaeyen, J. W., Kole-Snijders, A. M., Boeren, R. G., & van Eek, H. (1995a). Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain, 62*(3), 363-372.

Vlaeyen, J. W., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain, 85*(3), 317-332.

Vlaeyen, J. W., Seelen, H. A., Peters, M., de Jong, P., Aretz, E., Beisiegel, E., et al. (1999). Fear of movement/(re)injury and muscular reactivity in chronic low back pain patients: an experimental investigation. *Pain, 82*(3), 297-304.

Vlaeyen, J. W. S., de Jong, J., Geilen, M., Heuts, P. H. T. G., & van Breukelen, G. (2002). The treatment of fear of movement/(re)injury in chronic low back pain: further evidence on the effectiveness of exposure in vivo. *The Clinical journal of pain, 18*(4), 251-261.

Vlaeyen, J. W. S., Kole-Snijders, A. M., Rotteveel, A. M., Ruesink, R., & Heuts, P. H. (1995b). The role of fear of movement/(re) injury in pain disability. *Journal of occupational rehabilitation, 5*(4), 235-252.

Vlaeyen, J. W. S., & Linton, S. J. (2012). Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain, 153*(6), 1144-1147.

Vowles, K. E., Gross, R. T., & Sorrell, J. T. (2004). Predicting work status following interdisciplinary treatment for chronic pain. *European journal of pain (London, England), 8*(4), 351-358.

Waddell, G. (1987). 1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain. *Spine, 12*(7), 632-644.

Waddell, G. (2004). *The back pain revolution* (2 ed.). London, UK: Churchill Livingstone.

Waddell, G., Newton, M., Henderson, I., Somerville, D., & Main, C. J. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain, 52*(2), 157-168.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: exploring the central role of negative affectivity. *Psychological Review, 96*(2), 234-254.

Watson, P. J., Booker, C. K., & Maas, C. J. (1997). Evidence for the role of psychological factors in abnormal paraspinal activity in patients with chronic low back pain. *Journal of Musculoskeletal Pain, 4*(4), 41-55.

Westman, A., Linton, S. J., Ohrvik, J., Wahlén, P., & Leppert, J. (2008). Do psychosocial factors predict disability and health at a 3-year follow-up for patients with non-acute musculoskeletal pain? A validation of the Orebro Musculoskeletal Pain Screening Questionnaire. *European journal of pain (London, England), 12*(5), 641-649.

Wideman, T. H., Adams, H., & Sullivan, M. J. L. (2009). A prospective sequential analysis of the fear-avoidance model of pain. *Pain, 145*(1-2), 45-51.

Wideman, T. H., & Sullivan, M. J. L. (2011). Reducing catastrophic thinking associated with pain. *Pain Management, 1*(3), 249-256.

Woby, S. R., Urmston, M., & Watson, P. J. (2007). Self-efficacy mediates the relation between pain-related fear and outcome in chronic low back pain patients. *European journal of pain (London, England), 11*(7), 711-718.

Woods, M. P., & Asmundson, G. J. G. (2008). Evaluating the efficacy of graded in vivo exposure for the treatment of fear in patients with chronic back pain: a randomized controlled clinical trial. *Pain, 136*(3), 271-280.

Zautra, A. J., Johnson, L. M., & Davis, M. C. (2005). Positive affect as a source of resilience for women in chronic pain. *Journal of consulting and clinical psychology, 73*(2), 212-220.