"*Everyone, whether he be a plowman or banker, clerk or captain, citizen or ruler, is, in a real sense, a philosopher*."

(Frost (1962), "Basic Teachings of the Great Philosophers", pg. 1)

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Work, Injury, and Depression: The influence of work status on depressive symptoms for those recovering from musculoskeletal injury

by

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Dedication

This dissertation is dedicated to my loving wife, Erin, who put up with my ramblings, writings, and numerous commitments to this paper and my Doctoral program as a whole; since the day we first met. Thank-you, Erin.

I would also like to share the dedication with my beautiful children Anna Rose, and Thomas Charles, Jones. I hope that I can eventually share the lessons I learned about persistence, patience, and commitment as I worked through this paper and my program.

Lastly, I would like to share the dedication with my parents who instilled the foundations of persistence, patience, and commitment; thus providing me the skills necessary to achieve my academic goals.

Abstract

Many individuals obtain a sense of personal identity from work as well as the resources necessary for basic living. Musculoskeletal injury is a common barrier to continued employment in developed countries and despite numerous compensation programs, work absences can significantly disrupt an individual's sense of self, potentially predisposing the injured individual to significant symptoms of depression. The goal of this dissertation was to investigate the relationship between work status and subsequent depression following injury.

Chapter two provided an introductory, theoretical framework from which to view the contribution of work to the possible prevention or resolution of depressive symptoms following injury. In chapter 3, a cohort of whiplash patients were followed forward in time to investigate the temporal relationship between work status and depressive symptoms following injury. From this investigation it was found that work status significantly influence subsequent depression shortly after injury, but not in the longer term. In Chapter 4, a cohort of worker's compensation claimants were categorized by work status shortly after their injury and followed forward in time for depression outcomes. The relationships observed in chapter 3 were not replicated in chapter 4; however, the worker's compensation cohort suffered from loss to follow-up and the timing of assessments differed significantly from those used in chapter 3.

Chapter five provided a methodological comparison of differing methods with which to control for confounding in studies using continuous baseline health outcome measures which are also used to categorize disease status further on in follow-up. Through this investigation it became apparent that there is no one right answer for using related continuous and categorical outcomes in the same regression models; however, for our research, it was apparent that prior depressive symptom scores were best characterized as a confounding variable in the 'work-status and subsequent depression' relationship.

In conclusion this dissertation provided three important contributions to the study of depression following injury: 1. There is a plausible and visually explainable mechanism by which alterations in work status can influence subsequent depression following injury; 2. Work status is significantly associated with subsequent depression status shortly after whiplash injury; and 3. The alteration of a single modelling variable can substantially alter the conclusions drawn in follow-up studies of depression and depressive symptomatology; careful consideration must be made when including and excluding seemingly associated variables in a predictive regression model.

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Chapter 1 – Introduction

The influence of work on the mental health of individuals recovering from injury is not clear. This dissertation aims to: 1. Provide an introductory conceptual framework to help explain a portion of the relationship between work, injury, and depression. The goal of this framework is to provide clinicians a window into how work status might impact mental health after injury as well as provide researchers a baseline visual framework from which to view future investigations on this topic; 2. Present applicable research to help further our understanding of the relationships between work, injury, and depression; and 3. To make available clinically applicable research to help facilitate the best possible physical and mental health outcomes for individuals recovering from injury;

1.1. Background

Work and Role Theory

Merriam-Webster defines work as, "[the] sustained physical or mental effort to overcome obstacles and achieve an objective or result" or "b: the labour, task, or duty that is one's accustomed means of livelihood", and finally "c: a specific task, duty, function, or assignment often being a part or phase of some larger activity".[*] Synonyms of work include, but are not limited to, labour, employment and occupation. In general, employment provides for the necessities of life and

[*] Merriam-Webster's Online Dictionary. Work. 2008. [cited 27 Aug 2008]; Available from URL: http://www.merriam-webster.com/dictionary/work

may also contribute to healthy socialization, daily physical activity, and a sense of productivity.[1-3] Work enables individuals to fulfill a valuable life "role". Work often forms the crux of our identities. When asked "what do you do?", the underlying or intended question is often "what work do you do?". This theme of work as a major contributor to self identity is further reinforced in our responses; which are generally work related. People will often list work roles before more personal roles. For example, one is more likely to say "I am a researcher", than "I am a father" in response to the question "what do you do?". This underlines the importance we place on work in our lives.

Role theory presents the idea that an individual's behaviour is often contingent upon expectations (individual and societal) regarding behaviour and learned norms. This theory is comprised of a "triad" of concepts. Specifically, role theory concerns itself with i) patterned and characteristic behaviours, ii) assumed identities, and iii) expectations for behaviour that are understood by all.[4] Sub-fields of this theory have been proposed in the literature and include areas such as "functional role theory" and "cognitive role theory".[4] An individual's work status has important implications that can be best visualized through cognitive role theory; which focuses on expectation and behaviours.

Cognitive role theory can be used to frame issues around injury and work status in two ways. First, an alteration in the ability to work, as a result of injury, may alter an individual's identity, both from their own and from others'

perspectives. For example, by limiting their ability to fulfill their "expected" roles, injured individuals may no longer feel like "themselves". More precisely, prior to injury, these individuals would expect to work on a regular basis. This expectation to work would also be realized by those surrounding the injured person (spouse, supervisor, co-worker, etc.). When the worker can no longer work, their internal dialogue could become distorted (because they are now unable to carry out an expected, self-identifying role), predisposing that worker to psychological distress or a psychological disorder. In other words, if an individual strongly identifies with their work and place of work, not being able to participate in work could negatively influence that person's self-perceptions and they would no longer "feel like themselves". The words and actions of those around the injured person may facilitate or attenuate the injured worker's negative self-perceptions. Pragmatically, this could have observable effects on an individual's mental health through prolonged negative thoughts and/or a lack of support from friends, family, and colleagues. This idea has some support in the literature and will be discussed further in chapter 2.[3, 5-10]

Secondly, workplace absences may contribute to taking on negative roles. Specifically, when not connected to the workplace during recovery, the worker might more easily identify with, and therefore participate in, a "sick role" (aka "role taking").[4, 7, 10] The phrase "sick role" was first used by the sociologist Talcott Parsons (1951).[†] Currently, the term "sick role" refers to a social role that

[†] Parsons, Talcott, *The Social System,* New York, Free Press, 1951.[7]

is taken up by individuals to provide legitimacy of their inability to carry out a traditional role or roles (such as work).[7] Health care providers may inadvertently facilitate the uptake of such a role by an injured individual. By giving a diagnosis, the pre-requisite for treatment, a health care provider legitimizes that worker's "sickness". Additionally, should a health care provider recommend an individual abstain from their usual activities (such as work), they may again be (inadvertently) facilitating the injured person's full transition into the sick role. However, this issue is extremely complex. Whether or not an off-work recommendation during recovery facilitates a transition to the sick-role most likely depends on many factors. These factors likely include the injured person's actual injury severity, their coping style, the health provider's belief system (e.g. whether or not they think the workplace is a good place to be during recovery), the employer's flexibility (e.g. availability of modified work), and others.

Using role theory as a framework, it has been suggested that employment or work status may be central to an individual's identity while providing additional benefits such as financial resources, a sense of personal control and social integration.[4-7] Thus, it seems reasonable (as previously described) to assume that removing an individual from their work environment, further disrupting their "role", could prove detrimental to the individual's social, physical and mental health. As mentioned, time away from work may well promote an individual's self-identification with the "sick-role"; thus perpetuating self deprecating ideas and behaviours not conducive to recovery.[7] This assumption has received some

attention in recent years and is subject to debate. It could be argued that a removal from, or loss of, employment may result in fewer financial resources, a loss of social integration, and a reduction in personal control. These losses might lead to increased stress on the worker (whether it be difficulty paying one's bills or being thrown into the healthcare – compensation matrix [i.e. a perceived loss of personal control]) with potentially deleterious effects on physical, social, and mental health.[1] To add credence to this view, a recent review of literature related to work and health stated "there is extensive evidence that there are strong links between unemployment and poorer physical and mental health and mortality".[‡]

Conversely (and more frequently studied), many jobs are associated with a degree of perceived stress, and some have argued that removal from a stressful work environment may be beneficial.[1, 3, 5, 11] For example, workers who are employed in a position in which they report a high degree of psychological stress and low decision latitude (basically, a limited ability to make work-related decisions independently) are more prone to major depressive episodes.[11] It has been argued that removal from the work environment for a medical condition may provide benefits such as adequate time to attend treatment(s), an absence from occupational stresses, and a reduced likelihood of a work related (or an additional work-related) injury.[1] However, it has also been suggested that recommendations to remain away from work to recover from illness will facilitate lifestyle choices

[‡] Waddell and Burton[3], page 13.

which reinforce roles that are ultimately detrimental to an individual's health.[7] This issue remains unresolved.

Work Absence and Musculoskeletal Injury

A work absence may be described as a state of worklessness: "not being engaged in any form of work."[§] Musculoskeletal (MSK) injury is a significant contributor to workplace absences. In Canada it has been estimated that at least 70% of workers compensation claims are a result of a MSK condition and MSK conditions are the leading cause of workplace disability claims in Australia and the United States.[12, 13] Health Canada reported that MSK disorders accounted for $16.4 billion in societal costs in 1998, second only to cardiovascular disease.[14] Although a more up-to-date figures specific to MSK disorders and injuries are not available, a recent report by SmartRisk (2009) (on behalf of the Public Health Agency of Canada) stated that injuries in general (MSK and non-MSK) resulted in almost $20 billion of economic burden to Canada in 2004.[15] These estimates are comprised of direct (e.g., physician, hospital, and drug costs) and indirect costs (e.g., long and short-term disability, and mortality related costs). Indirect costs account for almost 50% of expenses attributed to all injury types but account for over 90% of expenses attributed to MSK disorders.[14, 15] Over 15.5 million visits were made to Canadian physicians in 1998-99 for a musculoskeletal concern, and there were approximately 212,000 hospitalizations and well over 3 million non-hospitalized treatments for injuries in 2004. [14-16]

[§] Waddell and Burton[3], page 4.

Healthcare practitioners often use "off-work" recommendations to facilitate recovery from a MSK condition. An "off-work" prescription is often given, or recommended, when a health care provider perceives work as an additional or unnecessary risk during the recovery process.[**] Conversely, it may be the injured patient or claims manager who advocates for an employee to take time off work after suffering an injury.[19] However, some authors suggest that time off work can be detrimental to an individual's overall health.[3, 7, 17-19] Although it is not clear who was advocating for time off work (worker or health care provider), Carroll *et al* found that 40-50% of individuals suffering whiplash injury reported being off work (at least briefly) as a result of their injury.[20]

The burden of musculoskeletal disorders and work absences are extensive. These burdens are realized in personal finances (through lost or reduced wages), and are realized socially through extensive health care utilization and lost productivity. Appropriate management of MSK related work absences is exceedingly important in light of these costs, and is made even more significant when considering the potential for negative impacts on an individual's mental, physical, or social health when they are removed from their work environment.[3, 17-19] The presence of depressive symptoms may complicate a worker's recovery and it is not clear if these symptoms are a cause of, or result of, a work absence.

[**] Although "off-work" prescription is not always explicit, it is often implied through a physician's judgment's regarding "impairment", "functional capacity", "limitations", and "restrictions"[19]

The topics of depression and the relationship between depression and work absence are explored in the following sections.

Depression

Depression and symptoms of depression are commonly associated with MSK conditions and contribute to delayed recovery from a musculoskeletal injury. [7, 10, 13, 20-22] The World Health Organization (WHO) defines depression as "a common mental disorder, characterized by sadness, loss of interest or pleasure, feelings of guilt or low self-worth, disturbed sleep or appetite, low energy and poor concentration".[††] The WHO also estimates that depression is the current leading cause of disability worldwide and ranks fourth in global burden of diseases.[23] Lifetime prevalence of major depression in Canada is estimated to be 11% for men and 16% for women, and the overall lifetime prevalence of depressive disorders in Alberta is estimated to be 9%.[24, 25] The incidence of major depression in Canada has been estimated to be between 3.1-9.6 % per year.[26] Others indicate that the prevalence of depressive symptomatology (which includes sub-clinical depressive symptoms as well as major depressive disorders) in the general population is over 20%.[27, 28] Despite the apparent discrepancies in the estimates of depression in the general population, it is obvious that a significant proportion of the population faces considerable symptoms of depression at one or more times in their life. It should be noted that from a

[††] World Health Organization. Depression [Online]. 2009 [cited 3 May 2009]. Available from URL: http://www.who.int/topics/depression/en/.

population perspective, sub-clinical depressive symptomatology has been shown to be responsible for a greater utilization of services and overall costs than actual clinical depression.[28, 29]

A clinical diagnosis of major depression is commonly made according to criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV); although, the exact causes of depression are not known. The gold standard assessment of major depression is a structured psychiatric interview. A structured psychiatric interview allows for the assessment of depressive symptoms giving consideration not only to symptomatology (as with self-report measures), but also to symptom duration, the functional impact of symptoms, and the personal perception of those symptoms. Nonetheless, researchers frequently utilize a variety of self-report measures which reflect the presence and degree of *depressive symptomatology* (as opposed to a clinical diagnosis of depression). Continuous outcomes of depressive symptomatology are often categorized to closely represent those who are likely to have an episode of clinical depression versus those who do not.[6, 30, 31] An example of a continuous measure that is often categorized, and is used in the research presented in this dissertation, is the Center for Epidemiological Studies - Depression Scale (CES-D).[30]

The CES-D scale is a validated and reliable tool for the assessment of depressive symptomatology in the general population and is commonly used in

epidemiological studies of depression.[6, 20, 27, 30, 32, 33] It is a 20-item self-report measure of depressive symptomatology with scores ranging from 0-60 on a continuous scale; higher scores indicate greater depressive symptomatology. It should be noted that the CES-D scale was not designed to serve as a diagnostic tool; however, when using a cut-off score of ≥16, the CES-D has been shown to be well correlated with episodes of clinical depression in the general population.[30] This cut-off has been used in past research, particularly in the area of work and disability.[20, 27, 32] Using a CES-D score of ≥ 16 to represent clinical depression is associated with good sensitivity (87% to 96%) but lower specificity (39% to 77%).[34, 35] Although this lower specificity might be subject to criticism, the fact remains that those with false-positive depression on the CES-D are more similar to true-positives than true-negatives in their current and future psychopathology.[36] The value of the CES-D scale is that it is a short form, well accepted self-report measure of depressive symptomatology that correlates well with other self-report measures of depression.[6] The CES-D scale has validated construct validity and can be generalized across a variety of subgroups including gender, race, and education level.[30] This scale is commonly used in musculoskeletal research since it has been validated with pain populations and is well accepted by participants.[33] Radloff (1977) reported good reliability with the CES-D and those without a negative life event had the high test-retest correlation in CES-D scores. The test-retest correlation declined if a life event was reported at one or more follow-ups (as should be expected if the CES-D is actually measuring 'current' depressive symptomatology).[30]

Theories of Depression

There is no consensus on the exact cause of depression. The development of depression, or significant symptoms of depression, occurs under a complex framework that involves many competing and complimentary theories. Although many sub-theories of depression exist in the literature, Dunn (2009) suggests that three *major* theories of depression currently exist to explain the aetiology of depression.[37] These three major theories include *biological theories, social and interpersonal theories*, and *cognitive theories*. Others also discuss a more recent and integrative perspective; a *diathesis-stress* theory of depression.[6, 8, 38] The following is a brief review of these four categories.

Theories of Depression - Biological Theories

Some view depression primarily in a biomedical paradigm. This position conceptualizes depression as primarily a biologically based phenomenon. Potential precursors to depression might include genetic factors (inheritance, or gene disturbance), neurophysiological dysfunction (disturbances in normal hormonal and neurotransmitter function), and neuroanatomical dysfunction.[6] Examples of biological causes of depression include disruptions in the noradrenergic systems of the brain (related to stress experiences), abnormalities in the seratoninergic (regulation of circadian rhythms) and dopaminergic (emotional expression and goal-directed behaviour) systems, as well as other neurotransmitter and hormonal abnormalities.[6] Specific to the research here is the relationship between pain and depression. Pain has long been associated with

depression and more recent studies have shown pain to be a risk factor for incident depression.[32, 39] When an injury is sustained, pain results. In fact, pain is likely the first indicator of an injury. From a biological perspective, pain results in a sequelae of hormonal pain-related and stress-related responses.[40] These responses eventually influence biological structures in the brain which have been shown to be important to mood (e.g. Hypothalamic-pituitary-adrenal axis).[6, 40]

Theories of Depression - Social and Interpersonal Theories

As an alternative perspective, in the influential work of Marmot and Wilkinson (2006) it is repeatedly demonstrated that social and interpersonal relationships are consistently associated with overall health and well-being; depression is but one example of an outcome that can be influenced by social and interpersonal factors.[41] The broad category of social and interpersonal models can refer to one's current close personal relationships (e.g. work colleagues and family members) or can extend to one's social position in their society (e.g. rich vs. poor, deprivation and/or social isolation). The contribution of life-stressors to depression can also be viewed within this category and there is no doubt that stressful life events contribute to depression. Kendler (1999) showed that those experiencing a stressful life event were 2.3 (serious personal crisis) to 25.4 (assault victims) times more likely to become depressed within one month of the adverse experience compared to who did not have a stressful life event.[42] Lastly, a predisposition toward depressive episodes is associated with early life experiences.[6, 43, 44] Although the early life experiences can refer to negative

biological exposures, these experiences are often discussed within the social context (e.g. parenting style, abuse, loss, etc.). In summary, in social and interpersonal theories of depression, depression and symptoms of depression are intimately tied to the stressors and strains of people's social worlds.[6]

Theories of Depression - Cognitive and Cognitive Behavioural Theories

Another prominent view of depression relates to a series of cognitive and cognitive behavioural theories. One such example is the cognitive behavioural theory proposed by Beck (1976).[45] In this view, depression is the end result of a chain reaction which is initiated by an experience of (physical, psychological interpersonal or social) loss.[45] The development of depression is more strongly attributed to an individual's internal dialogues and perceptions. Thus, depressive symptoms are related to cognitive biases in information processing.[6] For example:

> *"...the individual's problems [mood disorder] are derived largely from certain distortions of reality based on erroneous premises and assumptions... originated in defective learning during a person's cognitive development*".
> (Beck (1976), "Cognitive Therapy and the Emotional Disorders", pg: 3)[45]

Seligman's (1972) model of learned helplessness provides an additional framework from which to view the development of depressive symptoms and ties

closely to theories of *hopelessness*.[6, 46] Learned helplessness suggests that some humans (and animals) simply resign to negative stressors due to a perceived lack of control over the stressful event(s).[46,47] This skewed perception will lead individuals to think that negative outcomes are certain or, conversely, that positive outcomes are not a possibility. As with Beck's cognitive behavioural theory of depression, learned helplessness attributes depression more to the individual and focuses on the internal cognitions of those with, or at risk of developing, depression.

Theories of Depression - Diathesis Stress Model of Depression

The diathesis-stress theory of depression provides a theory that attempts to incorporate the interaction between personal attributes, or diathesis, and stressful life events in the development of depressive symptoms. In crude terms, the diathesis-stress theory links cognitive theories with social and interpersonal theories of depression.

> "*The basic premise is that stress activates a diathesis, transforming the potential of predisposition into the presence of psychopathology*."
> (Monroe and Simons (1991), Psychological Bulletin, pg. 406)[38]

Although Monroe and Simons suggest that "stress activates a diathesis", others have argued that this relationship is highly complex and not uni-

directional.[48] A recent study in the Journal of Clinical Psychology which sought to determine whether or not cognitive structure and stressful life events interact in the development of depression, suggested that an individual's cognitive structure (i.e. diathesis) is a relatively stable factor that *does* interact with stressful life events in the development of depressive symptoms.[49]

Theories of Depression – Summary

It becomes apparent when reviewing the theories of depression that the development and effects of depressive symptoms are extraordinarily complex. It appears to be widely believed that depression occurs within a complex biopsychosocial framework, and it should be noted that models of depression do not have to be considered mutually exclusive. A more likely position is that these models overlap and are likely context specific. For example, it is plausible that an individual's neurobiological function could adversely influence their cognitive interpretation of their current life circumstances. In other words, an individual might experience a depressed mood due to neurophysiological dysfunction; this depressed mood may in turn negatively influence an individual's cognitive processing with respect to stressful life events. Moreover, the predisposing factor of neurophysiological dysfunction might be the result of adverse social or personal experiences at a very young age, even in utero; creating a sequalae of events that negatively affect development and result in the said dysfunction.[‡‡, 38] In fact, Clark and Beck (2010) provide support for the inter-relatedness of

[‡‡] Goodman and Brand in Gotlib and Hammen (2009); ppg 249-274

cognitive and physiological aspects of depression; cognitive behavioural therapy appears to [positively] influence neurophysiology.[50]

How Does Depression Relate to Work and Injury?

This dissertation focuses on the potential causes of depressive symptoms through a biopsychosocial lens and does not ascribe to any one theory of depression. The attempt here is to investigate how loss of a regular social exposure (i.e. work) might contribute to symptoms of depression.

Prior research has indicated that both major depression and depressive symptoms delay recovery and subsequent return to work in those with a MSK injury.[21] It has been estimated that the incidence of depressive symptomatology in injured populations may be as high as 42-45% within 6-weeks of injury.[32, 51] These proportions are much higher than that observed in the general population (20-23%).[26, 27] Despite these differences, it does not appear that a great deal of research is available regarding the temporal relationships between time-off work and depressive symptoms, the course of depressive symptoms for those receiving compensation for an injury, or regarding the influence of work status on depressive symptoms. Specifically, we know from previous research that the presence of depressive symptoms is associated with delayed recovery and many individuals develop depressive symptoms shortly after an injury.[19-21, 32, 51] We do not know if time off work contributes to the development or persistence of depressive symptoms; potentially resulting in a longer duration of work absence, or vice versa. A recent review on the topic of physical disability resulting from

MSK disorders suggests early identification and treatment of depressive symptoms is crucial in minimizing disability duration in those recovering from a MSK condition.[22] However, little if any information is put forth in evaluating if prescribed time off work, as a health care intervention after MSK injury, has the unintended consequence of contributing to an individual's depressive symptoms. Does encouraging time away from work to recover from injury create or promote depression and/or the development of depressive symptoms in those who are recovering from a MSK injury; or does the endorsement of time away from work attenuate the development of depressive symptoms?

Research investigating the effect of work status on individual health outcomes has focused on the work-health relationship for the unemployed vs. the employed, for individuals nearing retirement, and for those who have already retired from work.[5, 9, 17, 18, 52-55] Absences from the work environment are consistently associated with greater all-cause mortality and poorer mental health; although, none of the studies have identified the existence or direction of a causal relationship. Research regarding the effect of work absence(s), for reasons other than retirement or unemployment, on health is limited. More frequently discussed are the potentially negatively influences of work on overall health and well-being[6, 8, 55] ; although, work can contribute to good health.[3] Acknowledgement of the importance of appropriately managing depressive symptoms of workers is reflected in a recent policy statement submitted by American authors.[35] Although they recognize the importance of depressive symptoms, they advocate for the

treatment/management of depression in the workplace without giving full consideration as to whether or not this is the most appropriate venue. These authors give primary consideration to cost minimization only. That being said, it would be expected that optimal management of depressive symptoms will be reflected by lower overall costs (accounting for lost productivity in addition to treatment costs). But what is optimal management?

With widespread societal costs, and the potential for negative impact on individual health, further investigation of how work might absences affect individual health is of utmost importance. This dissertation not only provides evidence related to the question: "For individuals recovering from a musculoskeletal injury, what role does time off work play in the development and resolution of depressive symptoms?" but also provides an introductory theoretical framework from which to view the relationship between work status and depression following injury.

1.2. Study Objectives

Depressive symptoms are closely related to longer recovery from injury; however, it is not clear if time off work contributes to or alleviates depressive symptoms following an injury. This dissertation presents three projects and four papers aimed at identifying the extent to which work status contributes to depressive symptoms. The goal of this dissertation is to provide further information regarding the course of depressive symptoms for those with a

musculoskeletal disorder and determine the direction of the relationship between being off-work and depressive symptoms (i.e. whether time off work promotes depressive symptoms or if depressive symptoms lead to a time away from the workforce). The first paper (chapter 2) discusses a theoretical perspective in which work absences provide a plausible causal mechanism for increased depressive symptomatology and/or depression. The subsequent hypotheses which are addressed include:

1. For those who are off work at least 6-weeks as a result of a MSK injury, return to work will be associated with a subsequent reduction in depressive symptoms, whereas those individuals who continue to remain off work during their recovery will not experience such a reduction in depressive symptoms.

2. The incidence of depressive symptomatology will be lower in those remaining at work during recovery from a MSK injury compared to those who take a work absence for their injury.

3. Individuals with depressive symptoms yet remaining at work during recovery from a MSK injury will exhibit a shorter course of depressive symptomatology compared to those with depressive symptoms and not at work.

4. For individuals with a MSK injury, those remaining at work while recovering will have less severe depressive symptoms compared to those who take a work absence while recovering.

It is the goal of this dissertation to provide evidence regarding the most appropriate use of time off work as an adjunctive intervention in the treatment of musculoskeletal disorders. This research will have meaningful clinical and financial implications in the management of musculoskeletal injuries.

1.3.Paper Overviews

Paper #1 – A Conceptual Discussion of Work and Depression

Many models of depression already exist in the literature.[6] The goal of this paper was to incorporate the phenomena of depression into context specific model and provide a visual framework illustrating the potential relationship between work status and depressive symptomatology after injury. This paper used modelling methodology presented by Jaccard and Jocoby (2010)[56] and focuses on personal and social characteristics supporting a relationship between work status and depression. The model developed here provides a rudimentary theory which visually demonstrates the manner by which a work absence could contribute to depressive symptoms following injury. The focus of this modelling exercise was to theorize on the interplay between an injured individual and their personal and social attributes, their work status, and subsequent depression status.

Paper #2 – Work status and depressive symptoms after injury: does work status play a role in the prognosis of depressive symptomatology after injury?

With a working model in place (paper #1) this paper meant to determine if in fact work absences after injury contribute to depressive symptoms during recovery. We followed a cohort of employed individuals from the general public who submitted an insurance claim for a whiplash injury resulting from a motor vehicle accident. To investigate the relationship between work status and depression, we built a series of logistic regression models to determine 1. the strength of the independent association between work status and depressive symptoms, and 2. how well past exposures to work predicts future depressive symptomatology.

Paper #3 – Work and depression in worker's compensation

Paper #3 serves not only as a confirmatory investigation with respect to paper #2, but also provides the opportunity to evaluate other variables and their influence on the work-depression relationship. In this paper, scores on the mental health component of the SF-36 were used to determine those likely to be depressed.[57, 58] In essence, the goal was to evaluate whether or not work status influences mental health functioning and depression as recorded on a valid, self-report health status measure in a population of workers compensation claimants.

Paper #4 – Methodological issues in assessing depression: controlling for baseline symptoms

As a general rule, when there are baseline measures of the outcome variable, researchers often include that baseline measure in their multivariable analysis when conducting statistical adjustments for confounding. However, Glymour et al (2005) makes a compelling argument that this strategy of controlling for baseline values of outcome scores in a follow-up studies introduces bias and that such analyses do not yield estimates of the effect of an exposure on the outcome variable.[59] This was a statistical dilemma faced in Paper #2 in this thesis. Paper #4 provides methodological examination of this issue, and a demonstration of how the decision of whether or not to adjust for baseline values of an outcome variable affects the estimate of the effect of the exposure.

1.4. Summary

Depressive symptoms are consistently associated with delayed recovery from musculoskeletal injury, but the role that work status plays in this relationship is not yet clear. There are valid arguments relating to both the potential positive and potential negative impacts an off-work prescription; however, this issue is far from resolved and additional research is necessary.

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Chapter 2 – A Conceptual Discussion of Work and Depression

2.1. Introduction

Investigating the relationships between work, injury, and mental health presents many challenges. Challenges are found in, but not limited to, the areas of study design, sampling, analysis, and measurement. This chapter focuses on further understanding exposure-outcome relationships through theory development. Specifically, this chapter centres on the personal and social influences that could impact a worker's emotional response to their work following injury. This perspective draws from historical social theories to help develop a practical framework from which to understand a human response (depressive symptoms) to a specific life event (injury) in the social context of work. Parts of the framework to be discussed are often implied in the injury-mental health arena, but rarely explicated.

A Theoretical Approach?

When a researcher pursues answers for questions of public health, that person is often directed to ask traditional questions with respect to person, place, and time (who, where, when).[1] These questions are often asked with pre-implied ideas of causal associations which are often limited to one particular level of analysis (e.g. focusing only on analysis at the individual); or even without much regard to how causal associations might operate. This type of investigation has been coined "black box" epidemiology.[2] Researchers frequently point to single

level relationships between exposures and outcomes at the whim of statistical analyses. Although this approach has been beneficial in the development of epidemiology, analysis without consideration (e.g. consideration through theoretical development) of causal influences puts full causal mechanism into unseen territory; thus, the "black box" analogy. Often forgotten are the philosophical and theoretical approaches that have advanced even the "hardest" of sciences (e.g. physics). Complexity is implied in the term "theoretical approach"; however, theories do not have to be complex. In fact, theories are nothing more than a set of statements describing the relationship(s) between two or more concepts.[3] Thus, complexity is not a requirement, but complexity is often ignored.[4]

The greatest challenge of modern day physics is cited as the merging of two seemingly incompatible theories of the physical world (quantum mechanics and general relativity) into a single unified theory.[5,6] Physicists hope to develop a single theory that will explain our physical surroundings from the largest planets to the smallest sub-atomic particle. Advancements in physics have been the result of embracing complexity and confronting it head on. Public health incorporates a multitude of layers for consideration; from the atomic to the biochemical, and from the personal to the social, institutional, and organizational. The public health scientist must consider both micro and macro levels (and those in between) and must often also deal with organizational structures and limitations. As public health scientists, we too must confront the complexities of our science.

This chapter attempts to avoid *black box* epidemiology by proposing an introductory theoretical model to explain the interrelatedness of individual and *social* factors connected to injury, work, and mental health. This model will be visualized using methods proposed by Jaccard and Jocoby (2010).[3] There is a need for theories which incorporate the complexity of the phenomena we are studying.[4] The following is an attempt to begin to address some of the complexities associated with work, injury, and mental health.

Injury and Mental Health

One might question the proposition that remaining at work after injury could be protective against the development or progression of depressive symptoms. This proposition might seem even more absurd in view of recent media reports of record job dissatisfaction among North American employees[7] coupled with rising levels of work-related stress, burnout, and depression.[8, 9] Recent texts reinforce the focus on work as a negative stressor. For example, in the book "Stress and Mental Health" there is a chapter dedicated to "The Daily Grind…", focusing entirely on workplace stressors.[10] On the face of it, getting away from work, whether through vacation, early retirement or injury, seems like the best thing that could happen.

Consider a manual labourer with a sprained ankle. Assume this individual has employee benefits and, as such, they receive a portion of their pre-injury income (this could occur through private disability insurance or worker's compensation,

depending on how and where the injury occurred). Now, would it not seem to be a positive turn of events for this individual to have time away from the stresses of work while avoiding the bitter cold (this injury is in winter) all while receiving a weekly paycheck; as a result of *only* a minor injury? Yet, it may not be that simple.

Depressive symptoms are endemic in the general population. One-week prevalence of depressive symptoms in the general population has been reported to exceed 20%.[11] Depressed mood (including subclinical depression) is a significant risk factor for clinical depression and because of its high prevalence in the general population, constitutes a greater societal burden than major depressive disorder.[12-15,16] What's more, greater than 40% of individuals report depressive symptoms shortly after injury.[17,18]

A recent review of work and health concluded that work is central to individuals' identity, social roles and social status.[19] Prior research has pointed to numerous factors being associated with post-injury depressive symptomatology.[§§] These factors include a history of mental health problems, anxiety, poor general health, and injury related symptoms (including pain).[20] For individuals who go off work after an injury, depression (and/or depressive symptoms) has also been shown to be associated with delayed return to work (RTW).[21] This premise can

[§§] Depressive symptomatology refers to a sum of depressive symptoms that correlates strongly with clinical depression

be turned on its head by asking "how does being off work influence depressive symptoms following injury?". To add further complication to the issue, one must consider that many work-related factors can contribute to stress and depression; these include job dissatisfaction, extreme job demands, low decision latitude (self direction at work), and anxiety about returning to work (to name a few).[10, 22, 24] To fully understand these relationships the complexities of their inter-relationships must be considered:

> "*Risk factors for disease do not operate in isolation but occur in a particular population context. Individual 'lifestyle' can only be understood in the historical, cultural, and social context in which it occurs.*"
>
> (Pearce and Merletti (2006) - Int J Epidemiol, pg. 517)[4]

The social sciences provide an excellent resource for existing theories that help explain why being of work *might* contribute to an increase in depressive symptoms. Relevant sociological theories include Goffman's "*Frame Analysis*" and Merton's "*Role-Sets*".[25,26] Additionally there is the widely held sociological belief in "identity theory", something Merton (1957) describes as a "basic fact" of sociology.[26] Before introducing the context specific theory, implicit to this dissertation, these three theories are briefly reviewed.

Identity Theory and Role Sets

Identity theory and the theory of role sets are included under one heading as they are intimately related and, for the current purposes, can be constructively discussed together. Identity theory suggests that each one of us occupies many roles (identities) in our lives.[27, 28] Merton refers to this relationship as the idea that each person occupies a variety of "statuses" and each status is associated with a particular role.[26] Identity theory and the theory of role-sets comprise slightly different articulations of a similar underlying concept; people, throughout their lives, participate in a variety of roles for which they divide certain attentions to and subsequently use to apply particular meaning(s) to who they are. Identities (roles) provide a standard for who one is.[27] Using this writer as an example; I am a student, a researcher, spouse, son, father, hockey player, etc. Because of the meaning individuals attach to their life roles, an alteration in one or more roles can be perceived as a loss (or gain) and as subsequently contributing to, or taking away from, one's well-being.[26, 29-32]

Merton (1957) draws an important distinction between *identity theory* and *role sets*.[26] Specifically, Merton illustrates that within each role we occupy, we are exposed to a number of other relationships; these comprise the "role-set" for a given role. For example, in my role as a student it is expected that I will have interactions with a supervisor, doctoral committee, faculty administration, etc. In effect, our roles (identities) and role-sets combine to form our social structure.[30] Building on this, social theorists suggest that our participation in these roles is

governed by expected behaviours.[26, 30] We can have expectations of ourselves and we will also have expectations of others within our role-set. Likewise, those within our role-set will have expectations of us. Even more specifically:

> *"unlike the problems centred upon the notion of 'multiple roles'* [identity theory], *this one* [role-sets] *is concerned with social arrangements integrating the expectations of those in the role-set"*.
>
> (Merton (2006) – Brit J Sociol, pg. 112)[26]

Although there appears to be significant overlap, the idea of role-sets more strongly addresses social dynamics like power differences, social supports, and conflicting demands.[26] Identity theory remains more relevant to how individuals perceive, and indentify with, themselves.

Goffman's Frame Analysis

In 1974 Erving Goffman introduced his theory of "Frame Analysis".[25] Goffman took this opportunity to introduce the idea that not only can individual actions and behaviours be viewed through a variety of lenses (frames of analysis – arguably Goffman's focus), but within these frames there are is a principle of organization which directs social interactions and an individual's perceived involvement in those social interactions.[25] In essence, the way in which we relate to an activity is a result of how we *frame* the activity.[33] As Goffman suggests, a

punch to the arm can be given in jest but it is the social context of the action, in addition to how individual receiving the blow interprets the joke (how they frame it), that will be the ultimate judge of whether or not the action was really a joke. Goffman's discussion of frames also seems to incorporate elements of Merton's "role sets" as well as "identity theory". Consider the following excerpt from Goffman (1974)[25]:

> *"… it has been argued that an individual's framing of an activity establishes its meaningfulness to him. Frame, however, organizes more than meaning, it also organizes involvement… it is understandable that the unmanageable might occur, an occurrence which cannot be effectively ignored and to which a frame cannot be applied, with resulting bewilderment and chagrin on the part of the participants… it is apparent that the human body is one of those things that can disrupt the organization and break the frame…"*
>
> (Goffman (1974) - "Frame Analysis" ppg. 345-347)[25]

Here, Goffman alludes to the fact that each of us partakes in various life roles. How we frame the activities we are involved in organizes us and provides meaning for that activity. It does not seem much of a stretch to imagine that injury could be the component of the human body that disrupts an individual's natural organization. Furthermore, work could be an example of an activity frame

that is filled with meaning and involvement. Work itself will also fill a particular social frame. In his discussion "The Manufacture of Negative Experience", Goffman illustrates how this "breaking of frame" can be detrimental.[25] The following is immensely applicable to work, injury, and mental health. Imagine the "sudden change" an injury, and the resultant inability of the worker to frame his or her experience within the usual social frames.[25, 33] In reference to "breaking frame":

> *"…belief suddenly changes… he becomes unreservedly engrossed both in his failure to sustain appropriate behaviour and in the cause of this failure. He is thrust into his predicament without the usual defences. Expecting to take up a position in a well-framed realm, he finds that no particular frame is immediately applicable, or the frame he thought was applicable no longer seems to be… He loses command over the formulation of viable response. He flounders… He has a "negative experience" – negative in the sense in it takes character from what it is not, and what it is not is an organized and organizationally affirmed response."*
>
> (Goffman (1974) - "Frame Analysis" pp. 378-379)[25]

In this part of the discussion, Goffman makes reference to "appropriate behaviour" and "expecting to take up…". Goffman's discussion is much broader

than expectations in general, yet this quote forces us to consider what is meant by expectations. Within the context of work and work injury, it is very likely that "appropriate behaviour(s)" are those behaviours others expect of us. These external expectations likely stem from those within a given role-set. Conversely, each individual will have his/her own expectations, not only for themselves (their identity) but for those within their role-set. An injury, for example, might lead us to recognize that we are now unable to meet not only our own work-related expectations, but the expectations of those within our work-related role-set. It is likely that a worker is not usually prepared for the change in social structure that results from an injury, and he "flounders". There is continuous interaction between self imposed expectations, an individual's perceptions of self identity, and between the individual and the expectations stemming from others who appear in their role-relevant role-sets. Additionally, the impact of these aforementioned interactions may be regulated according to how the individual frames the event and the event's outcome(s). These theories invariably merge.

Work, Injury, and Mental Health

What does all of this mean for work, injury, and mental health? As identity theory would suggest, work is an important role that many individuals identify with and use as a source of self-esteem.[19] However, work is not the only role we identify with and thus, there is the potential for role conflict.[27, 28, 31] Additionally, as the result of being part of the workforce, there are a number of expectations coming from other sources; expectations placed upon the worker. A worker is

confronted with expectations from his colleagues, supervisors, clients, and possibly his significant other; all as result of his employment. This is the worker's role set. Lastly, events affecting one's ability to work are likely to impact individuals in proportion to the value they attribute to their job, their relationships at work, and how important people in the worker's life perceive the event to be (enter frames and framing).

The previous paragraph outlines a basic framework for the theory to be proposed here. Although there are many previously illustrated relationships between injury and depression,[10, 11, 17, 20-24] these prior models do not focus on the interplay of injury and depression with work. Many attributes of the upcoming theory are addressed by the likes of Goffman, Merton, and the greater community of social philosophers. The theory presented within this chapter can be used as a road map for investigation, analysis, and discussion; its novelty comes from its specificity to the injured worker context. This theory attempts to include the most applicable and pragmatic aspects of the previously discussed theories in combination with clinical (as a practicing physical therapist) and academic experiences.

2.2. Model Building: Development of a Theory

Finally, A Theory

In the scheme of building a visual model or theory, a few key points need to be discussed. A direct causal relationship will be illustrated by a single direction

arrow extending from the exposure (X) to the proposed outcome (Y) (e.g. X→Y). When the relationship between X and Y varies according to a third variable, Z, this third variable is considered a moderator.[3] For example if treatment X influenced outcome Y depending on an individual's gender, then gender would be considered a moderator (similar to effect modification). A moderator is illustrated by intersecting lines. A mediating variable is a mechanism through which an exposure would lead to an outcome. For example: X → Y → Z; in this scenario X influences Z through its influence on Y. In this case, Y is a mediating variable.[3]

From this point forward depression and depressive symptoms will be used interchangeably, but ultimately in reference to a sum of depressive symptoms. "Framing" will be used as a synonym for an individual's perceptions (how they frame something) at various points in the model.[***] The first premise is that a change in work status after injury can contribute to depressive symptoms or delay recovery from depressive symptoms. *Work status* refers to going off work during recovery versus staying at work during recovery. It is clear that that injury will likely also contribute to depression independently. The injury-depression relationship is an important one and an in-depth investigation on this front will need to recognize the individual contributions of injury vs. work status in relation to depressive symptoms; however, the focus here is on the work-depression

[***] A major component of Goffman's "Frame Analysis" was the proposition that we (the researchers) could also analyze social relationships through different frames, or levels of social interaction; this is not part of the discussion here.

relationship following injury[†††]. The basic idea is illustrated by the causal model presented below.

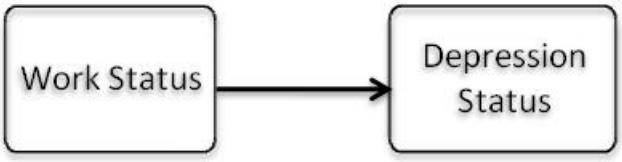

Figure 2.1 – Work Status and Depression: An Initial Model

Note that injury is not yet considered in this model. The model being presented is meant to represent those with an injury and there will be a variety of factors that moderate the relationship between injury and work status (e.g. job demands, prior health, work/return-to-work expectations); however, the focus is not on how injury might relate to work status, but how work status might influence depression. At this level one can already begin to see how identity theory, role-sets, and framing come into play. First, an individual could closely relate their personal identity to their job. The loss of their ability to work might then contribute to depressed mood because they are no longer able to meet their own expectations. On the other hand, an individual might more closely identify with a sick role as a result of injury. Should they remain at work, they too [the "sick" person] may develop symptoms of depression; again as a result of a mismatch between expectations and reality. This also illustrates some elements of

[†††] The focus here is on the work-role. Practically, it seems any investigation on this front would also need to consider how an injuries impact on other life-roles could mediate the injury-depression relationship.

framing. Arguably, the first individual more closely represents one who "flounders" as a result of their inability to control the disorganization that ensues as a result of being off work. This individual's organizational frames are heavily weighted to his working role. The second individual might be a person who is too adaptive. He or she may have shifted frames too quickly; from work-role to sick-role. Similar to the first example, this second individual also becomes "engrossed both in his failure to sustain appropriate behaviour [the sick-role or the work-role] and in the cause of this failure [the injury]". In the second case the *appropriate* behaviour is as someone who is sick rather than as someone who works. From this we can draw the following:

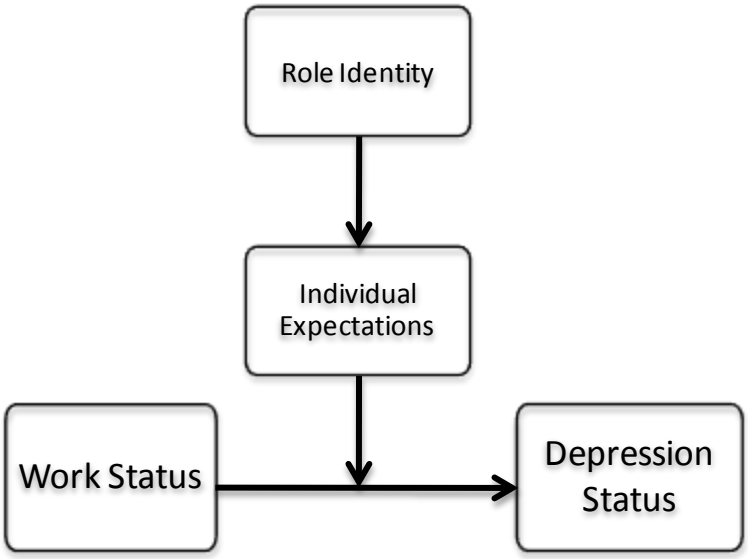

Figure 2.2 – Work Status and Depression: Model 1

Here the relationship between work status and depression is moderated (i.e. effect modification) by work and/or return-to-work expectations as discussed above. However, role identity is an important contributor to self appraised

expectations and will also moderate the work-depression relationship through expectations.[27, 28] In other words, role identity will act as a mediated-moderator through its relationship to role expectations.[3] Expressly, in this conceptualization, an individual's role-identity will contribute to their self-expectations with respect to work. It has been shown that positive RTW expectations can lead to faster self-reported recovery; we also know depression is associated with recovery expectations.[35,36] These concepts are not illustrated here. This is a result of asking a slightly different question; "How does (or could, i.e. pending ongoing research) a worker's social organization influence their emotional response after injury?" versus "Do positive expectations improve the rate of recovery" or "What factors are associated with recovery expectations".[35, 36] Arguably, the latter question is imbedded in the "individual expectations" box.

When evaluating an individual's expectations we must consider the expectations of those in the worker's role-set. The *basic* work-related role-set might include the worker's significant other, their colleagues, and their supervisor(s). The expectations from these three groups are likely to influence how much a person's work status influences their mood after injury. It also seems reasonable that others' expectations are mediated by our own expectations within the context given here. In effect, what others think influences what we think and vice versa. There is reciprocity in expectations. It seems likely that the '*self expectations – expectations of others*' relationship is moderated by how we *frame* these relationships. Simply, if I don't care (for whatever reason) what others

think (framing their expectations negatively) then the relationship between their expectations and mine will be quite weak; the opposite will be true for those who want to meet the expectations of others but can't. This follows a line of thinking presented by sociologist Peggy Thoits;

> "*Failing to meet normative expectations in identity performance should decrease self-esteem*".
> (Thoits (1991), Am Sociol Rev, pg. 105)[28]

If someone disapproves of themselves (lowered self-esteem) as a result of failing to meet their role expectations, it follows that they are likely to suffer psychological distress.[32] Thus:

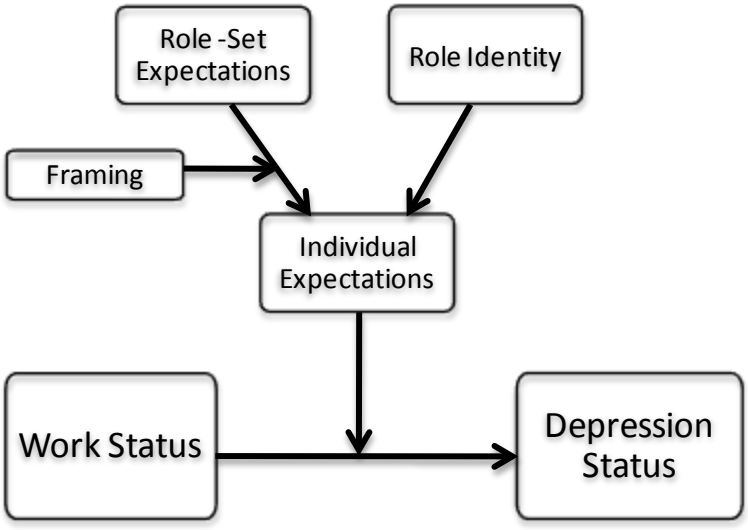

Figure 2.3 – Work Status and Depression: Model 2

The focus of this model is how work status could influence depressive symptoms following injury in relation to social structures and social influences. Absent from this model is the "larger" societal context, our culture :

> "*cultural beliefs shape and integrate the expectations that pattern the relationships among a social structure's constituent statuses and roles*".[30]
> (Schooler (1994), Social Psychology Quarterly, pg. 263)

Societal beliefs are important for consideration in two areas related to this model: 1. Framing – the manner in which we frame events (how important we perceive an event to be) is likely influenced by our culture.[30] For example, someone might not value the expectations of those in their role-set; why would this be? This could be a result of the greater cultural belief that it is not important what others think, you have to look out for yourself. If you subscribe to this belief (as a result of cultural influence) then the expectations of those in your role-set are unlikely to influence your individual expectations. 2. Injury – events like pain and hunger (and injury), are often considered outside of the social realm; however, these events are also subject to social influences and interpretations.[33] In this case, injury can be interpreted through the influences of cultural beliefs. Can I be *myself* while recovering from injury? This idea will be strongly guided by the role being evaluated in combination with current cultural beliefs. The ideas of maintaining a role-identity can also be influenced by the beliefs of sub-

cultures (e.g. the medical system vs. the hockey culture). These examples intentionally compare different sub-cultures. The medical system is designed to diagnose and treat. As a result, someone with an injury becomes "sick". Diagnoses are a substantiation of illness and, accordingly, people are pulled toward a sick role. On the other hand, hockey culture sees injury as a mild inconvenience and cultural pressures push the injured individual to maintain their role, to "suck-it-up" and carry on. These two approaches will obviously have their good and bad attributes, but that is for another discussion. Thus, injury will exert its influence directly onto an individual's role identity; and will be moderated via relevant sub-cultural beliefs and expectations. "Culture" could be envisioned as encompassing the entire model with arrows pointing toward each box represented below (not shown). These ideas are presented in the model below:

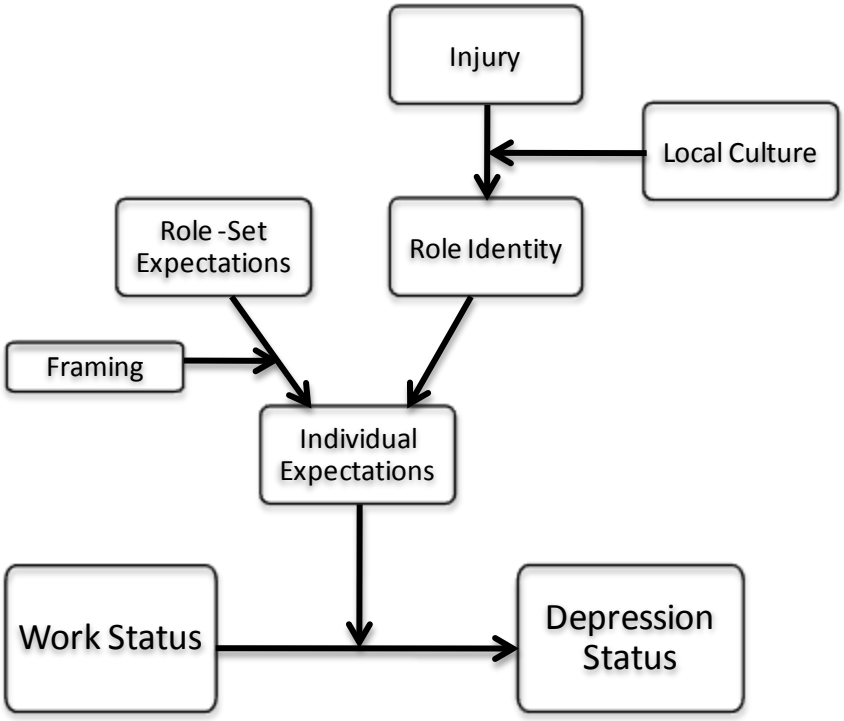

Figure 2.4 – Work Status and Depression: Model 3

Future Directions

The purpose of this model was to provide a framework for discussion and analysis with an emphasis on how social structures related to a person's job might influence depressive symptoms. It is a work in progress. The benefit of theorizing in this way is that it forces consideration of influences one would not have recognized otherwise. Furthermore, approaching social phenomena in this way helps to provide a slew of other relevant research questions.

The following is a list of questions/areas of enquiry that emerge when considering the model above:

- Is work status actually associated with depression status or psychological distress? Is the presence or lack of association consistent across subgroups (e.g. motor vehicle collision injuries versus occupational injuries)?
    - These questions are addressed in the next two chapters.
- To what extent do others' expectations influence the worker's expectations (are these visualized appropriately in the model?)?
- It seems the internal interpretation of work status could easily be complicated by ambiguous "normative expectations".
    - What are the worker's "normative expectations" with respect to work status after injury (note this is different than expecting to RTW... this asks the question, if you were injured, would you expect time off work?)?
- Rehabilitation professionals advocate prompt return to work after injury and may also advocate that the workplace be used as a place of rehabilitation.[19, 37] However, a diagnosis is often required before appropriate treatment can commence.
    - Does getting a diagnosis validate "sickness"?
    - What are worker's impressions of these conflicting expectations ("stay at work" versus "you are sick/injured")?
    - Do these conflicts lead to uncertainty; and if so, what is the result of this uncertainty?
    - What can be done to prepare worker for these pending interactions?

- Would preparing workers in some way minimize negative reactions to injury and/or a change in work status?

- How much self-identity value do worker's generally put on their role at work?
    - Should we be trying to assess a worker's 'work identity' when investigating work-injury-health relationships?

The scope of these questions is far too extensive to address in the current body of work. However, a beginning can be made in focusing on the first question, which forms the body of the current thesis.

2.3. Conclusion

Injury has important implications in mental health research for those in the workforce and can also be grounded in social theory. As a start for exploring the impact of work decisions for an injured worker, an initial approach involved the development of a visual conceptual model; a potential roadmap for discussion, debate, and ultimately future research. The introduction listed a series of factors which have been shown to be either directly or indirectly related to work stress and depressive symptoms. These factors included job dissatisfaction, extreme job demands, low decision latitude (self direction at work), and return-to-work anxiety. These factors were intentionally excluded from the modelling here with the idea that they are strongly reflected (i.e. inherently included) in the interplay between a worker's expectations and the worker's role-set expectations. As a

result, and in relation to most any visual model, the boxes presented here can be opened for further analysis. As the relationships between an individual's biology, psychology, and sociology are often very complex, we (as philosophers and researchers) are often limited to hierarchical models which include some details and omit others; we are forced to consider "boxes within boxes".[34]

S.E. Frost Jr. (1962) argues that everyone is a philosopher:

> "*Everyone, whether he be a plowman or banker, clerk or captain, citizen or ruler, is, in a real sense, a philosopher.*" (Frost (1962), "Basic Teachings of the Great Philosophers", pg. 1)[38]

Frost further suggests that this is part of being human.[38] This paper provides a portion of the philosophy behind the dissertation.

The attempt made here was to incorporate important social science philosophy into the questioning of phenomena traditionally viewed in the scope of biomedical model ideology. On a personal note, this has been a journey for me. Prior to this exercise, I would have viewed myself as a clinician who traditionally viewed work status as a result of injury severity and who gave little credence (until recently) to the idea that social roles, expectations, and individual perceptions could play such a significant role in the development of depression after injury. The model and ideas proposed here are preliminary, fairly crude and likely ripe

for criticism. This is what models are for; to be constructively criticised so that we as researchers and philosophers can re-visit our ideas, in light of the criticism, and further revise these ideas in hopes of better understanding and reflecting the world around us. Despite these limitations, it has been my goal to bring a fresh perspective to the realm of work, injury, and mental health. Bradford-Hill (1965) suggests that in order for an exposure to be considered a cause for a particular outcome, the cause effect relationship should be plausible.[39] Although Bradford-Hill was primarily making reference to biological plausibility, there is reason to consider plausibility in the social context. This paper provides some insight into the *plausibility* of work status contributing depression status. The next step is to determine if, in fact, work status can contribute to depression. That is the goal of the next paper of this dissertation.

Lastly, this section ends with a quote which represents an eloquent summary highlighting what can result from a mismatch between expectations and our actual experiences. It opens the question of how much general misery is engendered through our cultural perpetuation of inappropriate expectations, coupled with the inability of a great number of individuals to recognize the illusory nature of these expectations.

> *"I left [a company] newly aware of the unthinking cruelty discreetly coiled within the magnanimous bourgeois assurance that everyone can discover happiness through work and love.*

It isn't that these two entities are invariably incapable of delivering fulfillment, only that they almost never do so. And when an exception is misrepresented as a rule, our individual misfortunes, instead of seeming to us quasi-inevitable aspects of life, will weigh down on us like particular curses."

(Alain de Botton (2009) – "The Pleasures and Sorrows of Work" pg.127)

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Chapter 3 - Work status and depressive symptoms after injury: does work status play a role in the prognosis of depressive symptomatology after injury?

3.1. Introduction

For the majority of Canadians, work is a staple of everyday life. The ability to remain employed is essential for the necessities of life and may contribute to healthy socialization, daily physical activity and a sense of productivity.[1-3] A musculoskeletal (MSK) injury can impede one's ability to remain working at one's normal job and MSK injuries account for the majority of worker's compensation claims in Canada.[4] MSK conditions are also the leading cause of disability claims in Australia and the United States.[5]

Recovery from injury evolves in a complex psychosocial environment of which relatively little is known. What is known is that the longer an individual remains off work following an injury, the less likely it is that they will ever return to work.[6, 7] The association between MSK injuries and depressive symptoms is also well known.[8, 9] Depression has been shown to be a risk factor for troublesome pain, delayed recovery from injury, and is associated with a prolonged or complicated return to work following injury.[8, 10, 11] Furthermore, depressive symptomatology is common in the first days and weeks after a MSK injury.[8, 9]

Despite the previous research in this area, the direction of the relationships between pain, depression, and return to work following injury remains unclear.[11, 12] One postulate is that remaining off work during recovery will reduce the risk of depression as the non-working injured individual will have adequate time to seek appropriate treatment(s) while being removed from the stresses of the workplace.[1] Conversely, being off work often results in reduced financial resources, increased social isolation, inactivity, and increased anxiety about eventually returning to work which would suggest that being off work might increase the risk of depression.[1, 3]

In order to better understand the relationship between work status and depression during recovery from injury, we build upon previous research by following employed individuals with traffic-related whiplash-associated disorders (WAD). Our intention was to assess the relationship between being off work due to the injury and subsequent depression status. We hypothesized that remaining at or returning to work after a whiplash injury would be associated with less depressive symptomatology in the first three months following the injury. Our main goal was to assess the independent relationship between work status and an outcome of subsequent depressive symptomatology at six-weeks and three-months following injury.

3.2. Methods

Study Population

The study population consisted of a cohort of individuals making personal injury insurance claims for traffic-related whiplash injuries in the Canadian province of Saskatchewan. Two sub-cohorts were followed; one of which was depressed at the time of their initial injury claim (the first measurement point following the injury) and the second of which was not depressed at the time of their claim. The exposure of interest was being off work due to the injury (versus remaining at work); the analyzed outcomes were subsequent resolution of depression (for the group depressed at baseline) and development of depression (for the group not depressed at baseline).

Selection of the initial whiplash cohort is described elsewhere.[8, 13, 14] In brief, the whiplash cohort consisted of all Saskatchewan residents, 18-years or older, who made an injury claim for traffic-related whiplash between December 1, 1997 and November 30, 1999. Claims were processed by the government of Saskatchewan's provincially administered insurance system which, at that time, was a 'no-fault' insurance scheme (in this no fault system, health care and wage loss benefits were provided for all injured individuals, regardless of fault for the crash, and there was no compensation or litigation for pain and suffering). Those with serious injury (requiring extended hospitalization) or those unable to participate due to insufficient English language proficiency or serious unrelated illness were excluded. From the initial whiplash cohort (n=6021) we included

only those reporting either full-time or part-time paid employment at the time of their injury.

Data Sources and Follow-up

The information used for this study was self-reported. Baseline information was recorded by the participant as part of the 'Application for Benefits' form, and was obtained at the time of the participant's initial insurance claim, a median of 11-days post-injury. We had no information on how much compensation was granted. Participants reported on their pre-injury health, demographic and socioeconomic characteristics, pain (11-point numerical pain rating scale [NPRS]), work status, and depressive symptomatology. Subsequently, consenting participants completed follow-up questionnaires via structured telephone interview at six-weeks and three-months. Telephone interviewers were trained, monitored, and naïve to the study hypotheses.

Predictor of Interest

The main outcome, depressive symptomatology, was defined as a score of ≥ 16 on the Center for Epidemiologic Studies Depression Scale (CES-D) as measured at six-weeks and again at three months post-crash.[15] Although the CES-D was not designed as a tool for diagnosing major depression, scoring ≥ 16 on the CES-D is suggestive of depression.[15-19] When compared to DSM-IV criteria, a cut-off score of ≥ 16 was shown to have a sensitivity of 86.7% and a specificity of 76.6% for major depression in adults.[16] This cut-off is consistent

with previous research in this population and was used to form our baseline cohorts of "depressed" (baseline CES-D $\geq$ 16) and "not depressed" (baseline CES-D < 16).[8, 11, 13, 14, 17-19] For the purposes of this paper, the terms "depressive symptomatology" and "depressed / depression" are used interchangeably.

In addition to being grouped by baseline depression status, participants were further grouped by self-reported post-injury work status, the main exposure of interest. Participant's work status fell into one of four categories post injury: 1. Stayed at work following injury, 2. Initially at work followed by an off-work period, 3. Initially off-work followed by a return-to-work (RTW), 4. Stayed off-work. These comparisons were made from *baseline to six-weeks*. Work status to six weeks was used in a predictive model which assessed the risk of remaining depressed or becoming depressed at 3-months. Figure 3.1 outlines the grouping process.

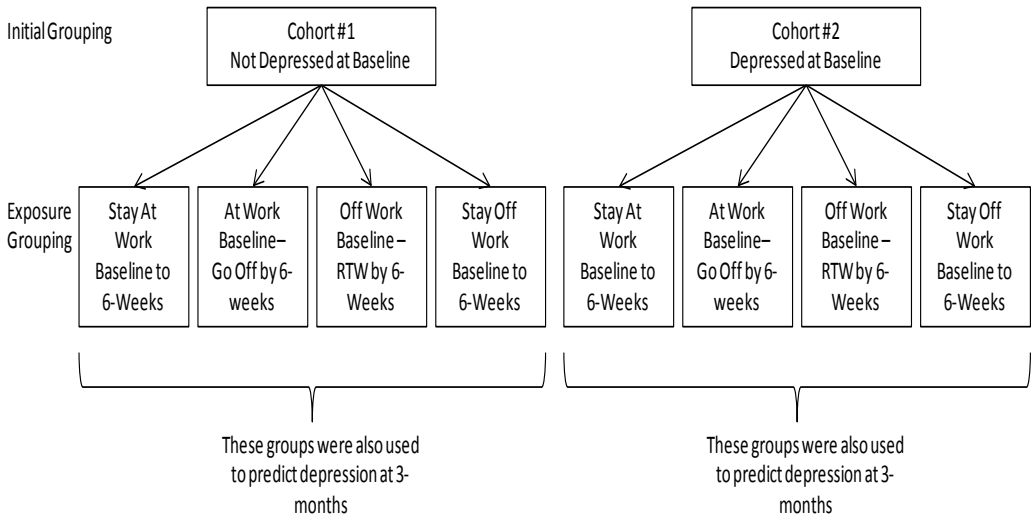

Figure 3.1. Cohort Selection and Exposure Grouping

Analysis

In order to assess the *independent* effect of work status on the main outcome, depressive symptomatology (depression), two multivariable logistic regression models were analyzed for each baseline sub-cohort (one for the outcome at six-weeks and one for three-months). The modeling strategy was as follows: We initially assessed the strength of the association between the exposure (work status) and our outcome (depression) via univariable logistic regression. Potential confounders were then selected according to prior research and theory (listed below).[13, 20, 21] The following self-reported attributes were assessed for potential confounding: education; marital status; income; impact direction; post crash numbness, dizziness, vision or hearing problems (yes/no); post crash anxiety (yes/no); prior mental health (absent/mild/severe); general health (excellent, very-good, good, fair-to-poor); % body in pain (assessed via pain diagram)[23, 24]; neck pain and headache pain (via numerical pain rating scale [NPRS])[23]; self-reported

recovery[25]; and satisfaction with pre-injury work (satisfied/not-satisfied). A series of multivariable logistic regression models were used to analyze each potential confounder's influence on the relationship between work status and subsequent depression in the following manner.

$$Logit\ [Odds_{depression}|Work\ Status] = \beta_1(Work\ Status) + \beta_2(Confounder)$$

A variable was considered as a confounder if it changed the relationship, as expressed by an odds ratio, between work-status and depression by ≥10%.[21] In order to estimate the crude and adjusted odds of depression at six-weeks and three-months, this procedure was repeated for the outcome at six-weeks and again at three-months post-injury. The final models assessed the association between work status and depression outcome, adjusting for all variables identified as confounders in the previous step.

3.3. Results

The initial cohort consisted of 4,271 individuals who reported part-time or full-time employment just prior to the injury. Fifty-six percent (56%, n=2411) of these employed individuals scored <16 on the CES-D at the time of their injury claim (baseline), and thus entered the study as the "not depressed at baseline" cohort. The remaining 44% (n=1860) scored ≥16 on the CES-D at the time of injury claim and entered the study as the "depressed at baseline" cohort. Each study cohort is described in Table 1.

Table 3.1. Baseline Characteristics (n=4,271)[†]

		Cohort 1 Not Depressed (n=2411)	Cohort 2 Depressed (n=1860)
Age (years) [n(%)]	< 24	492 (20.4)	433 (23.3)
	24 < 30	344 (14.2)	307 (16.5)
	30 < 40	615 (25.5)	487 (26.2)
	40 < 50	588 (24.4)	388 (20.9)
	≥ 50	372 (15.4)	245 (13.2)
Gender [n(%)]	Female	1532 (63.2)	1226 (65.9)
	Male	888 (36.8)	634 (34.1)
Education [n(%)]	< High School	362 (15.0)	354 (19.1)
	High School Grad	603 (25.0)	487 (26.2)
	Some Post Second	1046 (43.4)	807 (43.5)
	University Grad	397 (16.5)	209 (11.3)
Marital Status [n(%)]	Single	768 (31.9)	733 (39.5)
	Married	1396 (57.9)	898 (48.3)
	Separated	247 (10.2)	227 (12.2)
Income [n(%)]	$0 - $20,000	486 (20.7)	553 (30.4)
	$20,001 - $40,000	735 (31.3)	616 (33.9)
	$40,001 - $60,000	588 (25.1)	369 (20.3)
	above $60,000	538 (22.9)	281 (15.5)
Prior General Health [n(%)]	Excellent	930 (38.6)	645 (34.7)

	Very Good	959 (39.8)	727 (39.1)
	Good	437 (18.1)	396 (21.3)
	Fair - Poor	85 (3.5)	91 (4.9)
Prior Mental Health Issues [n(%)]	Yes	146 (6.1)	248 (13.3)
	No	2265 (93.9)	1612 (86.7)
Off Work at Baseline [n(%)]	Yes	578 (24.1)	862 (46.5)
	No	1825 (75.9)	992 (53.5)
Rated Neck Pain* [mean (sd)]		5.9 (2.0)	7.0 (1.9)

[†] not all variable cells add to 4,271 due to missing data

* 11-point numerical rating scale

Cohort #1 – Not Depressed at Baseline

Six Weeks Following Injury

Follow-up information on depression and work-status at six-weeks was available for 83% (n=1997) of those who were employed and not depressed at baseline. At six-weeks, the proportion of those who stayed at work, started at work and went off, started off-work and returned, and stayed off work was 74.5%, 1.4%, 14.7%, and 9.4% respectively. Of those scoring "not depressed" at baseline, 7.4% (n=147) reported depressive symptomatology at their six-week follow-up.

In the crude analysis, those staying off work after injury were almost five-times more likely to become depressed at six-weeks compared to those who stayed at work (see Table 2). This relationship was still observed after adjusting for aggregate confounding (adjusted OR = 3.55, 95% CI 2.22, 5.70). Crude analysis also showed that those who went off work initially but returned to work had slightly increased odds of depressive symptomatology compared to those who stayed at work after the crash; this association was not significant when adjusting for aggregate confounding (adjusted OR = 1.19, 95% CI 0.72, 1.96). Those who were at work at baseline, then went off work at some point during the first six weeks post injury were no more or less likely, in either the crude or the adjusted analysis, to become depressed than those staying at work throughout this period (Table 2).

Three Months Following Injury

Seventy-five percent (1803 of 2411) of participants who were not depressed at baseline completed the CES-D at three-months. Of these, 6.4% (n=116) scored ≥16 on the CES-D at three-months. Sixty-five percent (n=75) of those reporting depressive symptomatology at three-months were incident cases of depressive symptomatology, meaning that the remaining forty-one individuals who were depressed at three-months were also depressed at six-weeks. After adjusting for aggregate confounding, none of the work status groups differed from those who stayed at work in their risk of depression at three months, although there was a

crude association between staying off work and presence of depression at three months for those who were not depressed at baseline.

Despite an apparent relationship in our crude analysis, those who were not depressed at baseline yet stayed off work for at least six-weeks following their injury were no more likely to become depressed at three months than those who remained working after adjusting for aggregate confounding (Crude OR = 2.90, 95%CI 1.78, 4.75; versus Adjusted OR = 1.27, 95%CI 0.70, 2.33). These results are summarized in Table 3.2:

Table 3.2. Crude and adjusted odds of developing depressive symptomatology six-weeks and three-months following whiplash injury

	Six-weeks After Injury*		Three-months After Injury**	
	Crude OR (95% CI)	Adjusted (95% CI)	Crude OR (95% CI)	Adjusted (95% CI)
Stay At Work	1.0	1.0	1.0	1.0
At Work →Go Off Work	1.43 (0.33 to 6.12)	0.78 (0.17 to 3.52)	2.18 (0.64 to 7.40)	0.70 (0.14 to 3.4)
Off Work → RTW	1.65 (1.04 to 2.65)†	1.19 (0.72 to 1.96)	1.13 (0.65 to 1.97)	0.85 (0.44 to 1.65)
Stay Off Work	4.97 (3.29 to 7.52)†	3.55 (2.22 to 5.70)†	2.90 (1.78 to 4.75)†	1.27 (0.70 to 2.33)

* Adjusted for baseline neck pain, % body in pain, education level, headache pain, post crash dizziness, post-crash anxiety, and baseline CES-D score

** Adjusted education, pre-crash mental health, pre-crash general health, and the following measures at six-weeks: neck pain, headache pain, numbness, dizziness, vision problems, hearing problems, self-reported recovery, and CES-D score.

† Statistically significant, $p<0.001$

Cohort #2 - Depression at Baseline

Six Weeks Following Injury

Seventy-nine percent (1486 of 1860) of those who were depressed at baseline provided information on depressive symptomatology and work status six-weeks following their injury. The proportion of those who stayed at work, started at work and went off, started off-work and returned, and stayed off work was 50.0%, 3.7%, 22.5%, and 23.7% respectively. Forty-four percent (n = 651) of these individuals still scored ≥16 on the CES-D (i.e. remained depressed) at six-weeks.

Those who stayed off work from baseline to six-weeks were over twice as likely (after adjusting for aggregate confounding) to remain depressed at six weeks than those who stayed at work (adjusted OR=2.28, 95% CI 1.72, 3.0). Those who were initially at work but went off in the first six-weeks were also more likely to be depressed at six weeks than those staying at work (adjusted OR=1.93, 95% CI 1.06, 3.50). There was no significant difference in odds of depression at six weeks between those who returned to work and those who stayed at work.

Three Months Following Injury

Follow-up information on exposure and outcome at three-months was available for 67% (1245 of 1860) of those who were depressed at baseline. Of those who scored depressed at baseline, 30% were still depressed after three-months.

In the adjusted analysis, none of the work status groups differed significantly from those staying at work in their odds of being depressed at three months, although two of the groups (those initially at work then going off work, and those staying off work) had increased odds of remaining depressed in the crude analyses (before adjusting for aggregate confounding). Table 3 summarizes the outcomes for those who were depressed at the start of follow-up for both the 6-week and 3-month outcomes.

Table 3.3. Crude and adjusted odds of staying "depressed" six-weeks and three-months following whiplash injury

	Six-weeks After Injury		Three-months After Injury	
	Crude OR (95% CI)	Adjusted* (95% CI)	Crude OR (95% CI)	Adjusted** (95% CI)
Stay At Work	1.0	1.0	1.0	1.0
At Work →Go Off Work	2.40 (1.37 to 4.22)††	1.93 (1.06 to 3.50)†††	2.59 (1.43 to 4.69)††	1.18 (0.52 to 2.63)
Off Work → RTW	1.00 (0.77 to 1.31)	0.89 (0.67 to 1.19)	0.93 (0.67 to 1.27)	0.79 (0.53 to 1.18)
Stay Off Work	2.74 (2.11 to 3.56)†	2.28 (1.72 to 3.01)†	1.86 (1.40 to 2.48)†	0.90 (0.62 to 1.28)

* Adjusted for baseline CES-D score

**Adjusted for age and the following at six-weeks post injury: CES-D score, neck pain, headache pain, numbness, dizziness, vision problems, anxiety, and self-reported recovery

† Statistically Significant $p<0.001$, †† Statistically Significant $p=0.002$, ††† Statistically Significant $p<0.05$

3.4. Discussion

In a cohort of 4,271 working Canadian adults, remaining at work after injury was associated with less frequent development of depressive symptoms and more frequent recovery from depressive symptoms within the first six weeks following an injury claim, compared to staying off work. Despite this relationship early in the recovery process, work status was not associated with the prevention or resolution of depressive symptoms in the longer term (three months post injury claim).

Although the exact causal relationship was not clear, an interesting finding of this study was that for those who were initially depressed, going off work within the first six-weeks following injury was associated with an increased likelihood of depression at six-weeks (compared to those who stayed at work). However, this was not the case for those who were initially 'not depressed'. Those falling into the category of 'not depressed at baseline' were no more or less likely to have depressive symptoms at six-weeks despite going off work. This suggests that work status may be more important early in the recovery process for those who are already depressed compared to those who are not depressed as a result of their injury. Additionally, those who returned to work were no more or less likely, for either cohort, to be depressed at six-weeks after injury compared to those who stayed at work. This latter finding is what would be expected should work status be related to subsequent depression. In either case, these results support screening for depressive symptomatology early in the recovery process.

Hayden *et al* proposed a hierarchy of research phases for prognostic studies.[26] These *phases* include: Phase 1. Identifying groups of potential predictive factors (an exploratory prognostic study); Phase 2. Testing independent associations by identifying and adjusting for confounders (confirmatory prognostic study); and Phase 3. Understanding prognostic pathways.[26] Most available research has focused on exploratory studies, that is, identifying associations between a variety of factors and depression following injury (phase 1 research)[7, 13, 27, 28]; however, despite the many plausible avenues through which work absences might cause depression[1, 3, 27, 29], 'phase 2 prognostic' studies which focus on possible relationships between work status and depression following injury are limited.

This study provides evidence that a work absence may actually facilitate symptoms of depression which, in turn, could add to the likelihood of continued work absence. Although it has been previously shown that time away from work is *associated* with depressive symptoms (i.e. in cross-sectional analyses or phase-1 prognostic studies), the direction of this association remains unclear.[7, 11, 12] By grouping individuals by their baseline depression status immediately following whiplash injury, we were able to evaluate a potential causal association between work absences and subsequent depressive symptomatology. We observed that work status plays an important role in both the development and resolution of depressive symptoms shortly after an injury, but not necessarily in the longer term. It may be that, in this case, symptoms lasting longer than six-weeks are

more important than work status for subsequent depression; however, this is an area requiring further investigation.

Strengths and Limitations

Our results are based on the observation of a large cohort with a high proportion of individuals reporting complete information at each follow-up period. Another important attribute of this study was our ability to group individuals by depression status at the start of follow-up. By grouping according to baseline depressions status, and then following individuals forward in time, we were able to focus on the temporal sequencing of exposure and outcome and thus investigate a potential causal association between work status and subsequent depression; particularly for our comparison between those who stayed at work and those who stayed off work.[22]

Although our conclusions regarding depression outcomes for those staying off work versus those who stayed at work can be considered quite strong, our conclusions for those with variable return to work following injury(off-work and then RTW, or at work and then go off) are less clear. First, relatively few individuals reported variable work status following injury; effectively limiting the precision of our estimates due to the small sample sizes in these exposure categories. Secondly, depression and work status were measured at the same time. For those who reported inconsistent work following injury, we are not able to accurately measure which occurs first; a change in work status followed by

depressive symptoms or vice versa. However, as previously stated, we were able to accurately measure the association between work status, as an exposure, and subsequent depression for those who either stayed at work compared to those who stayed off work. One would need to measure depressive symptoms and work status on an almost daily basis to pin down the temporal sequence between work status and depression for those who change work status throughout the follow-up period. This is not a feasible avenue of research at this time.

The cohort we observed in this study has been reported on in previous studies and many individuals were observed up to one-year following their injury.[8, 13] In the current investigation we were unable to follow individuals past three-months. The inability to follow past three months was a result of our research question. Specifically, we were interested in how work status might influence depression status. After three-months of follow-up, many of our exposure categories become extremely small and, as such, limit the value of any results gleaned from analyses at these follow-up points (six-months to one-year following a whiplash injury). For example, for those who were not depressed at baseline, only 4% of those who initially reported being off remained off work at six-months; less than 3% reported being off at nine-months or one-year. Once these exposure categories are divided by subsequent outcome status the resulting samples are too small to assess for both potential confounding and the adjusted effect of work status on depression.

Lastly, it is possible that injury severity could account for any observed relationships between work status and depression. In other words, those with more severe injury may be more likely to be both off-work and depressed. To prevent confounding by injury severity we excluded individuals who required a hospital stay as a result of their injury. Furthermore, we adjusted for both neck pain and percent body in pain. Pain is used as a marker of injury severity for acute injury.[30] By controlling for pain (or assessing for confounding due to pain) we were able to further account for injury severity, to some extent. However it is possible that residual confounding may account for some of the associations observed.

Conclusions

Remaining at work shortly after an injury is both preventative and rehabilitative for depressive symptoms shortly after a whiplash injury. In order for an exposure to be considered a causal factor for a particular outcome, the exposure must precede the outcome.[20-22] Prior research in this area has yielded results without any clear conclusions as to the temporal relationship between work status and depression following injury.[11, 27] In this case, we have provided an analysis where temporality was fully established for two main exposures; staying at work, or staying off work. These findings have important implications for those treating individuals with an acute MSK injury and further reinforce the idea that work is an important component and contributor to overall well-being, at least in the short term.

The development of depression is a complex phenomena which is not only associated with stressful life events (such as being removed from the workplace), but likely also related life course exposures and individual characteristics such as coping.[14, 30, 31] It will be the challenge of further research to continually investigate these complexities in order to fully understand the personally taxing and generally burdensome condition of depression.

3.5. Reference

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Chapter 4 – Work and Depression in Worker's Compensation

4.1. Introduction

The decision of whether or not to remain at work during recovery from illness or injury can be a difficult one for both the worker and their health care provider, and little is known about the positive or negative outcomes that could result from a sub-optimal return-to-work (RTW) decision. For example, low decision latitude and high psychological demands at work are associated with more depressive symptoms and taking time away from this type of work environment, as a result of injury or otherwise, may prove beneficial.[1-3] At the same time, for many, work is an important source of personal identity, financial security, and can contribute to healthy socialization; and reinforcing time off-work following an injury could lead to continued *incapacitation*.[4,5] Not working is also associated with more frequent and severe mental health issues and is associated with higher rates of early mortality.[6,7]

Musculoskeletal (MSK) injuries can contribute to time away from work and MSK injuries are one of the leading causes of disability in developed countries. [8,9] Significant symptoms of depression frequently occur following injury and are associated with a delayed and complicated RTW.[10-13] The causal pathways of injury, depression, and delayed return to work are not clearly known; however, in light of the potentially positive benefits associated with work, it may be that

delays in return to work contribute to the onset and persistence of depressive symptoms.

Recent research has shown that time off work following an injury is not simply associated with depressive symptomatology, but is also associated with new and persistent episodes of depressive symptomatology in the general population. In a study of over four-thousand whiplash claimants (workers whose injuries were not work related), those who *stayed off work* were over three times more likely to develop significant depressive symptoms (if not depressed at baseline) and over two times more likely to have persistent depressive symptoms (when depressed at baseline) six-weeks after their injury when compared to those who *stayed at work* after their injury.[14]

For epidemiological studies, it has long been suggested that in order to establish a credible cause and effect relationship, there should be consistency in findings across different study samples.[15] The purposes of the current study were: 1. to investigate the evidence for continued incapacitation after injury by evaluating the temporal relationship between work status and depression following injury (asking, "Is being off work after injury associated with future depression?") in a sample of workers compensation claimants, and 2. To examine whether individuals with worker's compensation claims due to work injuries experience the same kind of association between work absence and depression found in those with traffic injury claims due to whiplash-associated disorders.[14]

The present study builds upon our prior research in the following ways. First, worker's compensation claimants differ substantially from the general population and receiving compensation has been associated with poorer pain and recovery outcomes following treatment for a musculoskeletal condition.[17-19] Participants enrolled in previous studies of this nature study have been from the general population or were entered into the studies as a result of a motor vehicle collision.[10,11,14,16] Lastly, those who previously investigated worker's compensation claimants did not evaluate, or were unable to determine, the temporal relationship between work status and depression when following injured workers.[12,13] This study provided an additional opportunity to evaluate the relationships between work, injury, and subsequent mental health for those in the worker's compensation system.

4.2. Methods

Study Population and Data Source

This study used administrative data from the Alberta Worker's Compensation Board (WCB). The data set included 7,708 WCB claimants in the province of Alberta who were referred for a WCB workplace injury assessment between September 30, 2009 and January 31, 2011, and who were subsequently enrolled in a WCB return to work program. The administrative data set included age, gender, marital status, education, injury and assessment dates, work status at time of assessment and discharge, job attachment, and pain (measured on an 11-point numerical rating scale).[20] The data set captured SF-36 scores at two time points;

first, at the completion of the injury assessment and the second at the completion of the WCB return to work program.

Outcome of Interest

This study used the SF-36 mental health composite score (MCS) to determine each claimant's depression status at the time of assessment and again at the completion of their return to work program.[21] The SF-36 has been shown to correlate well with the Centre for Epidemiological Studies Depression Scale (CES-D).[22] The CES-D is a valid and reliable questionnaire that records self-reported depressive symptoms on a continuous scale ranging from zero (indicating no depressive symptoms) to sixty (maximum depressive symptoms) and has been used in previous related research.[10,14,16,22-26] The SF-36 mental health composite score has been shown to correlate well not only with the CES-D, but is also well correlated with symptoms of depression as identified in a diagnostic interview.[22-24] When compared to a CES-D cut-off score of ≥ 19 (suggesting depressive symptomatology), a cut-off of ≤ 35 on the SF-36 MCS has a sensitivity of 80% and a specificity of 90% for identifying depressive symptomatology for individuals experiencing pain.[22]

Predictor of Interest

The goal of this study was to determine if work status following injury contributed to, or prolonged subsequent depressive symptomatology (as measured by the MCS) in a group of worker's compensation claimants recovering from

injury. As in previous work, the predictor of interest for this study was work status (stay at work versus stay off work) from baseline to follow-up.[14] Work status and depression status were measured at the same times (i.e. at baseline and again at follow-up). As a result, for those who reported changes in both work status and depression status at follow-up, it could not be determined if their depression status was responsible for their change in work status or vice versa.[14] Even if measures of depressive symptomatology had been available at the time of changing exposure status (e.g. at the time of returning to work for those initially off work), the difficulty in pinning down the temporal relationship between work status and depression status would remain. Although depression status does influence work status (those with depression are less likely to return to work), this study focuses on the influence of work status on subsequent depression status.[12, 13] Therefore, in this study we report only on individuals who either stayed at work or stayed off work throughout the follow-up. Subsequently, we were able to analyze how continued work (or continued work absences) influenced short term depression outcomes in this group of individuals.

Potential Confounders

The strength of the independent association between work status and subsequent depression was initially assessed using logistic regression. Potential confounders were selected according to prior research and their availability in the administrative data. The following measures were considered for potential confounding: age; gender; education; marital status; injury related pain (via

numerical pain rating scale [NPRS]); and baseline depression status (according to the MCS).[10, 14, 16, 20-22] Information regarding a worker's "job attachment" at baseline was also available. Job attachment refers to whether or not an injured worker remains connected to their pre-injury job or place of work during recovery. Specifically, workers may or may not have the opportunity to return to their pre-injury job after sustaining a work-related injury (i.e. some injuries limit an individual's ability to fulfill their job demands). Those who reported being unable to return to their pre-injury job, or place of employment, were classified as 'non-job attached'. Job attachment (yes/no) was also assessed a potential confounder.

Analysis

Workers were identified as *staying at work* if they reported working at both baseline (assessment) and follow-up (program discharge). Those identified as *staying off work* reported not working at both baseline and follow-up. Workers were followed prospectively, in their respective exposure categories, from the time of their WCB assessment to their program discharge. The proportions of those who developed, or recovered from, depressive symptomatology in each work status group (stay at work versus stay off work) were then compared.

Three main logistic regression models were used to statistically test the relationship between the exposure (work status) and the outcome (depressive symptomatology at follow-up. First, a univariable logistic regression model was

used to assess the crude (unadjusted) relationship between work status and subsequent depression. Second, potential confounders (age, gender, education, marriage status, job attachment, baseline depression, and pain) were added one at a time to the crude model to test for changes in the association between work status and subsequent depression after adjustment. A variable was identified as a confounder and was included in the final model if that variable changed the crude relationship between work status and subsequent depression by more than 10% when added to the work status-depression model.[27] Last, an adjusted model was used for those who were categorized as depressed at baseline and again for those categorized as not depressed at baseline; replicating our analysis for the whiplash cohort.[14]

Because of the small sample size and the rarity of reported depression in this cohort (see results), propensity scoring was also used to help control for confounding. An adjusted analysis was repeated using propensity scoring methods. Propensity scoring methods are a well-used tool which can increase statistical power when analyzing a large number of predictors with a small sample size or when working with relatively rare outcomes.[28,29] In this case, the propensity score helps to adjust for the chance of being exposed (the *propensity* to be off work) based on a number of prognostic variables (or worker characteristics); rather than controlling for those characteristics individually.[29] The propensity score helps to adjust for confounding by indication in observational studies.[30]

The propensity score was created using variables which had complete follow-up information and which could pragmatically predispose the worker to being off work (age, prior education, marital status, job attachment, baseline depression status, and injury related pain). These variables were included in a multivariable logistic regression model with work status (the exposure) as the outcome. The predicted probability of each worker remaining off work was calculated using STATA 10.1.[31] These probabilities were then categorized into quintiles and entered as a categorical variable in the final regression model assessing the association between work status and depression status. Gender has been shown to be independently associated with depression outcomes and, as such, was excluded from the propensity scoring but included in our multivariable analyses.[32] Finally, the distributions of each variable used to develop the propensity score were compared, by exposure category (work status), for adequate overlap. In other words, propensity scoring can be insufficient if the distributions of the confounding variables do not sufficiently overlap within each propensity category, across exposure categories. The overlap between exposure categories was tested both statistically and graphically. In summary, the propensity score was used to estimate, after controlling for the probability of being off work to begin with, the independent effect of work status on subsequent depression status.

4.3. Results

Of the original 7,708 WCB claimants, approximately 8% (n=639) completed the SF-36 at both their assessment and again at program discharge. After

excluding those who changed work status during the follow-up, 564 (7.3%) individuals were available for analysis. Compared to the larger WCB patient population (n=7,069), the individuals followed in this study were younger (30.0 ± 5.3 years vs. 44.0 ± 11.6 years) and more likely to be single (26% vs 16%). However, those included in the study did not statistically differ from those who were not included with respect to gender distribution, education, injury related pain, job attachment, or baseline depression status. These results are summarized in table 4.1.

Table 4.1. Baseline Characteristics[†]

		Baseline Sample (n=639)	Study Population (N=7,069)	p-value[*]
Age (years) [mean (±SD)]		30.0 (±5.3)	44.0 (±11.6)	<0.01
Gender [n(%)]	Female	208 (32.5%)	2,535 (35.9%)	0.09
	Male	431 (67.5%)	4,534 (64.1%)	
Education [n(%)]	< High School	86 (13.5%)	969 (13.7%)	0.15
	High School Grad	130 (20.3%)	1,202 (17.0%)	
	At Least some College	100 (15.7%)	1,318 (18.6%)	

	At Least Some University	53 (8.3%)	563 (8.0%)	
	Not Specified	270 (42.3%)	3,017 (42.7%)	
Marital Status [n(%)]	Single	169 (26.5%)	1,141 (16.1%)	<0.01
	Married	206 (32.2%)	2,724 (38.5%)	
	Separated	28 (4.4%)	571 (8.1%)	
	Not Specified	236 (36.9%)	2,632 (37.2%)	
Job Attached [n(%)]	Yes	102 (16.0%)	1,098 (15.5%)	0.77
	No	537 (84.0%)	5,971 (84.5%)	
Off Work at Baseline [n(%)]	Yes	337 (53.0%)	3,842 (54.5%)	0.47
	No	299 (47.0%)	3,211 (45.5%)	
Injury Related Pain [mean (±SD)] Ψ		5.0 (2.4)	5.1 (2.5)	0.24
'Depressed' at Baseline [n(%)]	Yes	85 (13.3%)	289 (13.8%)	0.73
	No	554 (86.7%)	1,799 (86.2%)	

† not all variable cells add to 7,708 due to missing data

* chi-squared or t-test where appropriate (results in appendix)

Ψ 11-point numerical pain rating scale (NPRS)

When compared to those who stayed off work, the group who stayed at work included a higher proportion of females, were more likely to be job attached, reported less pain, and were less likely to report marriage or education status (see table 4.2). Notably, there was no difference in baseline depression status for those who stayed at work compared to those who stayed off work.

Table 4.2. Baseline Characteristics: Those who stayed at-work versus those who stayed off-work

		Stay at Work (n=271)	Stay off Work (n=293)	p-value*
Age (years) [mean (±SD)]		30.3 (±5.3)	29.8 (±5.3)	0.21
Gender [n(%)]	Female	104 (38.4%)	75 (25.6%)	<0.01
	Male	167 (61.2%)	218 (74.4%)	
Education [n(%)]	< High School	25 (9.2%)	50 (17.1%)	<0.01
	High School Grad	47 (17.3%)	67 (22.9%)	
	At Least some College	36 (13.3%)	53 (18.1%)	
	At Least Some University	25 (9.2%)	21 (7.2%)	
	Not Specified	138 (50.9%)	102 (34.8%)	
	Single	59 (21.8%)	87 (29.7%)	<0.01

Marital Status [n(%)]	Married	72 (26.6%)	111 (37.9%)	
	Separated	15 (5.5%)	11 (3.8%)	
	Not Specified	125 (46.1%)	84 (28.7%)	
Job Attached [n(%)]	Yes	268 (98.9%)	200 (68.3%)	<0.01
	No	3 (1.1%)	93 (31.7%)	
Injury Related Pain [mean (±SD)] [Ψ]		4.7 (2.3)	5.2 (2.5)	0.02
'Depressed' at Baseline [n(%)]	Yes	30 (11.1%)	38 (13.0%)	0.48
	No	241 (88.9%)	255 (87.0%)	

* chi-squared or t-test where appropriate (results in appendix)

[Ψ] 11-point numerical pain rating scale (NPRS)

Individuals completed their first SF-36 and other self-report information a median of 100-days post injury; the follow-up SF-36 was completed a median 33-days later. Approximately thirteen percent of those followed (85 of 639) scored "depressed" (a MCS score ≤ 35) at their initial assessment; approximately eleven percent (69 of 639) scored "depressed" at follow-up.

In the crude analysis comparing the association between work status and subsequent depressive symptomatology, those who stayed off work were over two

times more likely to be depressed at follow-up than those who stayed at work (crude OR = 2.04, 95%CI 1.16, 3.58). After adjusting for potential confounders, work status was no longer significantly associated with depressive symptomatology at follow-up (adjusted OR = 1.55, 95%CI 0.78, 3.09). Similar results were obtained after adjusting with propensity score (propensity adjusted OR = 1.38, 95%CI 0.73, 2.63). Table 4.3 summarizes these results.

Table 4.3. Crude and adjusted odds of being "depressed" at follow-up.

	Stay at Work	Stay Off Work
Crude OR (95% CI)	1.0	2.04 (1.16 to 3.58)[¥]
Adjusted (95% CI)	1.0	1.55 (0.78 to 3.09)
Propensity Adjusted (95% CI)	1.0	1.38 (0.73 to 2.63)

* Adjusted for age, gender, education, marital status, baseline depression status, job attachment and pain.

**Adjusted for gender, job attachment, and propensity score. The propensity score included 5-categories and adjusted for age, education, marital status, baseline depression status, and pain.

[¥] Significant at p=0.01

Lastly, crude, adjusted, and propensity adjusted models were used to assess the risk of continued depression (for those who scored *depressed* at baseline) or

incident depression (for those who scored *not depressed* at baseline). Our crude analysis showed that those without baseline depression were 2.3 times more likely to develop depression if they stayed off work during the follow-up period (crude OR = 2.29, 95% CI 1.07, 4.92). This result was not repeated for those who scored depressed at baseline (crude OR = 1.8, 95% CI 0.67, 4.85). Work status did not appear to play a role in the development or resolution of depressive symptomatology in our adjusted models (see table 4.4 and 4.5).

Table 4.4. Crude and adjusted odds of developing depression over the follow-up.

	Stay at Work	Stay Off Work
Crude OR (95% CI)	1.0	2.29 (1.07 to 4.92) ¥
Adjusted (95% CI)	1.0	1.51 (0.64 to 3.58)
Propensity Adjusted (95% CI)	1.0	1.55 (0.66 to 3.67)

* Adjusted for age, gender, education, marital status, baseline depression status, job attachment and pain.

**Adjusted for gender, job attachment, and propensity score. The propensity score included 5-categories and adjusted for age, education, marital status, baseline depression status, and pain.

¥ Significant at $p<0.05$

Table 4.5. Crude and adjusted odds of staying "depressed" at follow-up.

	Stay at Work	Stay Off Work
Crude OR (95% CI)	1.0	1.8 (0.67 to 4.85)
Adjusted (95% CI)	1.0	1.93 (0.51 to 7.39)
Propensity Adjusted (95% CI)	1.0	1.77 (0.46 to 6.80)

* Adjusted for age, gender, education, marital status, baseline depression status, job attachment and pain.

**Adjusted for gender, job attachment, and propensity score. The propensity score included 5-categories and adjusted for age, education, marital status, baseline depression status, and pain.

Propensity Scoring

The adequacy of the propensity scoring method was assessed using both descriptive and statistical analysis. First, study participants were grouped into quintiles representing their propensity to be "off-work" at baseline; according to the methods previously described. Table 4.6 illustrates the distribution of participants within the propensity quintiles.

Secondly, the distribution of the confounding variables used to develop the propensity score were compared for differences between work status categories. These distributions were analyzed graphically or via chi-squared test where appropriate. Figures 4.1 to 4.2 provide examples of the distribution comparisons for propensity category 1; for age distribution and pain ratings, respectively. Appendix D contains the full propensity scoring results for each variable and propensity category.

Table 4.6. Propensity scoring, categorical distribution

Propensity Category	Frequency (n=550)	Percent
1	109	19.82
2	111	20.18
3	109	19.82
4	111	20.18
5	110	20.00

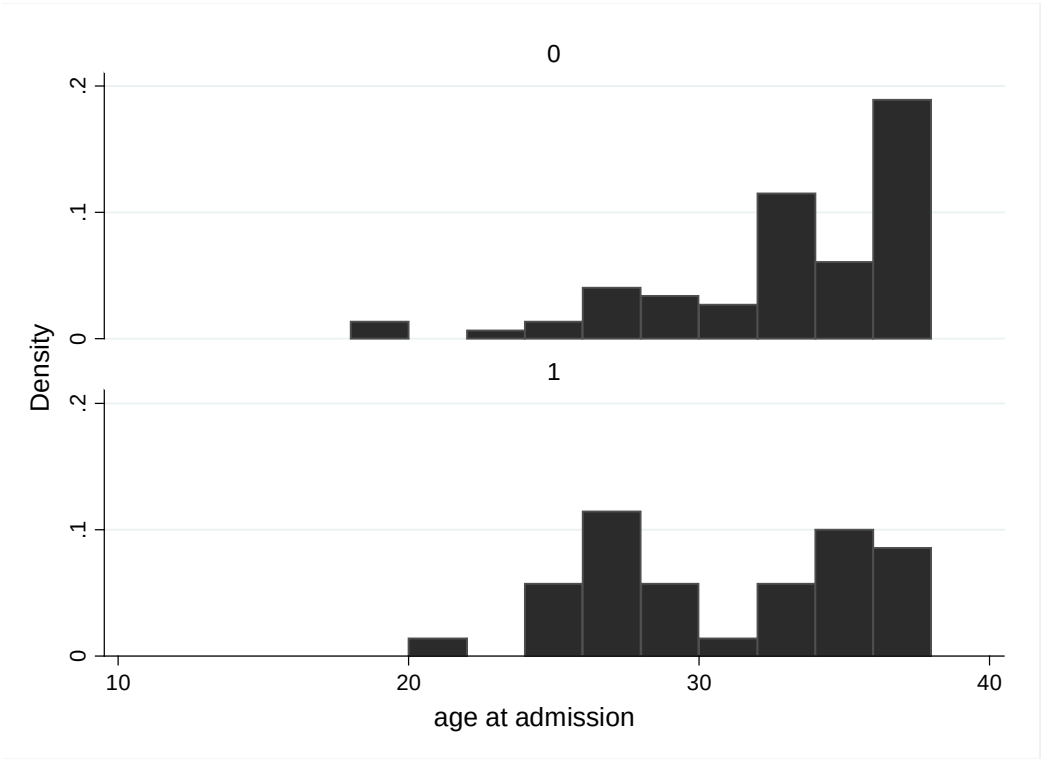

Figure 4.1 Age distribution by exposure for propensity category 1. (0=”stay at work”; 2=”off-work”)

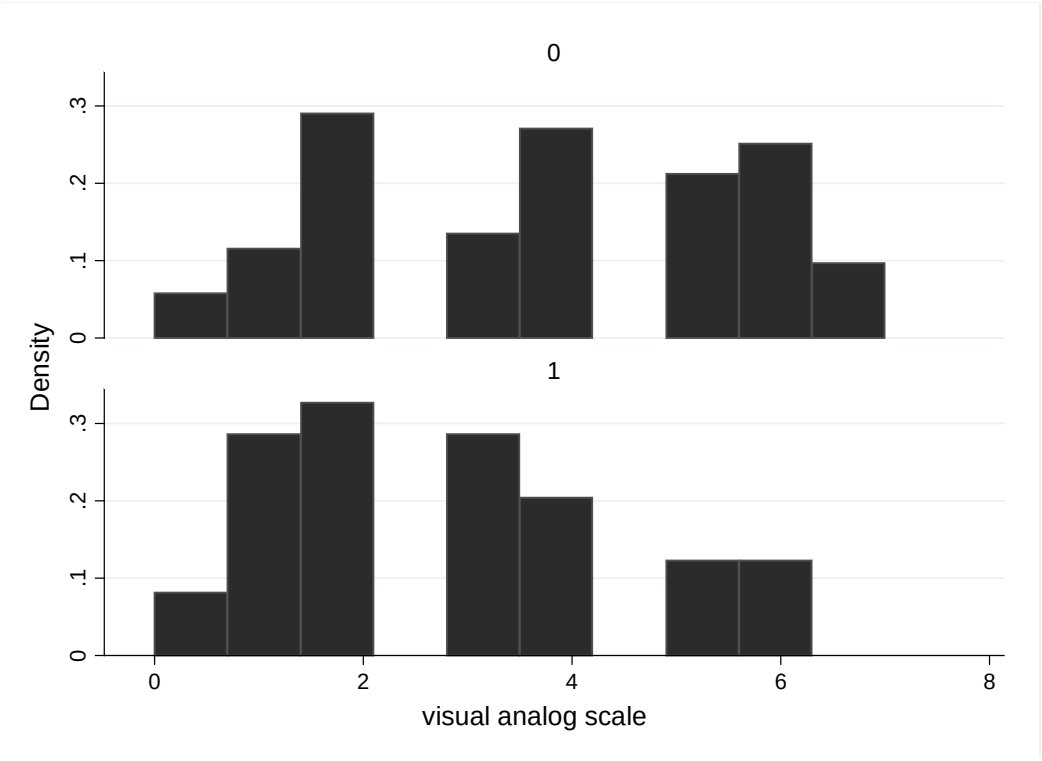

Figure 4.2 Reported pain distributions by exposure for propensity category 1. (0=”stay at work”; 1=”off-work”)

Finally, there were no significant differences between exposure groups for education, marital status, or baseline depression status within each propensity strata (see Appendix D).

4.4. Discussion

In a sample of almost 600 worker’s compensation claimants in the province of Alberta, Canada, the relationship between work status and future depressive symptoms was not statistically significant. In our adjusted models for the entire study group, the only significant predictor of depression at follow-up was

depression at baseline (OR = 10.4, 95% CI 5.3, 20.5). Although it was expected that job attachment would be a significant contributor to depressive symptoms (imagine being injured and then not having a job to return to), there was no association between job attachment and subsequent depression in any of our analyses. Furthermore, those who scored depressed at baseline comprised the only group for which baseline pain (from a visual analog scale [VAS]) contributed to depression at follow-up. Each single point increase on the baseline pain VAS was associated with 1.2 times higher likelihood of depression at follow-up in this group (OR=1.2, 95%CI 1.02, 1.41). In our adjusted models using propensity scoring methods there was no significant association between work status and depression outcomes.

It is interesting to note that those who stayed at work appeared fundamentally different than those who stayed off work at baseline. Specifically, from table 4.2 one can observe that those who stayed at work had a higher proportion of females, were less likely to report marital and education status, were more likely to be job attached, and reported slightly lower levels of pain. Despite this apparent difference, our propensity adjusted analysis accounted for this baseline discrepancy. The distributions of each confounding variable were compared between those who stayed at work and those who stayed off work, within each propensity category. The distribution of each variable included in the propensity displayed near complete overlap between the exposure categories within each propensity score quintile (see appendix D). As a result, the inherent differences

between exposure groups at baseline were eliminated in our analysis by using the propensity scoring methods described above. The described overlap ensured that the exposure groups were comparable at baseline and allowed an analysis of the influence of work status on subsequent depression.

The findings of this study showed partial agreement with previous research where it was observed that work status ceased to play a role in the development of depressive symptomatology later than six-weeks post injury. It may simply be that factors other than work status (e.g. pain) are more important with respect to depression after injury in the longer term. In our previous investigation, work status was a significant contributor to depressive symptoms early in the recovery process (within six-weeks).[14]

One possible explanation for the lack of consistency in findings could be due to the differences in timing of the measurements of the outcome and exposure. In the whiplash injury cohort, being off work for the first six-weeks following an injury was associated with depressive symptomatology six-weeks after the whiplash injury, but not at three-months.[14] The initial measures of depressive symptoms in the present study of WCB claimants occurred a median of 100-days post injury (3.4 months or 14.7 weeks). The lack of an association between work status and subsequent depressive symptomatology in this investigation might be due to the delayed initial assessment. The present study could have missed the critical time frame for which work status has an important influence on

subsequent depression; which, according to our previous study, is within six-weeks of the injury. Previous research supports this idea.

In a population based study from 2006, for those recovering from whiplash injury, it was found that over 40% of individuals experienced depressive symptoms within 6-weeks of injury; however, 62% of those same individuals experienced complete resolution of their depressive symptoms at the end of a one-year follow-up.[10] Furthermore, the median time to resolution of depressive symptoms in the 2006 study occurred within 92-days; the first measure in our present study took place at a median time of 100-days post injury, at which point the average individual (from previous research) would no longer be depressed.[10]

The use of different measurement tools to measure depression could be another potential contributor to the discrepancies observed between our studies. In our study of the whiplash cohort, depressive symptomatology was measured using the CES-D; the present study utilized the SF-36 mental health scale. There are a few key differences between these scales. The CES-D assesses one-week period prevalence for depressive symptoms, has been widely used in studies of depression, and has been validated against psychiatric diagnostic criteria for depression.[23-26] The SF-36 MCS assesses 4-week period prevalence of mental health problems. Although the MCS score is correlated with the CES-D score for those with musculoskeletal pain, it was designed as a measure of general mental health well-being and not as a measure of depressive symptoms.[21,22] With this in

mind, it is likely that the MCS is less sensitive and less specific to actual clinical depression than is the CES-D as the MCS is measuring a different construct. The differences between the constructs of measurement for the CES-D and SF-36 MCS are subtle but important. Depression is a common mental disorder characterized by feelings of low self-worth, loss of interest or pleasure, low energy, and/or an overall depressed mood.[33] The CES-D (and other depression scales) attempt to capture, often through self-report, the degree of depression specific symptoms an individual may be experiencing at a certain point in time. Well-being (in this case, mental health well-being) is more difficult to define but, in general, refers to broader constructs of being healthy, happy, and comfortable.[34, 35] Well-being has been described with three primary attributes: 1. it is subjective; 2. it includes positive aspects and is not merely the absence of negative factors; and 3. includes a global assessment of all aspects of one's life.[36] Depression can significantly influence well-being but does not encompass all aspects of well-being. Major depression has one of the strongest influences on SF-36 mental health scores but depression does not fully predict SF-36 mental health scores.[37] It may also be that an individual will report depressive symptoms but their symptoms do not immediately affect their overall well-being.

The idea that staying at work in some capacity following an injury may prevent or shorten significant symptoms of depression continues to remain a plausible cause-effect relationship.[38] This plausibility stems from the contribution of work to financial security and potentially positive social interactions. The

difficulty in fully describing the possible associations between work and mental health, in this case, is most likely a result of: 1. Missing information on the quality of the workplace experience (e.g. work satisfaction, perceived workplace support), and 2. The probable bi-directionality of the relationship between work and mental health.

First, in a review of social relationships and health, a low quantity of social relationships, as well as low quality relationships, were consistently associated with higher rates of mortality.[39] Specific to mental health, social isolation and a lack of social support is consistently associated with poorer mental health status.[‡‡‡] Thus, it would seem that being engaged with the workplace would prove beneficial not only to overall well-being, but to mental health as well. Work can provide the opportunity to experience seemingly needed social interactions and relationships. One would expect to observe fewer episodes of depression, or more resolved depression in a group of individuals staying at work after injury. The problem with this line of thought is that workplaces do not always serve as a positive influence on an individual's life. While stressful life events (such as a loss of productive work) are strongly associated with the onset of major depression, working in a highly demanding position with little perceived autonomy can also lead to depression.[41, 42] It may be, in our cohort of injured workers, that these two dichotomies cancel each other thus leaving work as an

[‡‡‡] Stansfield SA in Marmot and Wilkinson 2006[40]; Chapter 8: Social Support and Social Cohesion

unimportant factor in mental health outcomes. Specifically, many workers may feel the loss of social connectivity and productivity associated with work, but many of these same individuals may also be leaving a stressful work environment. Information about work satisfaction and psychological work demands were not available in the administrative data used in this study.

Second, it is possible that an individual with significant depressive symptoms would be more likely to remain off work after an injury as a result of their depressive symptoms, or due to the interaction between their physical health (the injury) and their mental health. Of equal possibility, an individual who is unable to return to work as a result of injury is likely to develop significant symptoms of depression. Prior research has indicated that depression affects work status (or time to return to work) and work status affects depression.[12-14] Despite this concern, the use of propensity scoring methods in this study helped to control for possibility of baseline depression resulting in a work absence. Thus, it is unlikely that our results were biased by the potential impact of baseline depression on work status following injury.

This study provides support to the idea that factors other than work status (possibly persistent symptoms and/or personal or situational attributes) are more strongly related to depression outcomes in the longer-term. In light of the discrepant findings between our previous study (chapter 3) and the present study,

further research is needed to evaluate whether or not work status influences subsequent depressive symptoms early in the recovery process.

Limitations

It should be noted that this study was characterized by numerous limitations. Firstly, the main outcome (depression status) was characterized by self-reported mental health scores on the SF-36.[21] Although the SF-36 correlates well with other self-report measures of depression status (e.g. CES-D), detailed information regarding the specific cut-off scores which would correlate the MCS to clinical measures of depression were not available.[22-24] Moreover, the SF-36 MCS is not a depression specific measure. In essence, this study used a proxy measure to determine depression status at baseline and follow-up, and might account for the low proportion of those reporting depression at baseline and the lack of effect observed due to work status.

Another concern was the low follow-up rate. The sample observed here consisted of less than 8% of the source population. For studies in epidemiology, follow-ups exceeding 80% are recommended before study findings are to be considered truly valid (pertaining mostly to generalizability).[37,38] The extent to which this standard poses a realistic goal for all research could be argued; however, the discrepancy is still quite large (8% compared to the recommended 80%).

Administrative data can be an extremely useful tool in the development and execution of epidemiological studies; however, administrative data is rarely captured with the goal of research in mind and poor data quality is the most frequently cited limitation of administrative data.[43-46] Primary data collection allows for extreme flexibility in inquiry, design, data collection, and analysis methods; provided funding is available. In contrast, administrative data provides a relatively inexpensive means of information on a wide variety of individuals; however, the information that is available can restrict what research questions can be formed. The present study attempted to answer an *a priori* research question using a dataset that was known to contain relevant variables. Despite containing the variables of interest, there were significant portions of missing information within the data obtained. The missing data led to a small sample which may not have been representative and provided only a few for the analysis of the outcome of interest (depression at follow-up). As a result, the precision of the estimates were substantially reduced.

Missing information can decrease a study's generalizability and lead to bias within the study. Bias occurs when the study methodology, or a measurement tool used, results in systematic error(s) that influences the observed association between exposure and outcome; i.e. a non-random error. Although many forms of bias have been discussed, a common grouping includes 1. selection bias and 2. measurement bias.[47, 48] The concern here is with selection bias; a bias that occurs

when there is a systematic error in the way subjects are selected into the study.[47, 48]

Our results suggest a potential for selection bias. In particular, previous research has suggested that younger age and being separated/divorced or widowed are associated with new episodes of depression in the general population,[49] but older age is associated with persistent symptoms of depression for those suffering whiplash injury.[16] Our study sample tended to be younger and were more likely to be single than the study population (table 4.1). One might expect a higher proportion of individuals reporting baseline depression as a result of the younger sample, but our sample showed a lower proportion than previous research. Despite the concern, this finding is unlikely to bias our results as the mean age of participants were equal between exposure groups (see table 4.2).

To minimize the limitations posed by the available data, this study provided two analysis strategies. First, a logistic regression model including identified confounders was used to determine the association between work status and subsequent depression. Secondly, a propensity scoring method (described previously) was used to create a categorical variable which ensured comparable exposure groups based on baseline characteristics. Our analysis of the propensity scores demonstrated that the propensity scoring method was successful in forming comparable exposure groups. As a result, we can be more confident in the results presented here despite the data limitations

In summary, a major limitation of this study was that fewer than 10% of the individuals available for inclusion had enough follow-up information to make a prospective analysis possible. Although many individuals completed the SF-36 at baseline and follow-up, these were rarely on the same individuals (i.e. only the 7.3% available for follow-up). This may be an indication to the organization collecting the data to review their data collection standards as it appears a great deal of time and energy has been put into the collection of data. In this case, despite the use of valid outcome measures, data collection was too inconsistent to allow for broader generalization to the WCB claimant population.

Conclusions

In the study of causal associations, it is important to consider not only the strength of potential cause-effect associations, but the consistency as well.[15] The goal of the current study was to assess the presence of a work-depression association in a worker's compensation sample. Previously, work status had been found to be associated with less recovery and more frequent development of depressive symptomatology shortly after a whiplash injury.[14] Those findings were not replicated in the worker's compensation sample after adjusting for potential confounders (including a propensity adjusted model [i.e., propensity to be off work]). Limitations in this study include missing data, resulting in a potentially non-representative sample and a relatively small number of events (depression at follow-up). These factors likely contributed to the low precision

observed in our results. Thus, it remains unclear whether or not being off work as a result of a work injury is an independent risk factor for depression in those seeking worker's compensation. Future larger investigations in worker's compensation samples, which correct for the limitations encountered here, are needed to clarify this important relationship.

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Chapter 5 – The Methodological Dilemma of Controlling for Baseline Depression Scores In a Follow-up Study of Work-Status and Depressive Symptomatology

5.1. Introduction

The purpose of this paper was to discuss the methodological dilemma of a specific statistical adjustment in longitudinal studies, and to present an example of how this can affect study findings and conclusions. When an outcome is a health status variable that was present and measureable at baseline, it is common practice to control for the baseline values of that outcome when conducting longitudinal studies. The common strategy of controlling for baseline scores in a multivariate regression analysis (where the baseline variable is related to the outcome) has received some recent attention, discussion, and debate.[1-3] We wanted to investigate how the issue of controlling for prior health status might influence the results in our investigation of the possible association between work status and subsequent depression.

For studies investigating the effect of work status on depression outcomes, controlling for baseline health status (i.e. depression) can: 1. Allow the investigator to control for, or determine the effect of, baseline depression on future depression (e.g. controlling for depression status at the start of follow-up); and 2. In the case of dichotomizing a continuous measure into yes/no categories (such as the Center for Epidemiological Studies Depression Scale [CES-D];

depressed/not-depressed), control for how close one is to the cut-off score at each preceding measurement.

In chapter 3, we investigated if work status influenced depression at the end of a six-week follow-up and again at the end of three-months. Individuals were stratified by depression status at baseline in order to address #1 above (control for baseline depression status); thus allowing the study to determine if work status influenced the risk of incident depression or risk of persistent depression (as measured by the CES-D).[4] We also included crude CES-D scores from each preceding time period to address #2 above (control for how close an individual measures to the cut-off score). Specifically, crude baseline depression scores were included in the regression model for depression status at the six-week follow-up, and the six-week crude depression scores were included in the model for depression status at the three-month follow-up. These baseline depression scores were included in the multivariable regression analysis to control for the total sum of depressive symptoms at the start of follow-up. For example, it seemed unlikely that a change in CES-D score from 13 to ≥16 was equivalent to a change in score from 2 to ≥16. Without consideration of the initial score, these two situations would reflect the same change in health status. We wanted to determine if a change in depression status was in fact a true shift from *depressed* to *not depressed* and vice versa. Specifically, did individuals report a change in depression status because they were already close to the CES-D cut-off score of 16 or did they experience a true shift in depression status (i.e. larger changes in

depression scores over time)? We anticipated that the inclusion of prior depression scores would prevent mixing the effect of having a baseline score that was close to the cut-off score versus experiencing a true transition in depression status. In effect, the outcomes reported in chapter three (while including baseline and prior CES-D scores) could be more confidently attributed to the true associations between work status and subsequent depression status.

To review the implications of regression models which include and exclude baseline measures of an outcome variable, the present paper compares estimates describing the associations between work status and subsequent depression (as assessed by the CES-D) when the analysis is adjusted and not adjusted for baseline CES-D scores. Specifically, the purpose of this study was to compare and discuss the analytical results given by Jones' whiplash study (where depression status, as determined by CES-D, score was the outcome: chapter 3) under two different analytical strategies: 1. Controlling for crude baseline CES-D scores, and 2. Not controlling for baseline CES-D scores.

5.2. Methods

This study utilized data from our prior chapter three study which evaluated the influence of work status on subsequent depression.[4] This study utilized a series of regression modelling strategies to determine the influence of work absences on subsequent depression status following injury.

5.2.1. Summary of Chapter 3 Study Subjects and Procedures

In the chapter three investigation, we followed a cohort of 4,271 individuals who reported paid employment prior to a whiplash injury. This main cohort was further divided into two sub-cohorts: 1. Depressed at baseline; 2. Not depressed at baseline.[4]

Briefly, the study population consisted of a cohort of individuals making personal injury insurance claims for traffic-related whiplash injuries in the Canadian province of Saskatchewan.[4] Two sub-cohorts were followed; one of which was depressed at the time of their initial injury claim (the first measurement point following the injury) and the second of which was not depressed at the time of their claim. The exposure of interest was being off work (due to the injury) or remaining at work, and the outcomes were subsequent resolution of depression (for the group depressed at baseline) and development of depression (for the group not depressed at baseline). Selection of the initial whiplash cohort is described previously in this dissertation and in other research.[4-6] As a reminder, Figure 5.1 outlines the grouping process:

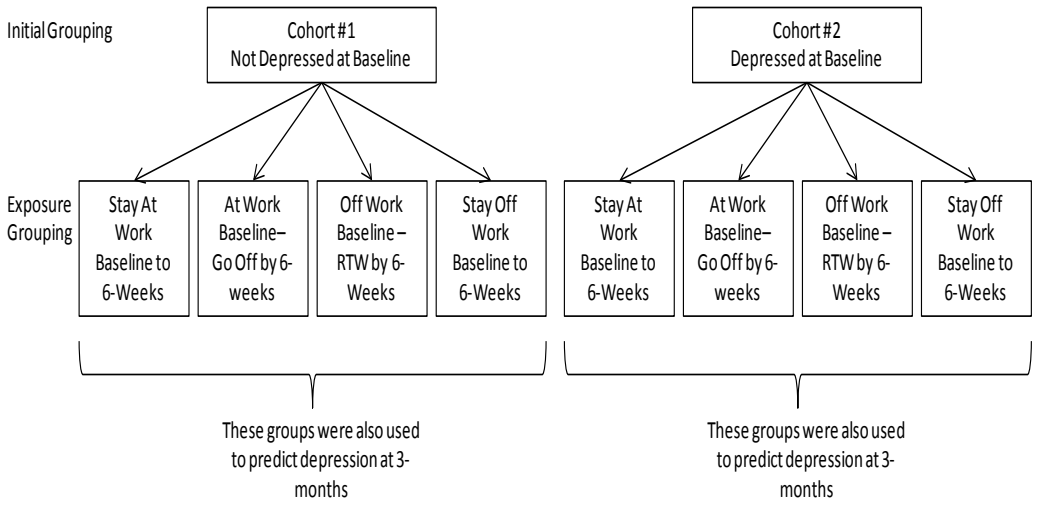

Figure 5.1. Cohort Selection and Exposure Grouping[4]

The information used for this study was self-reported and participants reported on their pre-injury health, demographic and socioeconomic characteristics, pain (11-point numerical pain rating scale [NPRS]), work status, and depressive symptomatology.[4] Participants completed follow-up questionnaires using structured telephone interviews which were implemented by trained interviewers.

5.2.2. Summary of Analysis Strategies: With and Without Baseline Depression Scores

In the chapter three study, logistic regression was used to assess whether or not a particular variable qualified as confounders in the relationship between work status (staying at work or going off work after injury) and subsequent depressive symptomatology.[4] Mutivariable logistic regression was then used to assess the independent effect of work status on depressive symptomatology while controlling for aggregate confounding (i.e. all qualifying confounders included in

the model). In each case (six-weeks or three months after injury) prior depressive symptoms scores, as reported on the CES-D, statistically qualified as a confounder. The remaining previously identified confounders were unchanged for this investigation.

The present chapter utilized the same logistic regression models, with one exception; the present chapter analyzed the influence of *excluding* baseline CES-D scores in each regression model. In other words, each regression model in chapter three adjusted for the effect of prior CES-D scores when looking at subsequent depression outcomes (at six-weeks, and again at three-months). The present investigation excluded baseline CES-D scores from those same (chapter three) regression models to determine any potential influence on the results.

5.3. Results

5.3.1. Summary of Results of Chapter 3 Study

The initial whiplash cohort consisted of 4,271 individuals who reported part-time or full-time employment just prior to their injury. Fifty-six percent (56%, n=2411) of individuals scored "not depressed" at baseline (CES-D < 16) with a mean baseline CES-D score of 7.1 (sd=4.6). The remaining 44% (n=1860) scored "depressed" at baseline (CES-D ≥16). The "depressed" at baseline group had a mean baseline CES-D score of 26.9 (sd=8.8). Eighty-three percent (1997 of 2411) of those who were employed and *not depressed* at baseline provided follow-up information on depression and work-status at six-weeks. Additionally,

seventy-five percent (1803 of 2411) of participants who were *not depressed* at baseline completed the CES-D at three-months.

In the group that scored *depressed* at baseline, seventy-nine percent (1486 of 1860) provided information on depressive symptomatology and work status six-weeks following their injury, and 67% (1245 of 1860) provided this same information at three-months. Of those who scored in the depressed range of scores at baseline, 30% remain depressed three-months following their injury.

Those who stayed at work or returned to work during the three month follow-up reported an average reduction in CES-D scores of 53% and 60% from baseline to three months post injury, respectively. Those staying off work reported a 43% relative reduction in the sum of depressive symptoms and those going off work during the follow-up period reported a 40% reduction in depressive symptoms as measured by the CES-D.

5.3.3. Predicting Subsequent Depression - Comparing to Results from Chapter 3

Tables 5.1 and 5.2 show the comparisons between adjusting and not adjusting for prior CES-D score in a multivariable logistic model for subsequent depression. Specifically, in chapter three, baseline CES-D scores were adjusted for in the final models describing the relationship between work status and depression at six-weeks. Additionally, six-week CES-D scores were included in the models

describing the relationship between work status to six-weeks and depression at three-months.[6]

Table 5.1 demonstrates that there is a discrepancy between including and not including six-week CES-D scores as a confounder for depression outcomes at three-months. This is the only scenario in which the results significantly differ. It can be seen that when prior CES-D scores are not included in the aggregate confounding model, staying off work for at least six-weeks becomes a significant predictor of depression at three months for those who initially scored "not depressed" at baseline (odds ratio for depression (OR) = 1.27, 95%CI 0.70, 2.33 [adjusting for prior CES-D scores] versus OR = 1.96, 95%CI 1.12, 3.45 [when prior CES-D scores are not included in the model]). In table 5.2 it can be observed that there was no change in the relationships between work status and depression at six-weeks or three-months when prior CES-D scores were excluded from the analytical models; for those who were "depressed" at baseline.

Table 5.1. Crude and adjusted odds of *developing* depressive symptomatology six-weeks and three-months following whiplash injury; with and without prior CES-D score.

	six-weeks After Injury		three-months After Injury	
	[i]Original OR* (95% CI)	Excluding CES-D Score (95% CI)	[i]Original OR** (95% CI)	Excluding CES-D Score (95% CI)
Stay At Work	1.0	1.0	1.0	1.0
At Work →Go Off Work	0.78 (0.17 to 3.52)	0.72 (0.16 to 3.27)	0.70 (0.14 to 3.4)	0.60 (0.13 to 2.81)
Off Work → RTW	1.19 (0.72 to 1.96)	1.17 (0.71 to 1.93)	0.85 (0.44 to 1.65)	1.18 (0.64 to 2.17)
Stay Off Work	3.55 (2.22 to 5.70)†	3.31 (2.09 to 5.26)†	1.27 (0.70 to 2.33)***	1.96 (1.12 to 3.45)***

i From Chapter 3

* Adjusted for baseline neck pain, % body in pain, education level, headache pain, post-crash dizziness, post-crash anxiety, and baseline CES-D score

** Adjusted education, pre-crash mental health, pre-crash general health, and the following measures at six-weeks: neck pain, headache pain, numbness, dizziness, vision problems, hearing problems, self-reported recovery, and CES-D score.

† Statistically significant, $p<0.001$

*** Discrepancy in findings, depending on analysis strategy

Table 5.2. Crude and adjusted odds of *staying* "depressed" six-weeks and three-months following whiplash injury; with and without controlling for prior CES-D score.

	Six-weeks After Injury		Three-months After Injury	
	[i]Original OR* (95% CI)	Excluding CES-D Score (95% CI)	[i]Original OR** (95% CI)	Excluding CES-D Score (95% CI)
Stay At Work	1.0	1.0	1.0	1.0
At Work →Go Off Work	1.93 (1.06 to 3.50)[†††]	2.40 (1.37 to 4.22)[††]	1.18 (0.52 to 2.63)	1.45 (0.72 to 2.95)
Off Work → RTW	0.89 (0.67 to 1.19)	1.00 (0.77 to 1.31)	0.79 (0.53 to 1.18)	0.93 (0.65 to 1.34)
Stay Off Work	2.28 (1.72 to 3.01)[†]	2.74 (2.11 to 3.56)[†]	0.90 (0.62 to 1.28)	1.09 (0.79 to 1.52)

i From chapter 3

* Adjusted for baseline CES-D score

**Adjusted for age and the following at six-weeks post injury: CES-D score, neck pain, headache pain, numbness, dizziness, vision problems, anxiety, and self-reported recovery

[†] Statistically Significant $p<0.001$, [††]Statistically Significant $p=0.002$, [†††]Statistically Significant $p<0.05$

5.4. Discussion

In the present investigation, an alteration in analysis strategies provided conflicting results regarding the influence of work status on depressive outcomes at three months for those who originally scored *not depressed* at baseline. All exposure groups demonstrated a trend toward decreasing CES-D scores throughout the follow-up, except for those who were initially "not depressed" and stayed off-work through the entire follow-up period. When six-week CES-D scores were *included* as co-variates in the regression analysis comparing the influence of work status on depressive symptomatology at three-months, prior work status was *not* a significant predictor of later depression (depression at three-months; OR 1.27, 95%CI: 0.70 to 2.33).[4] Conversely, if prior CES-D scores were *excluded* from the analysis, prior work status (to six-weeks) became a significant predictor of depression status at three-months (OR 1.96, 95%CI: 1.12 to 3.45). In fact, removing prior CES-D scores from our regression models strengthened the independent influence being off-work on three-month depression outcomes for all categories (tables 5.1 and 5.2). However, the only category in which the significance of this relationship was altered was for the three-month follow-up of those who were categorized as *not depressed* at baseline.

Recall, excluding prior CES-D scores in our logistic regression models changed the apparent influence of prior work status on depression outcomes at three-months for those who were originally categorized as *not depressed* at baseline. In considering this discrepancy, two primary questions arose: 1. Should

prior CES-D scores be considered a potential confounder (regardless of statistical findings)?, and 2. Is the above discrepancy in findings a result of an over-fitted model (i.e. does adjustment for baseline CES-D scores actually lead to over-adjustment resulting in bias)?

5.4.1. CES-D Scores as a Confounder?

Confounding occurs when there is a distortion of the relationship between an exposure and an outcome due to a mixing of effects between the exposure and a third (incidental) factor.[7-9] A confounder can also be described statistically (but must still meet conceptual criteria for a confounder). Confounding should not be confused with mediating. As described in chapter two, a mediator is a variable through which an exposure influences an outcome.[9, 10] Vittinghoff *et al* (2005) states that when attempting to identify a primary predictor of interest (as is the case here), potential confounders should be selected on face validity as well as statistical grounds; our confounders were selected on these criteria.[9] Vittinghoff *et al* (2005) further states that variables which mediate the effect of the exposure and outcome should be excluded as the inclusion of a mediator will artificially attenuate some of the association between the primary predictor (exposure) and the outcome under study.[9] It is easy to argue that baseline depression scores are related to work status and future depression and should therefore be included in our models (Figure 5.1 A). It is makes less sense to consider baseline depression as a mediator (Figure 5.1 B)

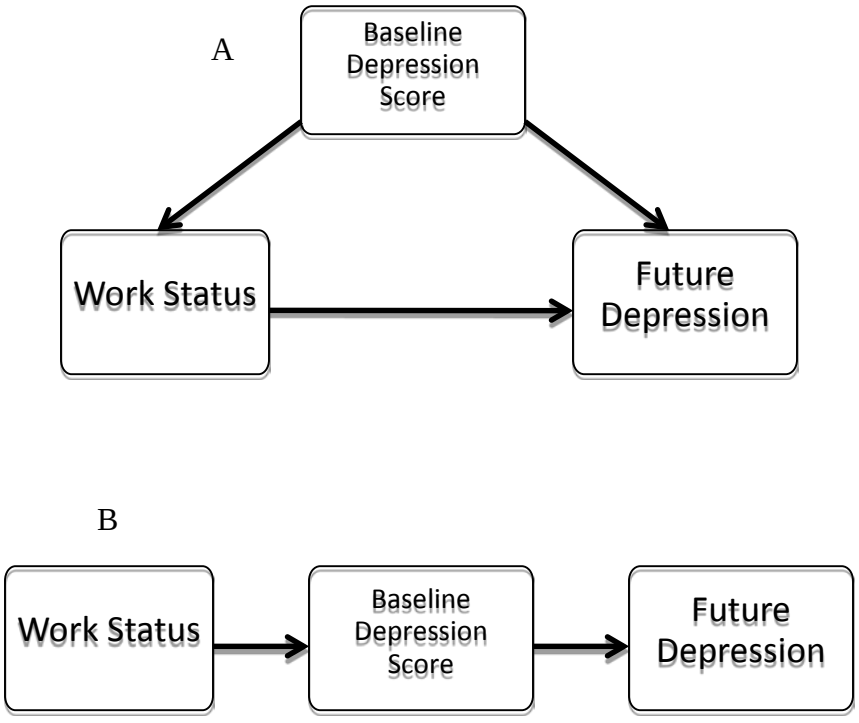

Figure 5.2 – Baseline CES-D as a Confounder (A) & as a Mediator (B)

The reason this latter view is less likely is because baseline depression scores are measured at the beginning of follow-up while work status is comprised of an individual's baseline and follow-up work status (i.e. stay on or off work); it would be impossible for ongoing work status to influence baseline depression scores resulting in future depression. This is the implied association if baseline depression score are considered a mediating variable and excluded from our models (Figure 5.1 B). Alternatively, baseline depression could influence work status and previous research supports the association between baseline depression and future depression.[11, 12] This latest idea again illustrates a confounding relationship. We can see, yet again, that baseline depression scores are most

appropriately viewed as a potential confounder and should be adjusted for as such (Figure 5.1 A).

In chapter three it was demonstrated that prior depression scores consistently qualified as a confounder, on a statistical basis, in our investigation of the influence of work status on subsequent depression in a cohort of individuals recovering from whiplash injury. In other words, including prior depression scores in the model changed the crude estimate of the association between work status and subsequent depression status by more than 10%, the criteria set for identifying a variable as a confounder. In review, a purposeful selection strategy was used to assess whether or not potential confounders independently altered the relationship (expressed as an odds ratio) between work status and subsequent depression by more than 10%. From a statistical standpoint, prior CES-D scores were considered an important confounder for all our final models.

From a non-statistical standpoint, why include prior CES-D scores when modelling the development or persistence of depressive symptomatology with respect to work status during recovery? Firstly, as mentioned in the introduction, it seemed reasonable to control for how close one was to the transition point between being categorized as depressed or not depressed ("depressed" if CES-D $\geq$ 16). We included prior CES-D scores to help distinguish between those individuals who underwent a true transition in depression status from those who experienced a change in depression status as a result of small random variations in

depressive symptoms over time. Secondly, it seemed likely that higher depression scores could be responsible for initial work status and may be related to subsequent depression. Specifically, we assumed that baseline CES-D would be associated with work status. In fact our assumption proved correct as those who were off work at baseline scored significantly higher on the CES-D than those who were at work (mean difference 6.26; SEM 0.37, p-value < 0.001). In conjunction with this finding, prior research demonstrated that a large proportion of those with high depressive symptoms at one-month following injury continue to have high depressive symptoms at six-months and those with high depressive symptoms take longer or are less likely to return to work.[11, 12] As a result, prior CES-D scores could be considered as associated with both our outcome and exposure; a major criteria when evaluating whether or not to include a variable as a potential confounder. With the above rationale in place, we could be confident that prior depression scores were indeed a confounding variable that needed to be adjusted for in our final models. But there was still more to consider.

5.4.2. Over-Adjustment and Potential Bias while including Prior CES-D Scores?

Bias occurs when there is a systematic error(s) in a study's design, or during sampling, data collection or analysis. In the present study, our concern was primarily with bias in the analysis phase. Specifically, we were concerned with whether or not the inclusion of prior CES-D scores in our regression models resulted in different conclusions for our proposed exposure-outcome relationship; did one model introduce bias (and thus differing results) not present in the other?

For example, in our present study, it is plausible that the inclusion of prior depression scores could have contributed to an over-fitted, or over-adjusted, and thus biased model. Over-adjustment (or over-fitting) a model can occur when one includes an adjustment variable that violates the criteria for a confounder.[8] The inclusion of such a variable has the potential to introduce damaging collinearity; damaging in the sense that the introduced collinearity can invalidate a model and/or give misleading results by degrading the precision of regression estimates; and therein lies the bias.[8, 9, 13] Collinearity is a "complex" mathematical issue and denotes excessive correlation between two or more regression variables.[9, 13] One view is that when two or more variables are highly collinear, they effectively share a portion of model variability when the outcome could have successfully been modelled with a single variable. As a result, the regression estimate for at least one of the collinear variables is no longer interpretable.[9] The inclusion of two or more collinear covariates can have the effect of creating large differences in regression estimates and yield a model that is unstable and lacks precision.[13]

Vittinghoff *et al* (2005) describes the concerns present in this study; specifically, what happens when the predictor of interest is collinear with an adjustment or confounding variable? 1. If the predictor of interest (e.g. our exposure variable, work status) remains significant after adjusting with the collinear confounder, the evidence in support of an independent effect of the exposure on the outcome is convincing. 2. There is also a "clear cut" result if the exposure variable is obviously confounded by the adjustment variable; the

adjustment variable must be included. 3. Lastly, if there is no statistical association between the primary predictor or the adjustment variable and the outcome, one must acknowledge that the data are too inadequate to disentangle the results.[9] In addition to these considerations, excessive collinearity can be statistically assessed using diagnostic criteria such as tolerance, variance inflation factors (VIF), or simple correlation matrices.[9, 13]

What is occurring in this study? Given the above argument, we could conclude that baseline CES-D score should in fact be included in our final model. Explicitly, when prior CES-D scores are included in the model describing depression outcomes at three-months, work status does not significantly predict depression at three-months (table 5.1); however, prior CES-D scores are a significant predictor of depression at three-months in the fully adjusted model. Precisely, each single point increase in 6-week CES-D scores for those who scored "not depressed" at baseline was associated with an 11% increased odds of developing depressive symptomatology by three-months (OR 1.11; 95%CI: 1.08 to 1.15). Furthermore, using the suggested regression diagnostics of variance inflation factor (VIF) and a Pearson product-moment correlation table in *post hoc* analyses, we did not find any evidence of substantial collinearity between our primary predictor (work status) and the adjustment variable of prior depression scores (see appendix).

The above discussion supports our original inclusion of prior depression scores in our final models aimed at analyzing the influence of prior work status on subsequent depression status. That being said, this study helps bring to light the possible limitations of categorizing a continuous outcome such as the CES-D. Although categorizing an outcome, such as the CES-D, can often make the results more interpretable, it is possible that work status played a more important role in overall depressive symptomatology and some of the influence might have been missed as a result of the CES-D score categorization. For example, it is possible that many individuals realized a true change in depressive symptomatology as a result of their work status without actually changing depression status (e.g. initial CES-D score = 45, follow-up score = 17). By using a categorical outcome (i.e. CES-D cut-off score for "depression"), it is possible that the true nature of the relationship between work status and depressive symptoms was not fully described. Although the categorization of our outcome improves on the interpretability of our results and provides a link to clinical outcomes, future research may want to more strongly consider the depressive symptomatology as a continuous variable to avoid the information loss inherent in the categorization of continuous data. It should be noted here that as yet, no minimally clinically important difference or minimal detectable change has been validated for the CES-D. If a minimal detectable change score is identified for the CES-D, use of such a score may help to resolve this problem. Further research in this area should continue to evaluate the ever important issues of outcome description and

predictor/adjustment variable selections to ensure valid result continue to be disseminated from our efforts.

This investigation illustrated the importance of fully considering how an outcome measure is obtained and used in follow-up studies of health status. One has to be wary of including variables which may inadvertently provide an over-adjusted estimate and mask or bias the relationship between the exposure and outcome of interest.[1-3, 9, 10, 13] On the other hand, if one or more variables is to be included despite the risk of over-adjusting or collinearity, the inclusion of these variables must be made with sound rationale and not violate any conceptual or statistical underpinnings. The present study provides an illustration of how results can differ in the presence of differing analysis strategies and, as a result, highlights the importance of completely considering how potential confounders are analyzed and the reasons behind their inclusion.

In conclusion, the inclusion of baseline depression scores (on the CES-D) in a follow-up study of the association between work status and subsequent depression following injury weakens the observed association prior work status has on depression status at three-months This previous result may have been a sign of over-adjustment in our regression models; however, we did not find substantial evidence to support this suspicion. In accordance with our evidence, baseline depression scores do confound the relationship between work status and subsequent depression. Once the confounding effect of an individual's previous

depressive symptomatology is controlled for, work status can no longer be considered a significant contributor to depression in the longer term (i.e. ≥ 3-months post injury).

5.5. References

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Chapter 6 – General Discussion and Conclusions

In chapter one, the following hypotheses were made to provide a direction for this dissertation:

1. For those who are off work at least 6-weeks as a result of a MSK injury, return to work will be associated with a subsequent reduction in depressive symptoms, whereas those individuals who continue to remain off work during their recovery will not experience such a reduction in depressive symptoms.

2. The incidence of depressive symptomatology will be lower in those remaining at work during recovery from a MSK injury compared to those who take a work absence for their injury.

3. Individuals with depressive symptoms yet remaining at work during recovery from a MSK injury will exhibit a shorter course of depressive symptomatology compared to those with depressive symptoms and not at work.

4. For individuals with a MSK injury, those remaining at work while recovering will have less severe depressive symptoms compared to those who take a work absence while recovering.

The goal of this dissertation was to investigate the relationship between work status and depressive symptoms following injury. The purpose of this direction

was to help provide evidence regarding the most appropriate use of time off work following and injury; giving consideration not only to an individual's physical health, but to their mental health as well.

Chapters one and two introduced the theoretical perspective for this dissertation, addressing hypothesis 1. Chapter 2 used a contemporary theorization strategy to help develop a model visualizing the relationship between work status and subsequent depression status following an injury.[1, 2]

The remainder of the dissertation addressed hypotheses 2 to 4 and evaluated the potential association between work status after injury and subsequent depression status. Chapters 3 and 4 used similar methodology to test the proposed relationship in two different patient populations while chapter 5 investigated and compared the findings of two variations in adjusting confounders in the type of research presented here.[1]

6.1. Summary of Research

Chapter Two

In chapter 2, I introduced a theoretical model to illustrate how work status could influence symptoms of depression following an injury. Using the methods of Jaccard and Jacoby, the final model suggests (with the support of previous research) that there is a probable association between work status and depression status.[2] In this model, an individual's injury moderates the work-depression

association through a mediated pathway involving role identity and individual expectations. The full model is presented below:

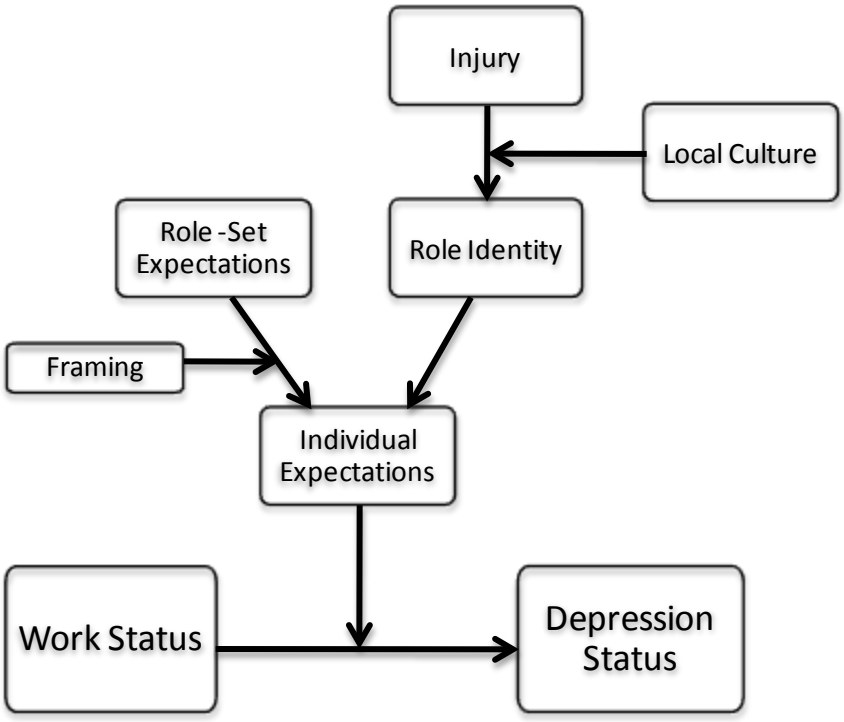

Figure 6.1. Modelling the work-status depression-status relationship following injury[1]

Chapter Three

Chapter 3 provided an initial investigation into whether or not an association between work status and depression actually exists. The results of this study provided an important contribution of knowledge regarding the relationship between work-status and depression-status following injury and suggests that work status plays an important role in the prevention and resolution of depressive symptomatology early in the recovery process. Specifically, it was observed that those who did not have significant symptoms of depression and stayed off work

during the first six-weeks following a whiplash injury were 3.55 (95%CI: 2.22 to 5.70) to develop significant symptoms of depression at the end of six-weeks. Additionally, those who were classified as "depressed" at their initial assessment were 2.28 (95%CI: 1.72 to 3.01) times more likely to remain "depressed" at the six-week follow-up if they stayed off work during that time; and were 1.93 (95%CI: 1.06 to 3.50) times more likely to remain depressed if they went off work during the first six-weeks. These associations were no longer apparent at the three-month follow-up, suggesting that work status did not play a significant role in the development or resolution of depressive symptoms after a minimum six-week recovery time.[1]

Chapter three provided evidence that for those in the general population, work status has an important influence on depression outcomes within six-weeks of injury and suggested work can cause subsequent depression early in the recovery process. The observations from chapter three are consistent with the idea of a connection between work and an individual's sense of self, their social networks, and suggests a positive association between employment and physical and mental health early in the recovery process.[3-7]

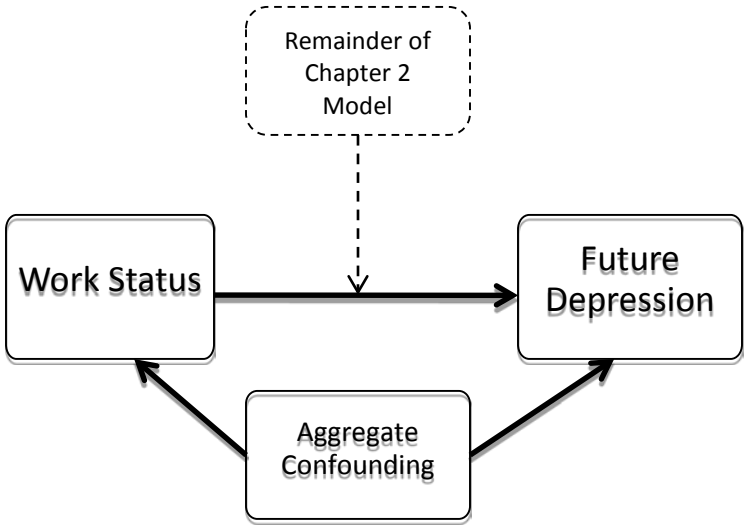

Figure 6.2. Work status and depression in the general population within six-weeks of injury

Interestingly, the only significant statistical confounder for those who were depressed at baseline was their baseline CES-D score. Conversely, for those who were categorized as not depressed at baseline, education level, neck pain, % body pain, headache pain, dizziness, vision problems, anxiety, and baseline CES-D scores qualified as confounders in the relationship between work status and subsequent depression.[1] It could be that once an individual is depressed, the influence of the depression itself far outweighs the effect of any other potential influences, besides work-status, on the development of depression early in the recovery process. Stated another way, the less depressive symptomatology an individual has, the more important other injury related factors appear to be in relation to the development of future depression; work status in the first six-weeks was one of these factors.

Chapter Four

Chapter 4 provided an attempt to evaluate the consistency of the chapter 3 findings and their applicability to an appropriate sub-population (worker's compensation claimants). In this study, 7,708 worker's compensation claimants were followed forward in time following a work-related musculoskeletal injury. This study used administrative data from the Alberta Worker's Compensation Board. Unfortunately, in this case, only 8% of the initial cohort had enough data recorded to address the pertinent research question. As result of the low follow-up, propensity scoring methods were used to adjust for aggregate confounding, as opposed to individually assessed confounders.

The chapter 4 investigation did not find the same associations observed in chapter 3. Although there was a crude association, work status did not appear to be an important predictor of subsequent depression in this group of workers compensation claimants after controlling for aggregate confounding. Other than the data limitations mentioned previously, what other attributes could account for this difference?

It has been well established that worker's compensation claimants differ from the general population.[8-10] Worker`s compensation claimants are more frequently male, less educated, work longer hours, have more strenuous work, take longer to recovery, and are more likely to take legal action pertaining to their injury.[8, 10] Furthermore, being a worker's compensation claimant was associated with feeling

unable to return to pre-injury work without surgery.[8] Arguably, this last finding could relate to a form of expectation. It would appear that worker's compensation claimants often expect surgery to treat their musculoskeletal injury prior to returning to work. Although this expectation was not measured in our research, consideration of the research by Atlas *et al* and others in the context of our research provides additional support for the model presented in chapter 2.[8-11] In the presence of aggregate confounding (a portion of which could possibly be attributed to individual expectations), work status ceases to be an important determinant of subsequent depression following injury.

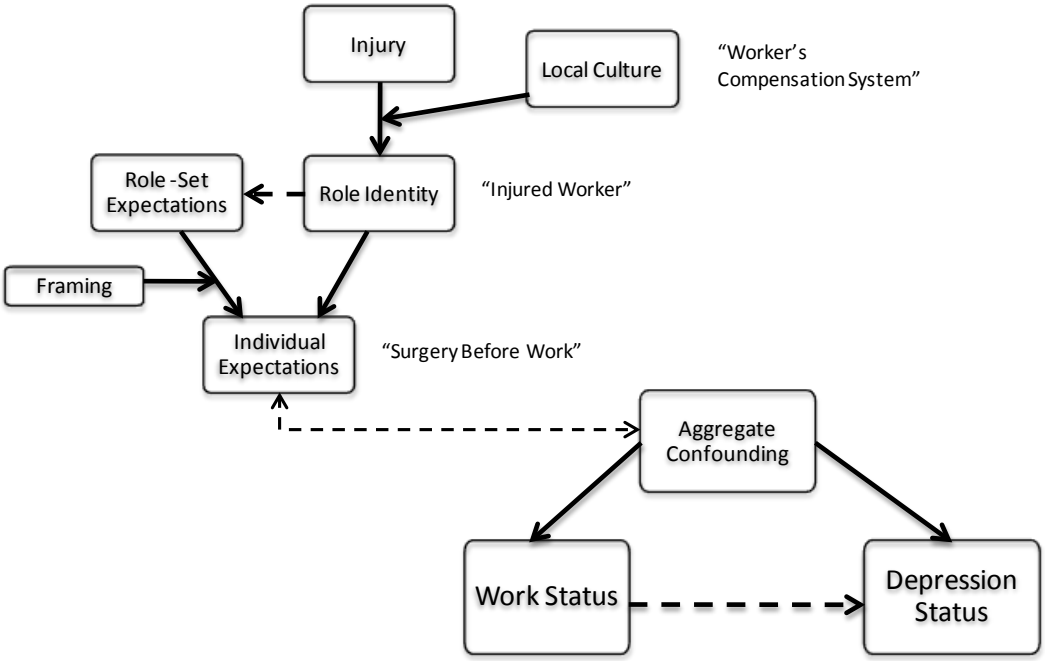

Figure 6.3. Work status and depression in worker's compensation claimants

In a recent review of psychosocial factors affecting return to work for those injured in the workplace, it was found that many employees feel frustration,

perceive discrimination, and lack understanding of the compensation system. With this in mind, we can envision the local culture of the compensation system having an influence on a worker`s role identity shortly after injury, potentially facilitating the transition to the injured worker role (Figure 6.3). Once full participation in this role is achieved, it is likely that new expectations for the injured individual are developed by not only themselves, but by those in their role set as well (coworkers, supervisors, WCB claims manager). These new expectations can be observed through assignment to modified duties or to a complete off work status until the injury is resolved. As a simplified example, the injured worker may view surgery as the only option for a return to their pre-injury status; this becomes their individual expectation and the inability to realize this expectation could contribute to depressive symptoms. It should be noted that in 2009, Atlas *et* al demonstrated that even when worker`s compensation claimants receive surgery for their injury, compensation claimant`s report poorer recovery than non-compensation individuals.[9]

Our research demonstrates that, although it does not independently predict depression for worker`s compensation claimants, one should consider work status as a potential confounder when evaluating the contribution of other factor to depression in those recovering from injury.

Chapter Five

The final paper, chapter five, provided a brief methodological investigation to determine potential differences in results when adjusting for baseline depression scores in addition to stratifying by depression status, versus not adjusting for baseline depression scores. In this study, the initial results (taken from chapter 3) remained relatively unchanged when CES-D scores were excluded from a model controlling for aggregate confounding in the work-depression relationship; however, one discrepancy was observed. For those who scored "not depressed" at the baseline assessment, the initial findings (including CES-D score as a confounder) suggested that staying off work for six-weeks did not influence depression at three-months. When CES-D scores were excluded from this model, those who stayed off work for six-weeks were almost two time more likely to be depressed at three-months compared to those who stayed at work for the first six-weeks (OR = 1.96, 95%CI: 1.12 to 3.45).

From our review, these findings confirm baseline depression as a confounding variable from both a theoretical and statistical perspective.[2, 12-14] The confounding effect of baseline depression is the prime reason for the discrepancy in the findings for the two analysis strategies. The findings of chapter five also suggest that further research is necessary to determine the full impact of work status on depressive symptoms alone; not just in relation to a clinical diagnosis. Our results show that future research needs to be wary of the mechanisms and rationale for the variables included in regression models. Simple drawings of

potential causal associations, while considering the temporality of these associations, can also facilitate a better understanding and improved analysis of the relationships between exposures and outcomes.[2, 14]

6.2. Significance of Research

The most significant finding of this dissertation was the observed association between staying off work and the persistence and development of depressive symptomatology early in the recovery process for members of the general population (see chapter 3). Previous researchers have also shown an association between work status and depression, but these same researchers have indicated that the temporality of the relationship was not clear.[15, 16] In chapter 3, individuals were stratified according to baseline depression status at the beginning of follow-up. As such, a temporal association between work status and subsequent depression status was established for those who either stayed at work or stayed off work. Despite this finding, the vagueness of the temporal relationship between work status and subsequent depression status remained for those who had a variable return work following injury (i.e. for those who changed work status during the follow-up period). The findings of chapter three were not replicated in chapter four. However, the data utilized in chapter four suffered from low rates of follow-up (~8%) compared to the data from chapter three (75%-85%), which may have led to selection bias in the former. As a result, the strongest finding remained to be that staying off work is associated with the

development of depressive symptoms within six-weeks of injury (as a result of chapter 3 study).

The research presented here is applicable to not only the lay-person suffering injury, but is applicable to the health care providers who provide health services to these individuals, the organizations that employ these individuals, and the compensation systems that provide financial reimbursement following injury (in addition to providing a variety of disability management services).

The lay-person might be interested to know that working in the early stages following an injury could help prevent or alleviate symptoms of depression after injury.[1] They might also be interested in the finding that there appears to be more important factors in the prevention of depressive symptoms than working when the injury is work related.[1] As we now know that depression is common after injury, the compensation system could use the research found in this dissertation as rationale to facilitate change in the way in which injuries are managed. Specifically, we found that remaining at work in after injury is beneficial for those in the general population; however, this relationship was not observed for those receiving worker`s compensation.

As it is in the best interest of worker`s compensation boards and employers to have workers remain in the workplace while preventing the development of co-morbid conditions (such as depression) during recovery, these organizations may

want to try and emulate the experiences of those in the general public who are recovering from injury. In other words, how can worker`s compensation boards and employers turn workplaces into an important, positive factor in the prevention and resolution of depressive symptoms?

Mentioned in chapter 1, another goal of this research was to develop clinically applicable knowledge to aid in the clinical management of musculoskeletal injuries. Health care clinicians are often relied upon to make decisions regarding an individual's work status following an injury. First, full consideration of the complexity of the circumstances surrounding work, injury, and depression cannot be ignored in the medical management of those recovering from injury. Our research can help clinicians as they make their recommendations regarding work after injury. Clinicians could ask themselves (and their patients): Is this injury work related? Does this individual have a positive work environment? What are the individual's expectations regarding their recovery and their return to work? Our research (again) suggests that members of the general public who are injured might be best treated with a recommendation to remain at work during their recovery. According to our research, work recommendations remain less clear for those with a work related injury and it is possible that a clinician's attention be focused on other individual attributes besides work status for those with a work related injury.

6.3. Future Research Directions

The seemingly simple act of being injured seems to grow to an ever expanding atmosphere of complexity. Recovery from injury relates to an individual's pre-injury physical and mental health, their support network; their age, gender, educational status, marital status; the place of injury (work-related?), and even an individual's expectations surrounding these events. The development of depression after injury could be a result of neurophysiological influences (pain pathways), cognitive predisposition (how injury is perceived individually), and/or social and interpersonal interaction (including work status).

The research presented here found that for those in the general population, remaining at work provided a positive influence on depressive symptomatology in the first six-weeks of recovery. We did not find this same association for those recovering from a work-related musculoskeletal injury. It seems unlikely that there would be significant depression related neurophysiological or social differences between those from the general public and those in the workers compensations system. More likely, the discrepancy regarding the importance of the workplace in the prevention/resolution of depressive symptomatology could highlight some important cognitive differences between those seeking worker`s compensation and those who are not in the compensation system (e.g. preconceived notions and expectations about their injury). In support of this idea, Hadler (1995) demonstrated that those receiving worker's compensation were characterized by a delayed recovery of their pre-injury sense of wellness; pointing

to an apparent cognitive difference between those in the compensation system and those who are not.[10]

Lastly, we found that when considering both the statistical and theoretical definitions of a confounding variable, baseline depressive symptomatology should be considered and most likely included in statistical models evaluating an exposures influence on depression outcomes for those who are employed yet injured.[12-14] Where do we go from here?

There seems to be no end to the possible avenues for future research in this area. The present research demonstrates that work status is important in the development of future depression but gives little information as to why and how work status is important in some cases and not others (general population versus worker's compensations claimants).

In chapter 2, a model was developed to visualize the relationship between work status and depression while giving consideration to other potential influences. Many additional questions for research can be drawn from this model and most seemed suited to a qualitative approach. For example: To what extent do other's expectations influence a worker's expectations and is this even important when recovering from injury? This question could most appropriately be addressed via a validated, structured interview process. Other pressing questions, more qualitative in nature, include: What are the worker's normative

expectations with respect to work status after injury? Do diagnoses validate sickness, and to whom? For example, does the worker seek a medical diagnosis to appease compensation managers and employers, despite the worker's internal conclusions regarding their work/injury status? What happens when the worker's internal conclusions do not match those of employers and compensation professionals? Is this a significant risk factor for depression or depressive symptomatology? Can a pre-work or a continuous educational program regarding injury, rehabilitation from injury, and the compensation system alleviate some of the post injury stress and anxiety experienced by a worker? How significant is work to an individual's identity? Does the impact of disabling injury change with the perceived significance of work?

In my opinion, systematically addressing the questions in the previous paragraph would provide significant advances in the area of disability management for injured workers (for both work-related and non-work-related injuries). At present, workers are often confused and frustrated, feel discriminated against, and are often depressed after injury.[11] However, very little is known about how worker's view their employment or the identity they attach to their work role. Although the influence of the work environment on mental health has been addressed in some research, little research is available regarding how the work environment influences the recovery process. Pinning down these questions would facilitate the development of more appropriate rehabilitation programs, both clinically and in the work environment.

It must also be recognized that work can be the source of the problem. Specifically, work can be stressful and a prompt return to such an environment could be detrimental to an individual's recovery. This author understands that it is an overly idealistic view to expect individualized programs for all injured workers, but it is hoped that the information found within this dissertation could help improve upon the current state of rehabilitation. In an era where the paradigm is 'return to work as early as possible', our research shows that this may true in some cases, but not in others. In those cases where work status does not positively influence symptoms of depression, rehabilitation professionals and compensation systems alike should strive to identify those factors which are the true barriers to a full physical and mental recovery. As a former clinician, I have observed the effort to identify the "real" barriers to recovery; however, this identification process is far outweighed by the pressure to return individuals to the workplace as soon as possible, regardless of most anything else.

6.4. Conclusions

Depression, and the development of depressive symptomatology, is a severely complex issue. Symptoms of depression may be the result of biological/genetic predisposition, symptoms may be the result of cumulative stress resulting from trauma or life experience, and symptoms may be the result of a negative life event (injury) or role alteration (change in work status). Trying to construct a framework that encompasses the plethora of variables contributing to the prevention or promotion of depression and its symptoms will be a future

challenge for all researchers in this area. It was the goal of this research to provide at least one component in the understanding of the complex machine that is depression and recovery. This dissertation provided an introductory, context specific model with which to visualize the relationship between work-status and depression-status following injury. Additionally, the research presented here successfully illustrated an important and significant relationship between staying off work after injury and subsequent depression following musculoskeletal injury in the general population (following a motor vehicle collision). Specifically, staying off-work appears to contribute to incident episodes of depressive symptomatology in those who initially score "not depressed" at the beginning of the recovery process; and staying off-work also contributes to persistent depressive symptomatology for those scoring "depressed" at the beginning of recovery (chapter 3).[1]

Lastly, this dissertation illustrated the importance of evaluating variables which are included in regression models as potential confounders. In chapter 5, it was demonstrated that the inclusion or exclusion of a single variable can substantially alter study results. Furthermore, discussion was provided regarding the rationales for including and excluding certain variables from analytical models. In the end, it became apparent that there is no single right answer, what is important is that irrespective of the strategy used, the researcher must be able to provide sound rationale to accompany the presentation of their results.

In consideration of the global impact on the health of individuals, and on the economies of nations across the globe, injury and mental health research is a necessary endeavour as we continually try to improve the quality of life for individuals and the health of populations. This is that research.

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Appendix A – Chapter 3 Crude and Adjusted Analyses: Depression after whiplash injury

NOT DEPRESSED AT BASELINE – Six-Weeks following injury

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude

```
Logistic regression                               Number of obs   =       1997
                                                  LR chi2(3)      =      21.43
                                                  Prob > chi2     =     0.0001
Log likelihood = -500.04189                       Pseudo R2       =     0.0475

------------------------------------------------------------------------------
 depress_fu1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.426835   1.061404     0.48   0.633      .332029     6.13157
_Ibase_i1_~3 |   1.657569   .3960356     2.12   0.034     1.037762    2.647558
_Ibase_i1_~4 |   4.974511   1.050092     7.60   0.000      3.28902     7.52375
```

Adjusted

```
Logistic regression                               Number of obs   =       1964
                                                  LR chi2(17)     =     141.00
                                                  Prob > chi2     =     0.0000
Log likelihood = -446.87928                       Pseudo R2       =     0.1363

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |    .782963   .5999129    -0.32   0.749     .1743987    3.515111
_Ibase_i1_~3 |   1.188084   .3034667     0.67   0.500     .7201596    1.960043
_Ibase_i1_~4 |   3.556683   .8559434     5.27   0.000     2.219203    5.700244
    neck_nrs |    1.06553    .060577     1.12   0.264     .9531772    1.191126
    drawperc |   1.007653   .0064582     1.19   0.234     .9950742    1.020391
_Ieducate_~2 |   .5753466   .1544626    -2.06   0.039     .3399449    .9737569
_Ieducate_~3 |   .5055727   .1247042    -2.77   0.006     .3117647    .8198612
_Ieducate_~4 |   .4999518   .1680441    -2.06   0.039      .258715    .9661277
 _Iheadcat_1 |   1.315297   .7193424     0.50   0.616     .4502955    3.841936
 _Iheadcat_2 |   1.085404   .3953116     0.23   0.822     .5315912    2.216179
 _Iheadcat_3 |   1.766044   .5135501     1.96   0.050     .9988027    3.122651
 _Iheadcat_4 |   1.572463   .4729657     1.50   0.132     .8720793    2.835339
 _Iheadcat_5 |   1.481534   .5514937     1.06   0.291     .7142594    3.073033
   _Idizzy_1 |   .9880602   .2050125    -0.06   0.954     .6579119    1.483881
  _Ivision_1 |   2.615171   .7625557     3.30   0.001     1.476722    4.631283
   _Iworry_1 |   1.784245   .3402518     3.04   0.002     1.227812    2.592846
    ces_base |   1.125215    .023333     5.69   0.000       1.0804    1.171889
------------------------------------------------------------------------------
```

NOT DEPRESSED AT BASELINE – Three-months following injury

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude

```
Logistic regression                               Number of obs   =       1803
                                                  LR chi2(3)      =      16.30
                                                  Prob > chi2     =     0.0010
Log likelihood = -422.29565                       Pseudo R2       =     0.0189

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.178082   1.359313     1.25   0.212     .6409886    7.401133
_Ibase_i1_~3 |   1.128723   .3213063     0.43   0.671     .6460724    1.971939
_Ibase_i1_~4 |    2.90411   .7294803     4.24   0.000     1.775009    4.751442
------------------------------------------------------------------------------
```

Adjusted

```
Logistic regression                               Number of obs   =       1722
                                                  LR chi2(22)     =     171.00
                                                  Prob > chi2     =     0.0000
Log likelihood = -323.49543                       Pseudo R2       =     0.2090

------------------------------------------------------------------------------
 depress_fu2 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   .6999986    .565702    -0.44   0.659     .1436154    3.411877
_Ibase_i1_~3 |   .8482113   .2876822    -0.49   0.627     .4363225    1.648923
_Ibase_i1_~4 |   1.274898   .3932409     0.79   0.431     .6965032    2.333606
    i1nk_nrs |   1.091684   .0560382     1.71   0.087     .9871953    1.207232
 _Ii1hdcat_1 |   2.005065   1.177348     1.18   0.236     .6343288    6.337861
 _Ii1hdcat_2 |   1.173182   .4429185     0.42   0.672     .5597658    2.458809
 _Ii1hdcat_3 |   1.585408   .5004055     1.46   0.144     .8540327    2.943117
 _Ii1hdcat_4 |   1.531481   .5269826     1.24   0.215     .7802131    3.006146
 _Ii1hdcat_5 |   .1401159   .1175072    -2.34   0.019      .027079     .725006
_Ieducate_~2 |   .6553656   .2140556    -1.29   0.196     .3455127    1.243092
_Ieducate_~3 |   .5378776   .1620235    -2.06   0.040     .2980425    .9707084
_Ieducate_~4 |   .7663495   .2819596    -0.72   0.470     .3726044     1.57618
_Ii1_dizzy_1 |   1.883925   .4672711     2.55   0.011     1.158616    3.063287
_Ii1vision_1 |   .7865508   .2995132    -0.63   0.528     .3729023    1.659046
_Ii1hearng_1 |   1.435231   .5508553     0.94   0.346     .6764294    3.045237
_Imenta_3c_1 |   2.074527   .7597668     1.99   0.046     1.011995    4.252652
_Iprhealth~2 |    1.08216   .2950383     0.29   0.772     .6341926    1.846552
_Iprhealth~3 |   2.122892   .6085124     2.63   0.009     1.210422    3.723223
_Iprhealth~4 |   1.167565   .6980219     0.26   0.796     .3617391    3.768486
_Ii1recov_~1 |   .8987848   .2395594    -0.40   0.689     .5330636    1.515418
_Ii1recov_~2 |    1.39419   .5634571     0.82   0.411     .6314104     3.07845
     cesd_i1 |   1.116878   .0167676     7.36   0.000     1.084493     1.15023
------------------------------------------------------------------------------
```

DEPRESSED AT BASELINE – Six-Weeks following injury

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude

```
Log likelihood =  -973.3419                     Pseudo R2       =     0.0345

------------------------------------------------------------------------------
 depress_fu1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.404989   .6907722     3.06   0.002     1.369704    4.222787
_Ibase_i1_~3 |   1.003295   .1370071     0.02   0.981     .7676987    1.311192
_Ibase_i1_~4 |   2.738887   .3678213     7.50   0.000     2.105044    3.563584
------------------------------------------------------------------------------
```

Adjusted

```
Logistic regression                             Number of obs   =       1468
                                                LR chi2(4)      =     232.03
                                                Prob > chi2     =     0.0000
Log likelihood = -892.11907                     Pseudo R2       =     0.1151

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.925103     .58855     2.14   0.032     1.057356    3.504989
_Ibase_i1_~3 |   .8918224   .1296834    -0.79   0.431     .6706591    1.185919
_Ibase_i1_~4 |    2.27987   .3245307     5.79   0.000     1.724824    3.013529
    ces_base |   1.089503    .007859    11.88   0.000     1.074208    1.105016
------------------------------------------------------------------------------
```

DEPRESSED AT BASELINE – Three-months following injury

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude

```
Logistic regression                               Number of obs   =       1301
                                                  LR chi2(3)      =      28.80
                                                  Prob > chi2     =     0.0000
Log likelihood = -779.23102                       Pseudo R2       =     0.0181

------------------------------------------------------------------------------
 depress_fu2 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.593216   .7839963     3.15   0.002     1.433833    4.690066
_Ibase_i1_~3 |   .9267003   .1505075    -0.47   0.639     .6740525    1.274045
_Ibase_i1_~4 |   1.863988   .2728631     4.25   0.000     1.399067    2.483406
------------------------------------------------------------------------------
```

Adjusted

```
Logistic regression                               Number of obs   =       1247
                                                  LR chi2(20)     =     407.15
                                                  Prob > chi2     =     0.0000
Log likelihood = -556.38299                       Pseudo R2       =     0.2679

------------------------------------------------------------------------------
 depress_fu2 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.176643   .4817333     0.40   0.691      .527414    2.625053
_Ibase_i1_~3 |   .7884824   .1611099    -1.16   0.245     .5282831     1.17684
_Ibase_i1_~4 |   .8952834   .1642152    -0.60   0.546     .6249294    1.282597
_Ii1recov_~1 |   1.309884   .2479611     1.43   0.154     .9038581    1.898302
_Ii1recov_~2 |   1.885837   .5280991     2.27   0.023     1.089277    3.264899
     cesd_i1 |   1.109199   .0097472    11.79   0.000     1.090259    1.128469
 _Ii1worry_1 |   1.259899   .2452162     1.19   0.235     .8603297    1.845043
_Ii1vision_1 |   1.578904   .3155796     2.29   0.022     1.067146    2.336079
_Ii1_dizzy_1 |   .8597633   .1488659    -0.87   0.383      .612344    1.207153
_Ii1armnmb_1 |   1.473204   .2339109     2.44   0.015     1.079224    2.011009
 _Ii1hdcat_1 |   1.029821   .5957764     0.05   0.959     .3313761    3.200387
 _Ii1hdcat_2 |   .9567867   .2866201    -0.15   0.883     .5318935    1.721098
 _Ii1hdcat_3 |   1.060628   .2515571     0.25   0.804     .6663119    1.688298
 _Ii1hdcat_4 |   1.169568   .2762088     0.66   0.507     .7362123    1.858009
 _Ii1hdcat_5 |   1.041407    .301261     0.14   0.888     .5907214     1.83594
    i1nk_nrs |   1.032391   .0383066     0.86   0.390     .9599759    1.110268
  _Iage_gp_2 |   .8753915   .2313786    -0.50   0.615     .5214558    1.469559
  _Iage_gp_3 |   1.583394   .3604047     2.02   0.043     1.013543    2.473634
  _Iage_gp_4 |   1.312881   .3098195     1.15   0.249     .8267133     2.08495
  _Iage_gp_5 |   2.130736   .5554487     2.90   0.004     1.278307    3.551602
------------------------------------------------------------------------------
```

Appendix B – Chapter 3 Sensitivity Analysis - Depression after whiplash injury: using a CES-D cut-off score of ≥ 21

A CES-D score of ≥ 16 has good negative predictive value (96.7%) but low positive predictive value (41.9%) for current depression; as such, a cut-off score of ≥ 21 has been suggested to compensate for this deficiency (Schulberg 1985, Shean and Baldwin 2008). However, is has been demonstrated that false positives on the CES-D are associated with similar health care utilization patterns, levels of social impairment, work disability, service use, and persistence of symptoms observed among those with a clinical diagnoses of major depression (Shean and Baldwin 2008). A lower cut-off would include more false positive values. The results presented here comprise a sensitivity analysis for the whiplash cohort discussed in chapter 3. The significant findings observed with CES-D cut-off score of ≥ 16 were not altered when the cut-off was raised to 21.

NOT DEPRESSED AT BASELINE

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude: Six-Weeks

```
Logistic regression                               Number of obs   =       1997
                                                  LR chi2(3)      =      21.43
                                                  Prob > chi2     =     0.0001
Log likelihood = -275.84023                       Pseudo R2       =     0.0374

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.644796   1.700246     0.48   0.630     .2168776    12.47411
_Ibase_i1_~3 |   1.819775   .6224027     1.75   0.080      .930868    3.557518
_Ibase_i1_~4 |   4.528028   1.369923     4.99   0.000     2.502557    8.192834
------------------------------------------------------------------------------
```

Adjusted: Six-Weeks

```
Logistic regression                               Number of obs   =       1964
                                                  LR chi2(17)     =      80.32
                                                  Prob > chi2     =     0.0000
Log likelihood = -241.91284                       Pseudo R2       =     0.1424

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   .8013067   .8477571    -0.21   0.834     .1007522    6.372988
_Ibase_i1_~3 |   1.101651   .4088188     0.26   0.794     .5323122    2.279932
_Ibase_i1_~4 |   2.528485   .8573667     2.74   0.006     1.300865    4.914604
    neck_nrs |   1.113574   .0924837     1.30   0.195     .9462933    1.310426
    drawperc |   1.003519   .0093801     0.38   0.707     .9853016    1.022073
_Ieducate_~2 |   .4767335   .1683744    -2.10   0.036     .2385862    .9525898
_Ieducate_~3 |   .3237955   .1093222    -3.34   0.001     .1670636    .6275663
_Ieducate_~4 |   .2288531   .1224233    -2.76   0.006     .0802071    .6529814
 _Iheadcat_1 |   1.169637   .9639837     0.19   0.849     .2325483    5.882864
 _Iheadcat_2 |   1.374452   .6841771     0.64   0.523     .5181044    3.646214
 _Iheadcat_3 |   .9591512     .44191    -0.09   0.928     .3887879    2.366254
 _Iheadcat_4 |   1.302387   .5673597     0.61   0.544     .5545369    3.058789
 _Iheadcat_5 |   1.466539   .7478857     0.75   0.453     .5397711    3.984537
   _Idizzy_1 |   1.096225    .329384     0.31   0.760     .6083287    1.975428
  _Ivision_1 |   3.707923   1.361113     3.57   0.000     1.805801    7.613628
   _Iworry_1 |   2.009751   .5534249     2.53   0.011     1.171513    3.447764
    ces_base |   1.104376   .0330612     3.32   0.001     1.041442    1.171114
------------------------------------------------------------------------------
```

Crude: Three-Months

```
Logistic regression                               Number of obs   =       1803
                                                  LR chi2(3)      =       8.54
                                                  Prob > chi2     =     0.0360
Log likelihood = -241.82463                       Pseudo R2       =     0.0174

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.578125   1.632786     0.44   0.659     .2077098    11.99018
_Ibase_i1_~3 |   1.791047   .6379973     1.64   0.102     .8910382    3.600124
_Ibase_i1_~4 |   2.874801    1.03369     2.94   0.003     1.420829     5.81666
------------------------------------------------------------------------------
```

Adjusted: Three-Months

```
Logistic regression                               Number of obs   =       1697
                                                  LR chi2(21)     =      90.87
                                                  Prob > chi2     =     0.0000
Log likelihood = -186.99998                       Pseudo R2       =     0.1955

------------------------------------------------------------------------------
depress~2_21 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~3 |   1.522559   .6280495     1.02   0.308     .6783548    3.417365
_Ibase_i1_~4 |   1.540264   .6523189     1.02   0.308      .671586    3.532551
    i1nk_nrs |   1.115339   .0774537     1.57   0.116     .9734104    1.277961
 _Ii1hdcat_1 |   1.156152   .9971223     0.17   0.866      .213254    6.268056
 _Ii1hdcat_2 |   1.160572     .56973     0.30   0.762     .4434178    3.037601
 _Ii1hdcat_3 |   1.532889   .6336252     1.03   0.301       .68181    3.446339
 _Ii1hdcat_4 |   .4604721   .2599264    -1.37   0.169     .1523066    1.392156
 _Ii1hdcat_5 |   .0975099   .1130744    -2.01   0.045     .0100457    .9464954
_Ieducate_~2 |   .7723883   .3473797    -0.57   0.566     .3198968    1.864925
_Ieducate_~3 |   .7219859   .3008451    -0.78   0.434     .3190367    1.633867
_Ieducate_~4 |   .9245497   .4888391    -0.15   0.882     .3279992     2.60608
_Ii1_dizzy_1 |   1.710127   .6046419     1.52   0.129     .8552105    3.419668
_Ii1vision_1 |    1.62936   .7574311     1.05   0.294     .6551243     4.05238
_Ii1hearng_1 |   .8768354   .4840861    -0.24   0.812     .2971556    2.587332
_Imenta_3c_1 |   1.225336   .6624723     0.38   0.707     .4246753    3.535521
_Iprhealth~2 |   1.080622    .418465     0.20   0.841     .5058832    2.308325
_Iprhealth~3 |   2.565175   .9904472     2.44   0.015      1.20353     5.46735
_Iprhealth~4 |   .6677799   .6180317    -0.44   0.663     .1088541    4.096586
_Ii1recov_~1 |   .8318696   .3086404    -0.50   0.620     .4020151    1.721346
_Ii1recov_~2 |   2.436726   1.224322     1.77   0.076     .9101738    6.523629
     cesd_i1 |   1.104108   .0208806     5.24   0.000     1.063932    1.145801
------------------------------------------------------------------------------
```

DEPRESSED AT BASELINE

- Analyses were made with the "stay at work" exposure group as the reference category
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude: Six-Weeks

```
Logistic regression                               Number of obs   =       1468
                                                  LR chi2(3)      =      52.18
                                                  Prob > chi2     =     0.0000
Log likelihood = -861.66922                       Pseudo R2       =     0.0294

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.909483   .8341584     3.73   0.000     1.658721    5.103382
_Ibase_i1_~3 |   1.222222   .1861429     1.32   0.188     .9068036    1.647355
_Ibase_i1_~4 |    2.52701   .3524485     6.65   0.000     1.922595    3.321438
------------------------------------------------------------------------------
```

Adjusted: Six-Weeks

```
Logistic regression                               Number of obs   =       1468
                                                  LR chi2(4)      =     233.75
                                                  Prob > chi2     =     0.0000
Log likelihood = -770.88689                       Pseudo R2       =     0.1317

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.316936    .719685     2.71   0.007     1.260418    4.259059
_Ibase_i1_~3 |   1.088336   .1775692     0.52   0.604     .7904672    1.498449
_Ibase_i1_~4 |   2.017348   .3032008     4.67   0.000     1.502616    2.708405
    ces_base |   1.098723   .0081947    12.62   0.000     1.082778    1.114902
------------------------------------------------------------------------------
```

Crude: Three-Months

```
Logistic regression                               Number of obs   =       1301
                                                  LR chi2(3)      =      21.84
                                                  Prob > chi2     =     0.0001
Log likelihood = -656.13056                       Pseudo R2       =     0.0164

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.460829   .7834471     2.83   0.005     1.318521    4.592784
_Ibase_i1_~3 |    .886399   .1663612    -0.64   0.521     .6135839    1.280515
_Ibase_i1_~4 |   1.786836   .2890289     3.59   0.000     1.301364    2.453412
------------------------------------------------------------------------------
```

Adjusted: Three-Months

```
Logistic regression                               Number of obs   =       1250
                                                  LR chi2(19)     =     213.57
                                                  Prob > chi2     =     0.0000
Log likelihood = -534.99755                       Pseudo R2       =     0.1664

------------------------------------------------------------------------------
depress~2_21 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.206769   .4533439     0.50   0.617     .5779033    2.519955
_Ibase_i1_~3 |   .8802232    .184252    -0.61   0.542     .5840039    1.326691
_Ibase_i1_~4 |   1.009477   .1867232     0.05   0.959     .7025048    1.450585
_Ii1recov_~1 |   1.570123     .32225     2.20   0.028     1.050109    2.347649
_Ii1recov_~2 |   2.445853   .6846407     3.20   0.001     1.413067    4.233483
 _Ii1worry_1 |   4.663556   1.074664     6.68   0.000     2.968707    7.326002
_Ii1vision_1 |   1.664857   .3148357     2.70   0.007     1.149235     2.41182
_Ii1_dizzy_1 |   1.369692   .2362824     1.82   0.068     .9767519     1.92071
_Ii1armnmb_1 |   1.469583   .2412986     2.34   0.019     1.065199    2.027484
 _Ii1hdcat_1 |   1.181885   .7269934     0.27   0.786     .3539891    3.946033
 _Ii1hdcat_2 |   1.280539   .3756133     0.84   0.399      .720634    2.275469
 _Ii1hdcat_3 |   1.202279   .2949341     0.75   0.453     .7433529    1.944535
 _Ii1hdcat_4 |   1.174968   .2842118     0.67   0.505     .7313577    1.887654
 _Ii1hdcat_5 |   1.252463   .3567201     0.79   0.429     .7166875    2.188768
    i1nk_nrs |   1.061444   .0412925     1.53   0.125     .9835208    1.145541
  _Iage_gp_2 |   .6510403   .1827232    -1.53   0.126     .3755838    1.128519
  _Iage_gp_3 |   1.111351   .2608831     0.45   0.653     .7015143     1.76062
  _Iage_gp_4 |   1.057357   .2553419     0.23   0.817     .6586647    1.697379
  _Iage_gp_5 |   1.571524   .4086381     1.74   0.082     .9440302    2.616111
------------------------------------------------------------------------------
```

Appendix C – Chapter 4 Analysis Tables: Depression after work-related injury

- Analyses were made with the "stay at work" exposure group as the reference category and a single comparison to those who "stayed off work"
- The adjusted model included gender, job attachment, and propensity score. The propensity score included 5-categories and adjusted for age, education, marital status, baseline depression status, and pain.

Crude

```
Logistic regression                               Number of obs   =        564
                                                  LR chi2(1)      =       6.52
                                                  Prob > chi2     =     0.0106
Log likelihood = -189.98886                       Pseudo R2       =     0.0169

------------------------------------------------------------------------------
_Idepressi~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Istay_off_1 |   2.041865    .585921     2.49   0.013     1.163514    3.583295
------------------------------------------------------------------------------
```

Adjusted

```
Logistic regression                               Number of obs   =        550
                                                  LR chi2(7)      =      26.62
                                                  Prob > chi2     =     0.0004
Log likelihood = -176.22511                       Pseudo R2       =     0.0702

------------------------------------------------------------------------------
_Idepressi~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Istay_off_1 |   1.383961   .4521685     0.99   0.320     .7294893    2.625602
  _Iprops2_2 |   3.415074   2.020262     2.08   0.038     1.071157    10.88797
  _Iprops2_3 |   2.600891    1.57629     1.58   0.115     .7929481    8.530992
  _Iprops2_4 |    1.60457   1.039033     0.73   0.465     .4509898    5.708877
  _Iprops2_5 |   6.472854   3.675667     3.29   0.001     2.126834    19.69963
  _Igender_1 |   .8558672   .2744748    -0.49   0.627     .4564864    1.604667
  _Iadmjob_1 |   .7109182   .2530651    -0.96   0.338      .353849    1.428306
------------------------------------------------------------------------------
```

Appendix D – Chapter 4: Propensity Scoring

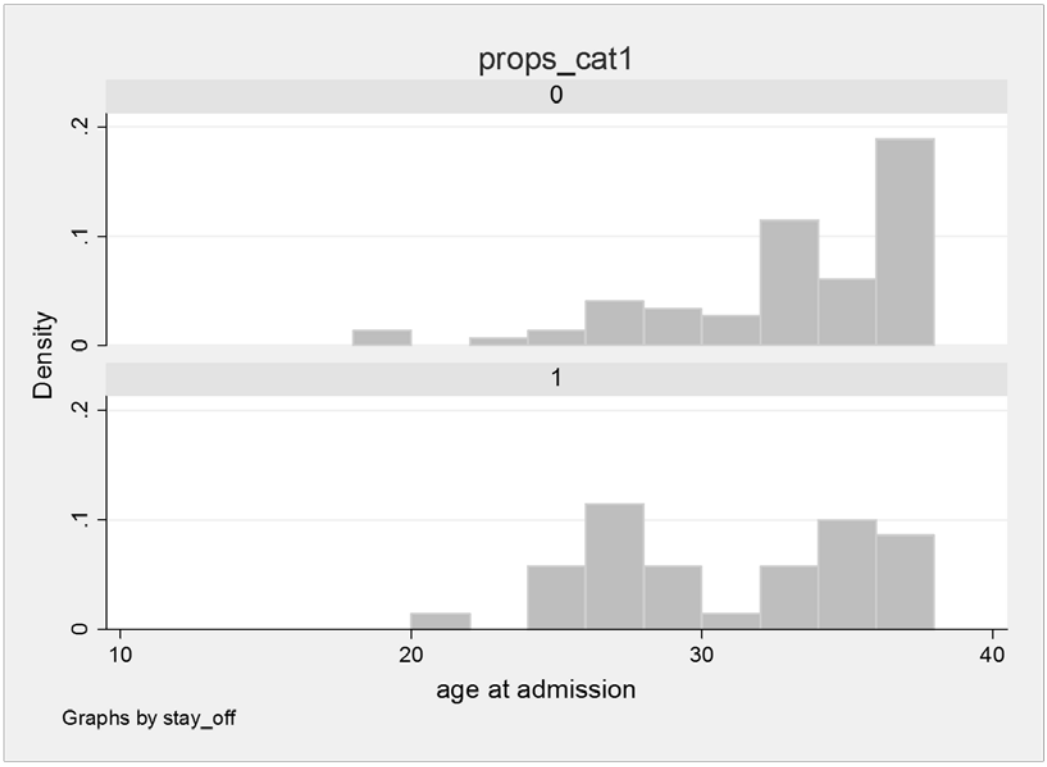
- Propensity groupings were compared to ensure equal distributions of exposure category in each propensity category

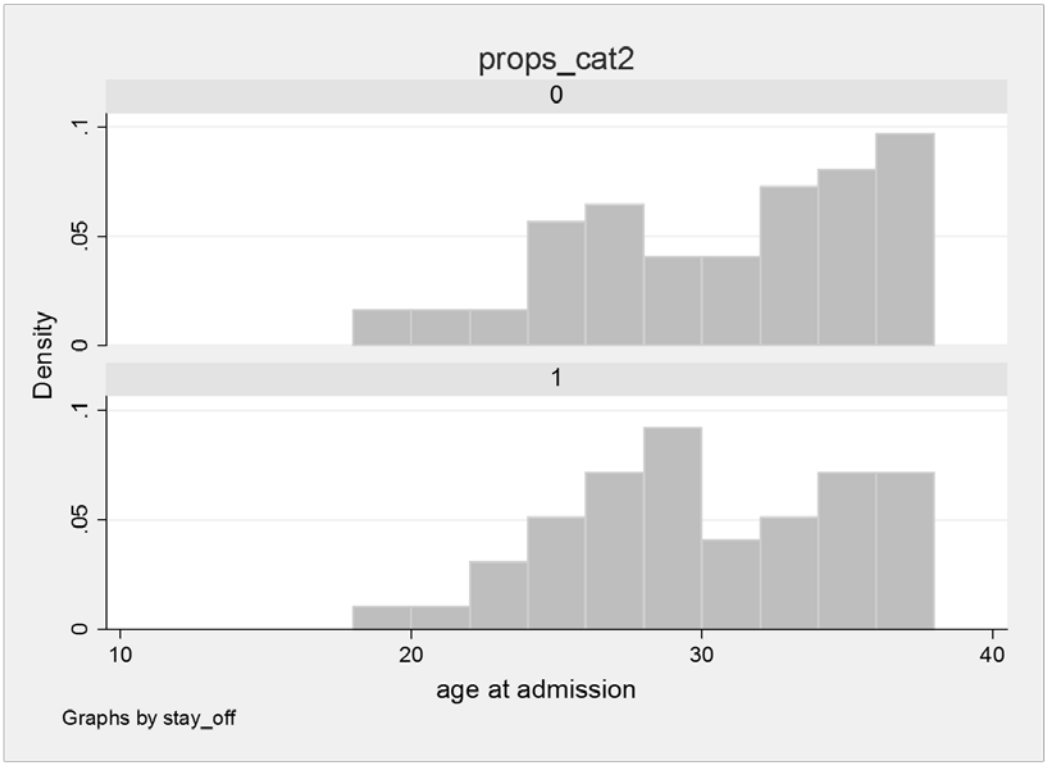
Creating the propensity scores

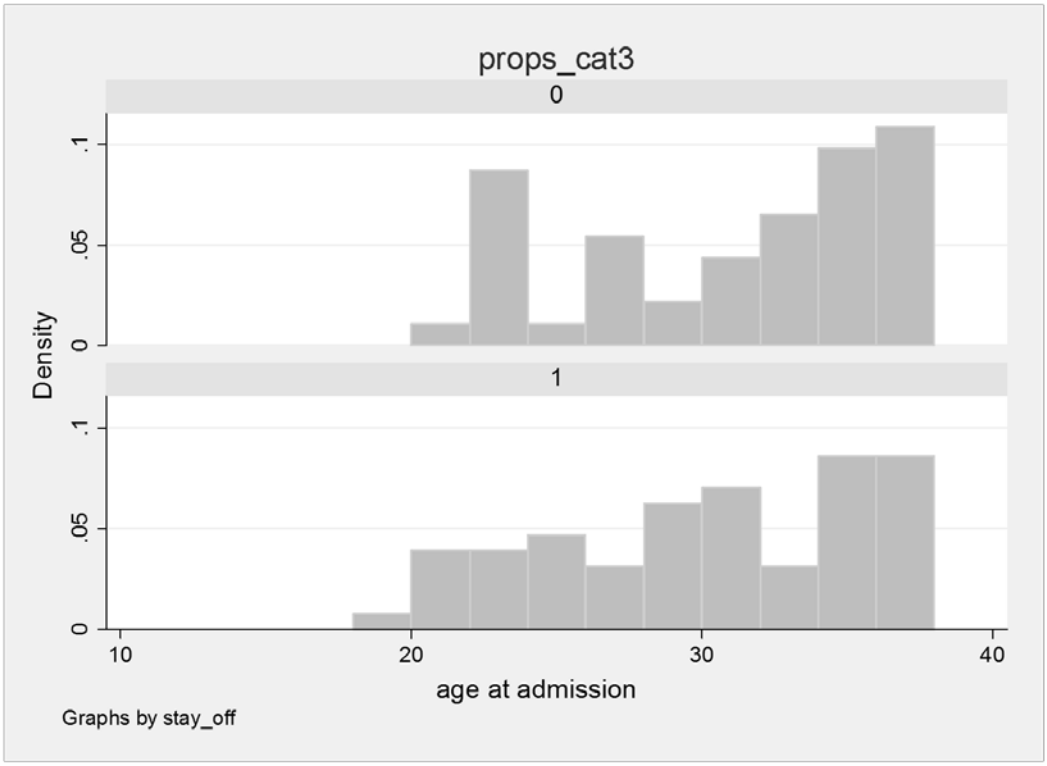
```
. xi:logistic i.stay_off age i.education_cat i.married_cat i.depression1 VASa
i.stay_off        _Istay_off_0-1      (naturally coded; _Istay_off_0 omitted)
i.education_cat   _Ieducation_1-5     (naturally coded; _Ieducation_1 omitted)
i.married_cat     _Imarried_c_1-4     (naturally coded; _Imarried_c_1 omitted)
i.depression1     _Idepressio_0-1     (naturally coded; _Idepressio_0 omitted)

Logistic regression                               Number of obs   =        550
                                                  LR chi2(10)     =      34.92
                                                  Prob > chi2     =     0.0001
Log likelihood =  -363.1571                       Pseudo R2       =     0.0459

------------------------------------------------------------------------------
_Istay_off_1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |   .9732198   .0172992    -1.53   0.127     .9398979    1.007723
_Ieducatio~2 |   .7664502   .2427203    -0.84   0.401     .4120258     1.42575
_Ieducatio~3 |   .8637698   .2909521    -0.43   0.664     .4463539    1.671539
_Ieducatio~4 |   .5381579   .2139994    -1.56   0.119     .2468471    1.173252
_Ieducatio~5 |   .6271358   .2284328    -1.28   0.200     .3071237    1.280589
_Imarried_~2 |   .5672423   .2538788    -1.27   0.205     .2359386    1.363761
_Imarried_~3 |   1.122614   .2735204     0.47   0.635     .6963672    1.809765
_Imarried_~4 |   .5439209   .1794741    -1.85   0.065     .2848855    1.038487
_Idepressi~1 |   1.250245   .3570069     0.78   0.434      .714389     2.18804
        VASa |   1.098051   .0415052     2.47   0.013     1.019643    1.182489
------------------------------------------------------------------------------


     props2 |      Freq.     Percent        Cum.
------------+-----------------------------------
          1 |        109       19.82       19.82
          2 |        111       20.18       40.00
          3 |        110       20.00       60.00
          4 |        110       20.00       80.00
          5 |        110       20.00      100.00
------------+-----------------------------------
      Total |        550      100.00
```

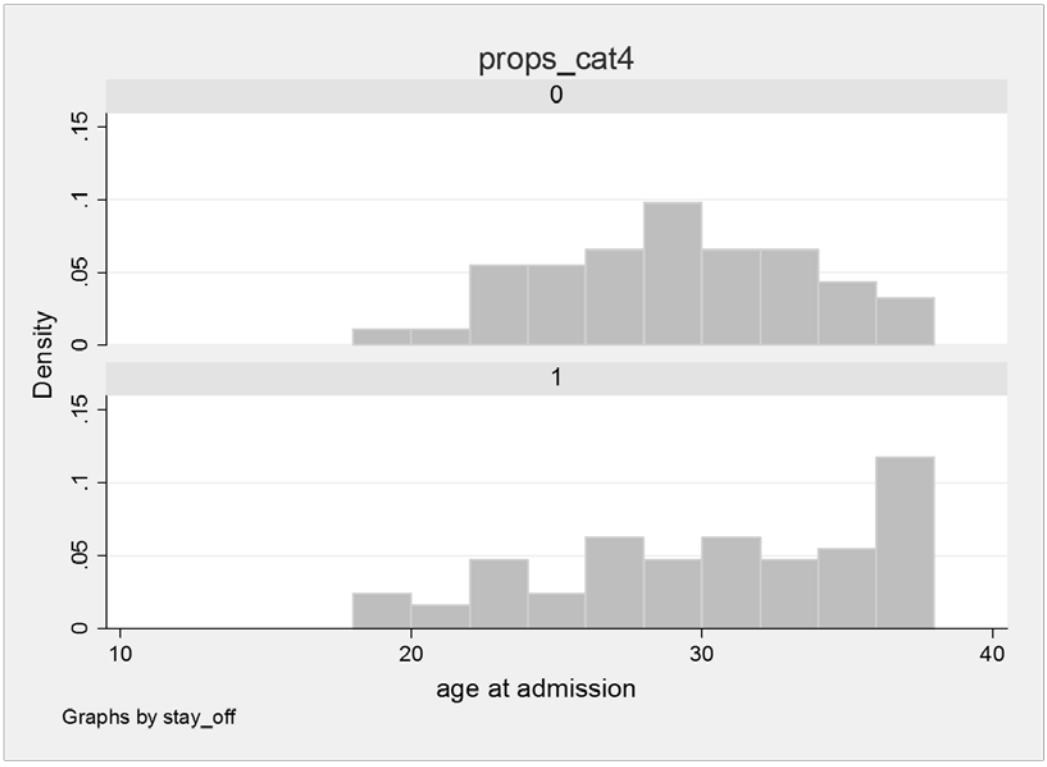
Comparing Distributions – Continuous Variables

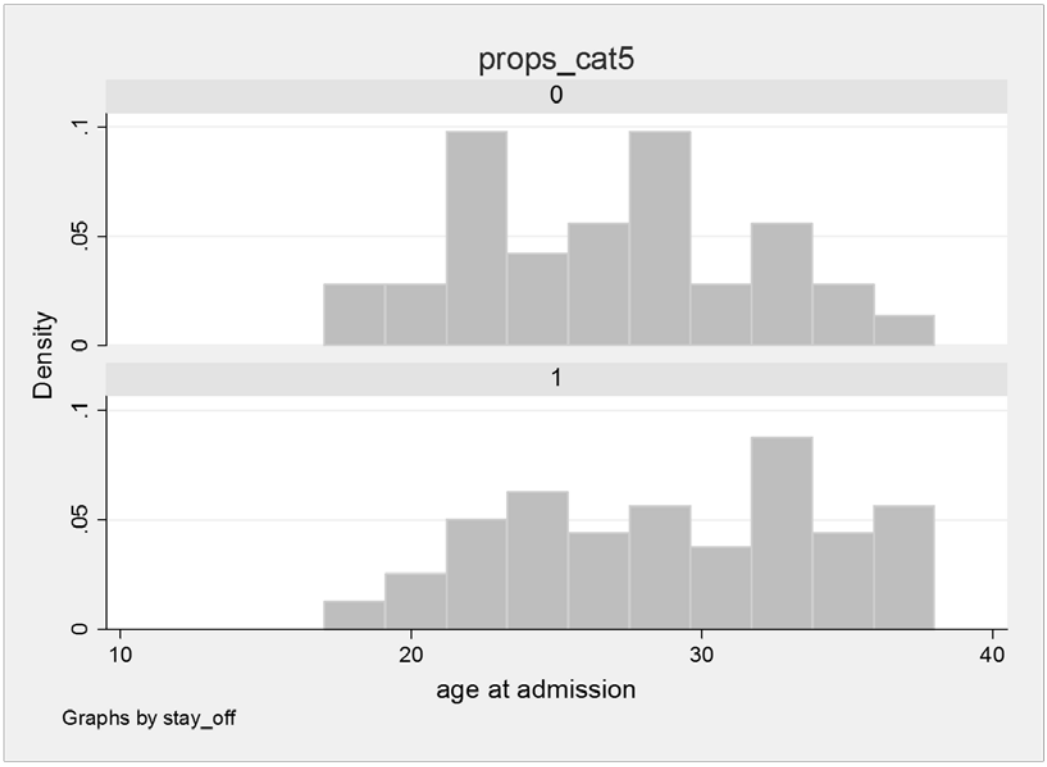
AGE (0 = at work; 1 = off work; props_cat = propensity quintile)



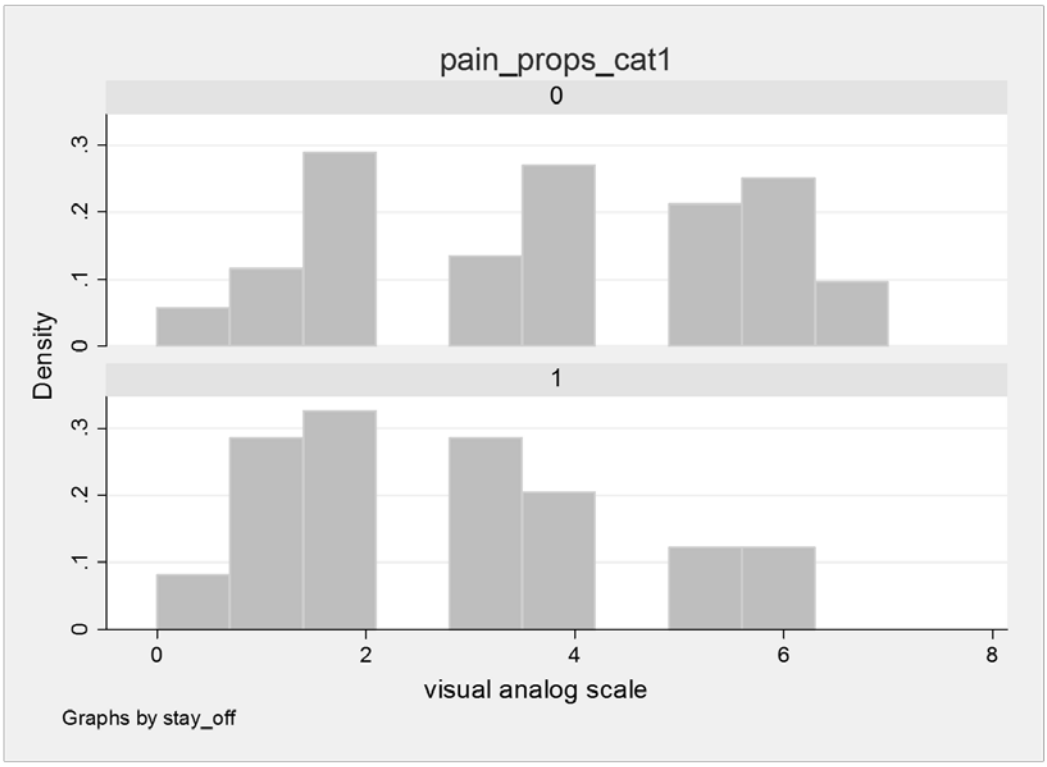


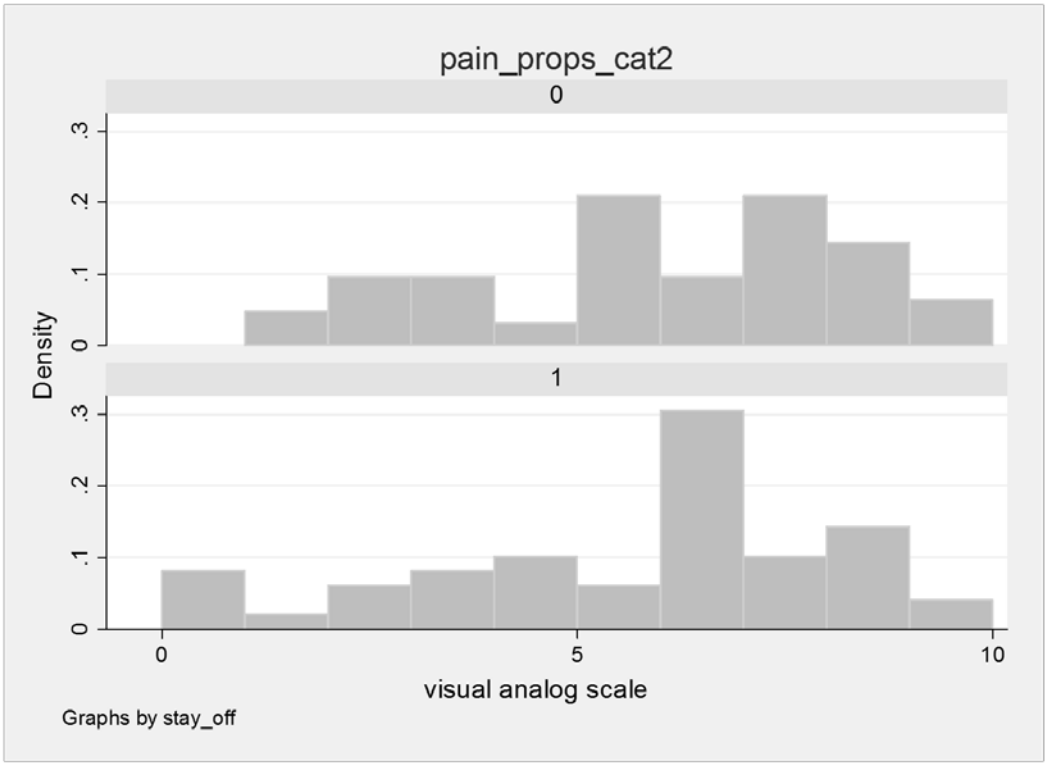

props_cat3
0
1
Density
0
.05
.1
10
20
30
40
age at admission
Graphs by stay_off

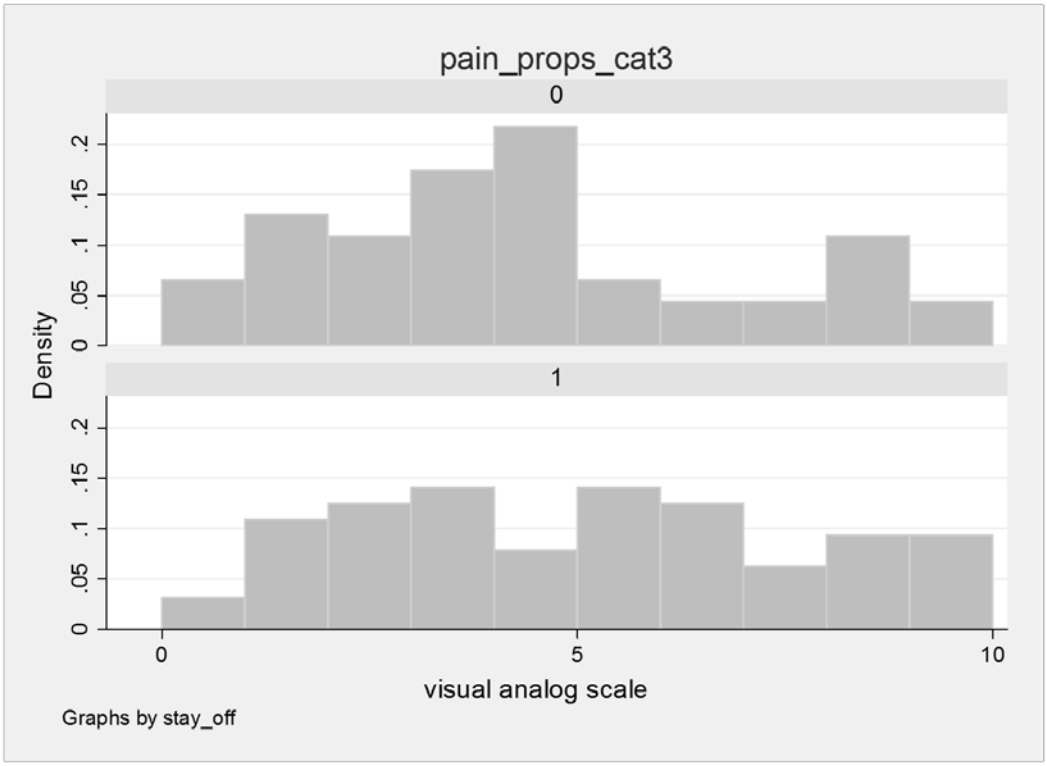

props_cat4
0
1
Density
0
.05
.1
.15
10
20
30
40
age at admission
Graphs by stay_off

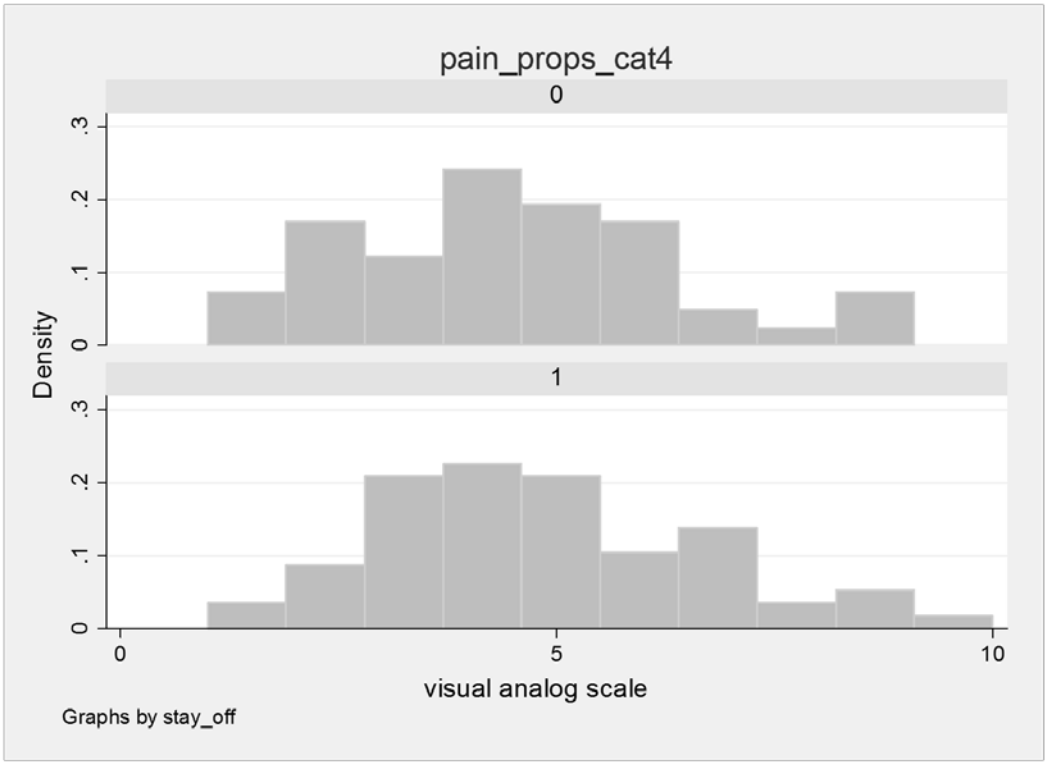

props_cat5
0
1
Density
0
.05
.1
10
20
30
40
age at admission
Graphs by stay_off

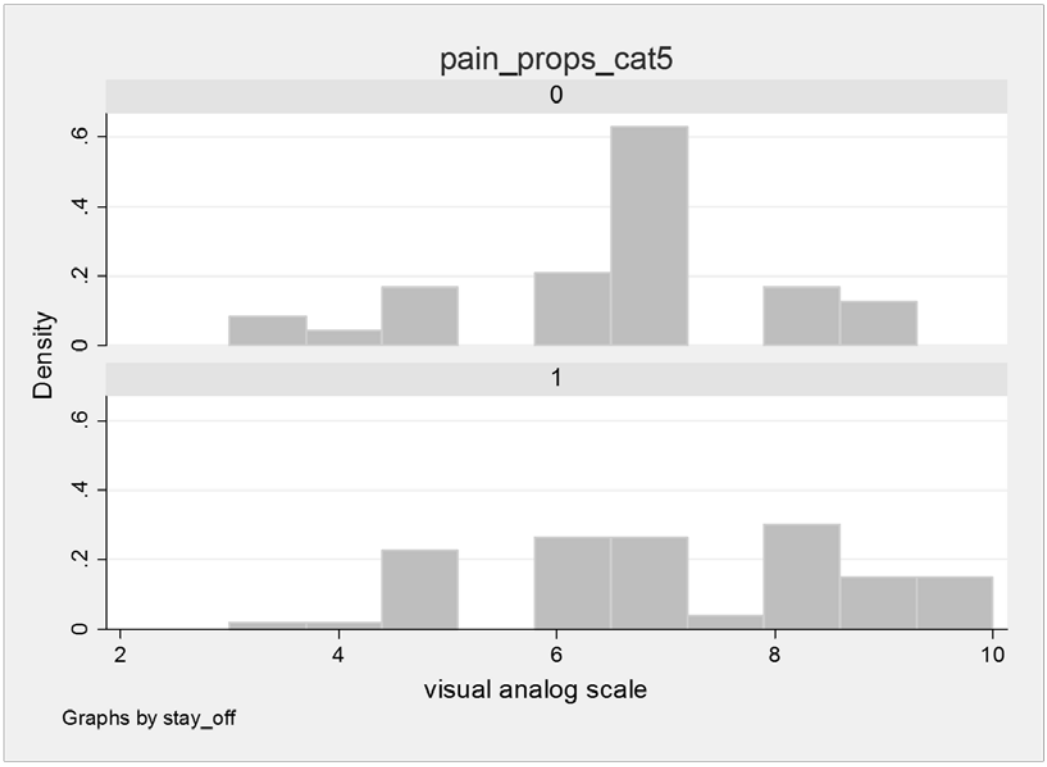
PAIN (props_cat = propensity quintile)






pain_props_cat3
0
1
Density
0
.05
.1
.15
.2
0
5
10
visual analog scale
Graphs by stay_off


pain_props_cat4
0
1
Density
0
.1
.2
.3
0
5
10
visual analog scale
Graphs by stay_off


pain_props_cat5
0
1
Density
0
.2
.4
.6
2
4
6
8
10
visual analog scale
Graphs by stay_off

Comparing Distributions – Categorical Variables

Education - Chi-squared test was used to assess for variable distribution differences between exposure groups

Propensity Category	Chi2	p-value
1	3.62	0.46
2	4.78	0.19
3	2.07	0.72
4	0.61	0.96
5	1.77	0.78

Marital Status - Chi-squared test was used to assess for variable distribution differences between exposure groups

Propensity Category	Chi2	p-value
1	5.21	0.16
2	0.21	0.98
3	3.81	0.28
4	0.06	0.97
5	2.75	0.25

Baseline Depression Status - Chi-squared test was used to assess for variable distribution differences between exposure groups

Propensity Category	Chi2	p-value
1	1.96	0.16
2	0.33	0.56
3	2.74	0.10
4	0.19	0.67
5	0.08	0.77

Appendix E – Chapter 5 Analysis Tables: Excluding prior CES-D scores from analysis

NOT DEPRESSED AT BASELINE

- Analyses were made with the "stay at work" exposure group as the reference category. In this series, most recent CES-D score is excluded as a potential confounder.
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Adjusted: Six-Weeks

```
Logistic regression                                     Number of obs   =       1964
                                                        LR chi2(16)     =     106.19
                                                        Prob > chi2     =     0.0000
Log likelihood = -464.28745                             Pseudo R2       =     0.1026

------------------------------------------------------------------------------
_Idepress_~1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   .7209957   .5560772    -0.42   0.671      .159013    3.269135
_Ibase_i1_~3 |   1.169616   .2988207     0.61   0.540     .7088811    1.929806
_Ibase_i1_~4 |   3.310364   .7818299     5.07   0.000     2.083733    5.259076
    neck_nrs |    1.07771   .0599768     1.34   0.179     .9663422    1.201913
    drawperc |   1.009276   .0063376     1.47   0.141     .9969311    1.021774
_Ieducate_~2 |   .5787378   .1531983    -2.07   0.039     .3444763    .9723092
_Ieducate_~3 |   .5278212   .1281212    -2.63   0.008     .3279969    .8493839
_Ieducate_~4 |   .4674708   .1539473    -2.31   0.021     .2451529    .8913985
 _Iheadcat_1 |    1.37365   .7293739     0.60   0.550     .4851869    3.889044
 _Iheadcat_2 |   1.140185   .4123423     0.36   0.717     .5612317    2.316371
 _Iheadcat_3 |   1.884454   .5440579     2.19   0.028     1.070129    3.318445
 _Iheadcat_4 |   1.630268   .4884895     1.63   0.103     .9061641    2.932992
 _Iheadcat_5 |   1.556386   .5749522     1.20   0.231     .7545197    3.210434
   _Idizzy_1 |   1.043193   .2153789     0.20   0.838     .6960243    1.563525
  _Ivision_1 |    2.57632   .7445718     3.27   0.001     1.462168    4.539438
   _Iworry_1 |   1.976254   .3719578     3.62   0.000      1.36658    2.857923
------------------------------------------------------------------------------
```

Adjusted: Three-months

```
Logistic regression                               Number of obs   =       1727
                                                  LR chi2(21)     =     116.90
                                                  Prob > chi2     =     0.0000
Log likelihood = -353.55344                       Pseudo R2       =     0.1419

------------------------------------------------------------------------------
 depress_fu2 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   .5977978   .4715991    -0.65   0.514      .127364    2.805835
_Ibase_i1_~3 |   1.181151   .3670502     0.54   0.592     .6423761     2.17181
_Ibase_i1_~4 |   1.961503   .5652334     2.34   0.019     1.115075    3.450437
    i1nk_nrs |   1.121241   .0574929     2.23   0.026     1.014035    1.239782
 _Ii1hdcat_1 |   1.968283   1.047554     1.27   0.203     .6935276    5.586136
 _Ii1hdcat_2 |   1.329125   .4773041     0.79   0.428      .657491    2.686842
 _Ii1hdcat_3 |   1.860744   .5648008     2.05   0.041     1.026401    3.373308
 _Ii1hdcat_4 |   1.601348   .5331855     1.41   0.157     .8338139    3.075403
 _Ii1hdcat_5 |   .1996218   .1607552    -2.00   0.045     .0411847    .9675639
_Ieducate_~2 |   .6099223   .1893223    -1.59   0.111     .3319389    1.120704
_Ieducate_~3 |    .538695   .1527531    -2.18   0.029     .3090117    .9390979
_Ieducate_~4 |   .7097227    .250631    -0.97   0.332     .3552189    1.418017
_Ii1_dizzy_1 |   2.261357   .5356681     3.44   0.001     1.421469    3.597501
_Ii1vision_1 |   1.012102   .3511919     0.03   0.972     .5127002    1.997951
_Ii1hearng_1 |   1.829378   .6632854     1.67   0.096     .8988346    3.723293
_Imenta_3c_1 |   2.365814   .8239531     2.47   0.013     1.195442    4.682012
_Iprhealth~2 |   1.071524   .2808028     0.26   0.792     .6411159    1.790882
_Iprhealth~3 |   2.584588   .7066585     3.47   0.001     1.512384    4.416929
_Iprhealth~4 |   1.616716    .885511     0.88   0.380     .5526012     4.72994
_Ii1recov_~1 |   1.108051   .2861958     0.40   0.691     .6678896    1.838292
_Ii1recov_~2 |   1.687212   .6618885     1.33   0.182     .7820705    3.639935
------------------------------------------------------------------------------
```

DEPRESSED AT BASELINE

- Analyses were made with the "stay at work" exposure group as the reference category. In this series, most recent CES-D score is excluded as a potential confounder.
- _Ibase_i1_~2, _Ibase_i1_~3, and _Ibase_i1_~4 refer to those who were initially at work and then went off, those who were off work and returned, and those who stayed off work during the follow-up period; respectively

Crude: Six-Weeks (no other variables qualified as a potential confounder)

```
Log likelihood =  -973.3419                     Pseudo R2       =     0.0345

------------------------------------------------------------------------------
 depress_fu1 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   2.404989   .6907722     3.06   0.002     1.369704    4.222787
_Ibase_i1_~3 |   1.003295   .1370071     0.02   0.981     .7676987    1.311192
_Ibase_i1_~4 |   2.738887   .3678213     7.50   0.000     2.105044    3.563584
------------------------------------------------------------------------------
```

Adjusted: Three-months

```
Logistic regression                             Number of obs   =       1250
                                                LR chi2(19)     =     238.21
                                                Prob > chi2     =     0.0000
Log likelihood = -644.47611                     Pseudo R2       =     0.1560

------------------------------------------------------------------------------
 depress_fu2 | Odds Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
_Ibase_i1_~2 |   1.458497   .5241925     1.05   0.294     .7210721    2.950071
_Ibase_i1_~3 |   .9320252   .1709372    -0.38   0.701     .6505997    1.335185
_Ibase_i1_~4 |   1.094934   .1836831     0.54   0.589     .7881207    1.521189
_Ii1recov_~1 |   1.449053   .2527989     2.13   0.033     1.029403    2.039779
_Ii1recov_~2 |   2.465851   .6298966     3.53   0.000     1.494615    4.068221
 _Ii1worry_1 |   3.080282   .5330519     6.50   0.000     2.194258    4.324077
_Ii1vision_1 |   1.826609   .3294821     3.34   0.001     1.282645    2.601264
_Ii1_dizzy_1 |   1.208602   .1876769     1.22   0.222     .8914664    1.638557
_Ii1armnmb_1 |   1.401217   .2041956     2.31   0.021     1.053081    1.864441
 _Ii1hdcat_1 |   .9169631   .5164594    -0.15   0.878     .3040404    2.765492
 _Ii1hdcat_2 |   .9864715   .2641466    -0.05   0.959     .5836588    1.667286
 _Ii1hdcat_3 |   1.196316   .2595757     0.83   0.409     .7819032     1.83037
 _Ii1hdcat_4 |   1.279374   .2742395     1.15   0.250     .8405016    1.947405
 _Ii1hdcat_5 |   1.378969   .3554197     1.25   0.212     .8320761    2.285313
    i1nk_nrs |   1.063342   .0361682     1.81   0.071     .9947651    1.136647
  _Iage_gp_2 |   .8620727   .2099796    -0.61   0.542     .5348256    1.389554
  _Iage_gp_3 |   1.423355   .2975896     1.69   0.091     .9448164    2.144269
  _Iage_gp_4 |   1.281293   .2775058     1.14   0.252     .8380949    1.958863
  _Iage_gp_5 |   1.883375   .4489502     2.66   0.008     1.180405    3.004984
------------------------------------------------------------------------------
```

Variance Inflation Factors for Three-Month Analysis: with and without prior CES-D

	CES-D		NO CES-D	
Variable	VIF	1/VIF	VIF	1/VIF
i1nk_nrs	4.23	0.236339	4.18	0.239396
cesd_i1	2.43	0.411999	--	--
li1recov~1	2.38	0.419558	2.35	0.426019
leducate~3	1.95	0.511659	1.94	0.514505
_lprhealth~2	1.88	0.533198	1.85	0.539889
_li1_dizzy_1	1.61	0.620861	1.57	0.635979
_li1hdcat_4	1.56	0.640166	1.56	0.641493
leducate~2	1.5	0.664937	1.49	0.669067
_li1hdcat_3	1.5	0.667041	1.49	0.671375
_lprhealth~3	1.41	0.706834	1.37	0.729715
leducate~4	1.29	0.775841	1.28	0.7784
_li1hdcat_5	1.28	0.778392	1.28	0.779988
li1recov~2	1.28	0.782767	1.26	0.791509
_li1hdcat_2	1.25	0.799786	1.25	0.797918
_li1vision_1	1.24	0.809552	1.22	0.817622
_lbase_i1_~4	1.23	0.813784	1.18	0.846897
_lbase_i1_~3	1.2	0.835652	1.18	0.850978
_li1hearng_1	1.15	0.869269	1.14	0.877502
_lprhealth~4	1.1	0.913124	1.08	0.922137
_lmenta_3c_1	1.09	0.918409	1.08	0.924361
_li1hdcat_1	1.07	0.930461	1.07	0.930861
_lbase_i1_~2	1.05	0.954138	1.05	0.95227
Mean VIF	1.58		1.52	

Correlation Matrix for Regression Variables (Chapter 5)

	depres~2	b~1_rt~t	i1nk_nrs	i1hdcat	educat~t	i1_dizzy	i1vision	i1hearng	menta_3c	prheal~2	i1reco~B	cesd_i1
depress_fu2	1											
base_i1_rt~t	0.0865	1										
i1nk_nrs	0.1429	0.12	1									
i1hdcat	0.124	0.0615	0.578	1								
educate_cat	-0.0653	-0.2235	-0.0542	-0.0303	1							
i1_dizzy	0.1702	0.1146	0.3003	0.3459	-0.0595	1						
i1vision	0.0707	0.0384	0.1615	0.1955	-0.0862	0.2885	1					
i1hearng	0.1062	0.0753	0.1075	0.1214	-0.016	0.2	0.2107	1				
menta_3c	0.0509	-0.0065	0.0371	0.0459	0.0053	0.0252	0.0051	0.0451	1			
prhealth2	0.1004	-0.0325	0.103	0.0893	-0.0742	0.0583	0.0642	0.0445	0.0943	1		
i1recov_B	0.1247	0.0945	0.507	0.3986	-0.0597	0.243	0.0905	0.0881	0.0114	0.0712	1	
cesd_i1	0.3072	0.229	0.3415	0.2991	-0.1217	0.3114	0.1989	0.1819	0.0839	0.1702	0.3128	1

Appendix F – The Center for Epidemiological Studies Depression Scale

(CES-D; Radloff 1977)

Centre for Epidemiological Studies – Depression Scale
CES-D

Using the scale below, indicate the number which best describes how often you felt or behaved this way -- DURING THE PAST WEEK.

0 = Rarely or none of the time (less than 1 day)
1 = Some or a little of the time (1-2 days)
2 = Occasionally or a moderate amount of time (3-4 days)
3 = Most or all of the time (5-7 days)

DURING THE PAST WEEK:

_____ 1. I was bothered by things that usually don't bother me.
_____ 2. I did not feel like eating; my appetite was poor.
_____ 3. I felt that I could not shake off the blues even with help from my family or friends.
_____ 4. I felt that I was just as good as other people.
_____ 5. I had trouble keeping my mind on what I was doing.
_____ 6. I felt depressed.
_____ 7. I felt that everything I did was an effort.
_____ 8. I felt hopeful about the future.
_____ 9. I thought my life had been a failure.
_____ 10. I felt fearful.
_____ 11. My sleep was restless.
_____ 12. I was happy.
_____ 13. I talked less than usual.
_____ 14. I felt lonely.
_____ 15. People were unfriendly.
_____ 16. I enjoyed life.
_____ 17. I had crying spells.
_____ 18. I felt sad.
_____ 19. I felt that people disliked me.
_____ 20. I could not get "going".

Appendix G – Ethics Approval Forms

APPROVAL FORM

Date: May 1, 2009

Principal Investigator: Linda Carroll

Study ID: Pro00006726

Study Title: Work, Injury and Depressive Symptoms

Date of Informed Consent: Approval Date Expiration Date Approved Document

Expiration Date: April 30, 2010

Thank you for submitting the above study to the Health Research Ethics Board (Health Panel). Your application has been reviewed and approved on behalf of the committee.

The ethics approval is valid until April 30, 2010. A renewal report must be submitted next year prior to the expiry of this approval if your study still requires ethics approval. If you do not renew on or before the renewal expiry date, you will have to re-submit an ethics application.

Approval by the Health Research Ethics Board does not encompass authorization to access the patients, staff or resources of Capital Health or other local health care institutions for the purposes of the research. Enquiries regarding Capital Health administrative approval, and operational approval for areas impacted by the research, should be directed to the Capital Health Regional Research Administration office, #1800 College Plaza, phone (780) 407-1372.

Sincerely,

Glenn Griener, Ph.D.,

Chair, Health Research Ethics Board, Panel B

Note: This correspondence includes an electronic signature (validation and approval via an online system).

Re-Approval Form

Date:	March 17, 2010
Amendment/Renewal ID:	Pro00006726_REN1
Study ID:	MS1_Pro00006726
Study Title:	Work, Injury and Depressive Symptoms
Principal Investigator:	Linda Carroll
Approval Expiry Date:	April 29, 2011

The Health Research Ethics Board - Health Panel has reviewed the renewal request and file for this project and found it to be acceptable within the limitations of human research.

The re-approval for the study as presented is valid for one year. It may be extended following completion of the annual renewal request before the approval expires. Beginning at 45 days prior to expiration, you will receive notices that the study is about to expire. Once the study has expired, you will have to resubmit. Any proposed changes to the study must be submitted to the Health REB for approval prior to implementation.

For studies where investigators must obtain informed consent, signed copies of the consent forms must be retained, as should all study related documents, so as to be available to the Health REB upon request. They should be kept for the duration of the project and for at least five (5) years following study completion.

Sincerely,

Glenn Griener, Ph.D.
Chair, Health Research Ethics Board - Health Panel

Note: This correspondence includes an electronic signature (validation and approval via an online system).

Re-Approval Form

Date:	May 3, 2011
Amendment/Renewal ID:	Pro00006726_REN2
Study ID:	MS3_Pro00006726
Study Title:	Work, Injury and Depressive Symptoms
Principal Investigator:	Linda Carroll
Approval Expiry Date:	April 27, 2012

The Health Research Ethics Board - Health Panel has reviewed the renewal request and file for this project and found it to be acceptable within the limitations of human research.

The re-approval for the study as presented is valid for one year. It may be extended following completion of the annual renewal request before the approval expires. Beginning at 45 days prior to expiration, you will receive notices that the study is about to expire. Once the study has expired, you will have to resubmit. Any proposed changes to the study must be submitted to the Health REB for approval prior to implementation.

For studies where investigators must obtain informed consent, signed copies of the consent forms must be retained, as should all study related documents, so as to be available to the Health REB upon request. They should be kept for the duration of the project and for at least five (5) years following study completion.

Sincerely,

Dr. Jana Rieger
Chair, Health Research Ethics Board - Health Panel

Note: This correspondence includes an electronic signature (validation and approval via an online system).

Approval Form

Date:	February 1, 2010
Principal Investigator:	Linda Carroll
Study ID:	Pro00008533
Study Title:	Remaining at work vs. time off work after a musculoskeletal injury: What are the consequences?
Approval Expiry Date:	January 31, 2011

Thank you for submitting the above study to the Health Research Ethics Board - Health Panel . Your application, along with final revisions received February 1, 2010, has been reviewed and approved on behalf of the committee.

The Research Ethics Board assessed all matters required by section 50(1)(a) of the Health Information Act. Subject consent for access to identifiable health information is required for the research described in the ethics application, and appropriate procedures for such consent have been approved by the REB Panel.

In order to comply with the Health Information Act, a copy of the approval form is being sent to the Office of the Information and Privacy Commissioner.

A renewal report must be submitted next year prior to the expiry of this approval if your study still requires ethics approval. If you do not renew on or before the renewal expiry date, you will have to re-submit an ethics application.

Approval by the Health Research Ethics Board does not encompass authorization to access the patients, staff or resources of Alberta Health Services or other local health care institutions for the purposes of the research. Enquiries regarding Alberta Health Services administrative approval, and operational approval for areas impacted by the research, should be directed to the Alberta Health Services Regional Research Administration office, #1800 College Plaza, phone (780) 407-6041.

Sincerely,

Glenn Griener, Ph.D.
Chair, Health Research Ethics Board - Health Panel

Note: This correspondence includes an electronic signature (validation and approval via an online system).

Health Research Ethics Board

308 Campus Tower
University of Alberta, Edmonton, AB T6G 1K8
p. 780.492.9724 (Biomedical Panel)
p. 780.492.0302 (Health Panel)
p. 780.492.0459
p. 780.492.0839
f. 780.492.9429

Re-Approval Form

Date:	July 4, 2011
Amendment/Renewal ID:	Pro00016880_REN1
Study ID:	MS1_Pro00016880
Study Title:	Development of a Triage Decision-Making Tool for the Rehabilitation of Injured Workers
Principal Investigator:	Douglas Gross
Approval Expiry Date:	August 14, 2012

The Health Research Ethics Board - Health Panel has reviewed the renewal request and file for this project and found it to be acceptable within the limitations of human research.

The re-approval for the study as presented is valid for one year. It may be extended following completion of the annual renewal request before the approval expires. Beginning at 45 days prior to expiration, you will receive notices that the study is about to expire. Once the study has expired, you will have to resubmit. Any proposed changes to the study must be submitted to the Health REB for approval prior to implementation.

For studies where investigators must obtain informed consent, signed copies of the consent forms must be retained, as should all study related documents, so as to be available to the Health REB upon request. They should be kept for the duration of the project and for at least five (5) years following study completion.

Sincerely,

Dr. Jana Rieger
Chair, Health Research Ethics Board - Health Panel

Note: This correspondence includes an electronic signature (validation and approval via an online system).