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The impact of obesity on treatment outcomes in a multidisciplinary chronic pain management program

Dana Mackin Pilchik
Pacific University

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The impact of obesity on treatment outcomes in a multidisciplinary chronic pain management program

Abstract
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THE IMPACT OF OBESITY ON TREATMENT OUTCOMES IN A
MULTIDISCIPLINARY CHRONIC PAIN MANAGEMENT PROGRAM

A DISSERTATION
SUBMITTED TO THE FACULTY
OF
SCHOOL OF PROFESSIONAL PSYCHOLOGY
PACIFIC UNIVERSITY
HILLSBORO, OREGON

BY
DANA MACKIN PILCHIK

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PSYCHOLOGY
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Dedication

To my family and, especially, to my extraordinary husband Evan.
Who could have thought that any of this was possible,
Except that you have made it so.
Thank you for believing me in and carrying me through
This incredible adventure.

To the me of 10 years ago,
Who first thought she might like to be a clinical psychologist; and
To Kristy,
Who was the first to agree that this idea seemed totally plausible:

Thank you.
I have never looked back.

To my darling Evie,
You are my sunshine.
Mommy can come play now.

Heartfelt thanks to the Patients and Staff at Progressive Rehabilitation Associates.
Abstract

This study examined the relationship between chronic pain and obesity, specifically examining the effects of obesity on multidisciplinary chronic pain treatment outcomes. Participants completed a 20-day intensive multidisciplinary chronic pain treatment program, and completed pre-post treatment measures assessing chronic pain. These measures were retrospectively analyzed to compare treatment response between obese and non-obese individuals. Mixed between-within ANOVA revealed that both obese and non-obese individuals responded dramatically to MCPM and there were no significant differences between groups on any of the provided measures. These findings suggest that even the most obese individuals benefitted significantly from MCPM, and suggest that chronic pain treatment may be a valuable precursor or adjunct to obesity and weight-loss treatment programs.

*Keywords: chronic pain, multidisciplinary chronic pain management, obesity*
Introduction

Chronic pain causes extensive damage in terms of public health, economic costs, and human suffering. The effects on an individual’s life of long-lasting pain that is not alleviated by drugs, surgery, or physical therapy can be ruinous, leading to depression, disability, and isolation. Individuals can become defined by fear and catastrophization, by their ongoing search for a cure, and by their perceived inability to cope with the basic elements of existence. Pain has been a primary focus of medical treatment since the advent of healthcare, and continues to be the number one reason people visit their physician, accounting for 70-80% of all doctor’s visits (Gatchel & Okifuji, 2006; Turk, 1996). Despite this, despite advances in understanding of pain mechanisms, and despite the development of advanced diagnosis and treatment, there remains no cure available that reliably and permanently eliminates pain for all patients (Turk). Biomedical approaches such as surgery and medications are often pursued relentlessly; unfortunately a significant portion of patients find that their pain has not been alleviated, but rather may have worsened. The distress of constant pain is often compounded by an accompanying hopelessness and demoralization brought about by a patient’s futile search for relief. Because this wide range of psychosocial factors both contribute to and are consequences of chronic pain, comprehensive treatments focus on incorporating these in order to better address the full range of an individual’s suffering. While there may not yet be a cure for chronic pain, contemporary approaches focus on restoring emotional and physical functionality to an individual despite their persisting pain and disability.

Multidisciplinary (or interdisciplinary) chronic pain management (MCPM) programs have become a popular treatment option for chronic pain because they provide cost-effective and
comprehensive care for treatment-refractory patients (Gatchel & Okifuji, 2006; Tunks, Weir, & Crook, 2008; Schatman, 2012; Turk & Burwinkle, 2005). MCPM was initially developed in response to the complexity of treating chronic pain, and is defined primarily as a rehabilitation program that incorporates multiple therapy modalities (Gatchel & Okifuji; Schatman). The focus within the most comprehensive of these is upon functional restoration, an intensive treatment approach that targets the physical deconditioning, chronic disability, pharmacological dependence, and the psychosocial and socioeconomic sequelae often associated with chronic pain. Patients are ideally taught methods to self-manage their pain rather than maintaining a focus on eliminating their pain. MCPM programs may offer important ways for patients to significantly improve their lives and general well-being.

Numerous studies evaluating treatment outcomes of chronic pain management programs provide evidence that patients experience outcomes such as reduced pain intensity, improved mental health, increased functional tolerances, increased ability to self-manage pain, decreased life interference, decreased disability, improved health-related quality of life, and increased return to work (Flor, Fydrich, & Turk, 1992; Gatchel & Okifuji, 2006; Hoffman, Papas, Chatkoff, & Kerns, 2007; Morley, Eccleston, & Williams, 1999). However, a substantial proportion of patients do not appear to benefit equally from chronic pain management programs, as these studies also show significant variability in response to treatment and small to medium effect sizes (Thorn, Cross, & Walker, 2007; Turk & Rudy, 1990; Vlaeyen & Morley, 2005). Investigations of factors that predict treatment response have also produced inconsistent findings, as measuring and interpreting interaction effects between treatment and patient characteristics becomes exceedingly complex (McCracken & Turk, 2002; Turner, Holtzman, & Mancl, 2007). In a review of the literature, McCracken and Turk concluded that “no set of variables has been
shown to predict treatment outcome consistently,” (p. 2568) noting that results appear to depend on various patient characteristics, length of follow-up evaluation, and the particular outcome variable under study.

However, it does not appear that treatment outcome studies have addressed a potentially moderating variable found frequently in chronic pain populations. Though previous findings (Burns, Johnson, Mahoney, Devine, & Pawl, 1998) indicated that physical and demographic characteristics of patients did not appear to influence treatment outcomes, Sellinger, Clark, Shulman, Rosenberger, Heapy, and Kerns (2010) determined that non-obese patients showed statistically significantly greater improvement following cognitive-behavioral treatment for chronic low-back pain than did their obese counterparts. Obesity occurs frequently with chronic pain and chronic pain is a prominent characteristic of obesity. Both obesity and chronic pain have been shown to be strongly associated with lower levels of emotional and physical well-being and quality of life (Doll, Petersen, & Stewart-Brown, 2000). Despite this, the body mass index (BMI) of patients is rarely reported within pain literature (Sellinger et al.). Though there are examples in the literature noting the impact of chronic pain on the treatment for obesity, to date there are no other published studies that have compared the treatment outcomes of obese and non-obese chronic pain patients, and there have been no published studies comparing treatment outcomes of obese and non-obese patients specifically within a MCPM program.

The function of a moderating variable is to affect the direction and strength of the relationship between variables; it specifies when and at what specific levels certain effects will occur; these conditions occur causally, at the same level as predictors, and are typically analyzed through ANOVA interaction (Vlaeyen & Morley, 2005). If obesity is a moderator, then it is an uncontrolled variable that may be affecting the strength of the relationship between treatment
and outcome and may account for variance in treatment outcomes. It may also indicate a patient characteristic that can addressed in order to better develop treatment interventions and target limited resources to those most likely to benefit (MacKinnon & Luecken, 2008). There has not been sufficient empirical evidence of obesity’s moderating effect on treatment to warrant its designation as an *a priori* moderator in an RCT of chronic pain treatment (Kraemer, Frank, & Kupfer, 2006), however an archival study may detect a treatment interaction effect, particularly with a relatively large sample size.

*About This Study*

The objective of this study was to see if treatment outcomes for obese patients differed from those of non-obese participants who completed a 20-day intensive MCPM program. A retrospective review of patient medical records from a multidisciplinary chronic pain treatment program was performed to determine if patients with a BMI of 30 and over (the standard cut-off measure of obesity used by the World Health Organization) showed different treatment outcomes than patients with a BMI of under 30. In comparing the mean amount of change on each measure that non-obese patients make against the amount of change made by obese patients, this study expected to show that non-obese patients will show greater positive change (improvement) from pre- to post- treatment on measures of pain intensity, emotional distress, perceived disability, and pain-related fear and anxiety.

The first goal was to evaluate the whether the effectiveness of the pain management program, as shown by significant improvement in patient functioning following treatment, varied according to whether a patient was classified as obese or non-obese. Although randomized, controlled trials are ideal for gauging treatment efficacy, previous studies have provided evidence of the overall effectiveness of MCPM programs, thus the non-randomized, non-control
group design based on archival records was determined to be sufficient to show a time effect attributed to treatment.

The primary hypotheses were that patients who participated in and completed the treatment program would show statistically and clinically significant improvements in the domains of pain intensity, depression, pain related anxiety ability to self-manage pain, and perceived disability; the null hypothesis would be that participants showed no difference in test scores from pre- to post-treatment. Additionally, these improvements would vary according to whether the participants were classified as obese or non-obese. Finally, obese participants would show greater severity of impairment across treatment on self-report instruments of average pain intensity, emotional functioning, perceived disability, and self-efficacy.

The second goal was to explore if obesity is associated with less improvement in response to multidisciplinary treatment. Thus second set of hypotheses stated that following treatment, obese chronic pain patients would show significantly less change on measures of pain intensity, disability, emotional distress, and ability to self-manage pain than non-obese chronic pain patients. The null hypothesis in this instance is there will be no statistically significant difference in average change scores on the measures when comparing obese patients to non-obese patients.

The following review of the literature will provide an overview of the problem, including the prevalence of chronic pain, the global economic and health impact of chronic pain, and the associated biopsychosocial sequelae. Further, I will also describe the phenomenon of chronic pain and how it is conceptualized by contemporary biopsychosocial pain models. Understanding the biopsychosocial conceptualization of pain is essential to understanding the rationale for multidisciplinary treatment of chronic pain. I will also provide a brief review of the extensive
literature on treatment outcomes and the role of the specific psychosocial variables measured in this study in chronic pain treatment. Lastly, I will consider the significant comorbidity between obesity and chronic pain, review their interdependent aspects, discuss the impact that obesity has upon treatment for other conditions, and discuss how these indicate its possible role within chronic pain management.

*Chronic Pain*

The International Association for the Study of Pain (IASP) states that “chronic pain is among the most disabling and costly afflictions in North America, Europe, and Australia” (2003). It is believed the effects of chronic pain are likely similar in developing countries, though data is not available. The global prevalence of chronic pain is estimated to be approximately 20%. Based on a systematic review of 13 studies evaluating chronic pain in 20 countries, with sample sizes ranging from 410 to 17,496, the IASP estimated the weighted mean prevalence of chronic pain in adult populations was 35.5%, with estimates ranging from 10.1% to 55.2%. The data also indicated higher prevalence of CP among females and dramatically increased use of healthcare resources by CP patients. In 2011, the American Academy of Pain Management estimated that 100 million Americans suffer from chronic pain and their epidemiological studies estimated that 10 – 20% of the general North American population experiences chronic pain of over three months, with a lifetime rate of 29%. It was also reported that 57% of adult Americans experienced recurrent or chronic pain in the past year, with 62% of these individuals reporting pain lasting over one year. Furthermore, individuals age 50 years and older were found to be twice as likely to be diagnosed with chronic pain, while there are approximately 35 million US residents 65 and older, and the proportion of the population aged 65 is predicted to increase by 57% by 2030 (AAPM, 2011).
Thus, chronic pain is a pervasive and substantial problem for the American economy, directly affecting over 50 - 100 million Americans, costing over $70 billion annually in healthcare costs, lost productivity, tax revenue, and disability benefits, and accounting for more than 80% of all doctor visits (Gatchel & Okifuji, 2006; Turk, 1996). Moreover, these costs were usually compounded by the comorbid physical, functional, and/or psychiatric disorders found in about 87% of chronic pain patients. With 312 million prescriptions for analgesics written and 317,000 lumbar surgeries performed each year, the medication and surgical costs add to the figure substantially (Gatchel & Okifuji). Similar findings for healthcare and economic costs have been found in the United Kingdom, The Netherlands, New Zealand, Australia, Denmark, Canada, Spain, and Italy (Gatchel & Okifuji).

Pain itself is an adaptive response to bodily harm and is a natural result of injury or disease, and which likely abates with healing (Kerns, Sellinger, & Goodin, 2010). Chronic pain differs considerably from acute pain and should be understood as a complex interaction between physiological and psychosocial factors conceptualized within a biopsychosocial model (Engel, 1977; Main & Spanswick, 2000). While acute pain sounds the alarm that alerts us to damage, chronic pain persists beyond the point of serving an adaptive purpose. Chronic pain can have a number of etiologies or related conditions including spinal injury or degradation, cancer, fibromyalgia, migraine headache, neuropathic injury, musculoskeletal injury, and rheumatoid arthritis. In some cases, however, the etiology is unknown or may have no objective pathophysiology. The correlation between objective physical findings that might be found on an MRI or X-ray and complaints of pain is often fairly low, with patients with severe pain demonstrating no objective evidence of injury while patients with abnormal findings on imaging tests will report little to no pain. This may be due to the limitations of current technology;
inflammation is difficult to detect though is a common factor in chronic pain. This may also be
due to the fact that severe pain can develop secondary to systemic conditions such as diabetic
neuropathy and nervous system dysfunction (Gatchel, Peng, Peters, Fuchs, & Turk, 2007).

The IASP (2003) defines chronic pain as pain with little physiological purpose that has
continued beyond expected appropriate healing time (suggested to be three months). Others have
loosely defined chronic pain as any pain that lasts longer than six months (Gatchel et al., 2007).
In many cases, chronic pain is diagnosed at the point where there is simply no longer an
objective finding for its cause (Main & Spanswick, 2000). When an assessment focused solely
on determining a physical cause for the pain is unable to do, the pain is often attributed to
disordered psychology, perhaps resulting in diagnoses of malingering or somatoform disorder
(Gatchel & Okifuji, 2006). The effect of this is that, too often, frustrated physicians imply that a
patient’s pain is imaginary. People with chronic pain often feel that they are put in the position of
needing to express the degree of pain they are in, lest their suffering otherwise not be taken
seriously. Moreover, individuals are often inadvertently reinforced for behavioral expressions of
pain by solicitous coworkers and family members (Fordyce, 1976). Thus, even in terms of
obtaining a diagnosis, psychological and social factors play a critical role in the subjective
experience of chronic pain.

There are a range of different pain syndromes that fall under the rubric of chronic pain,
including musculoskeletal pain, myofascial pain and fibromyalgia; neuropathic pain such as
complex regional pain syndrome, diabetic neuropathy, or neuritis; and different types of
headaches and conditions such as cancer (Gatchel & Turk, 1996). And while there any many
individuals in the US with chronic pain, most do not develop a complicated and problematic
“chronic pain syndrome” that negatively impacts their functioning in a wide range of areas.
Many people are able to address elements of chronic pain through simple physical therapy or low-grade analgesics. However for a portion of patients, the following psychosocial sequelae begin to impact a significant portion of their daily existence, including: reduced activity, social withdrawal, poor sleep, depression, suicidal ideation, irritability, fatigue, memory deficits, and attention problems, damaged self-esteem, reduced libido, relationship difficulties, fear of movement, helplessness, hopelessness, substance abuse, guilt, anxiety, disability, employment and financial troubles, and low-self efficacy (US Dept. of Veterans Affairs, 2013). Accordingly, these factors develop over time and contribute an important aspect to an individual’s suffering. These factors may pre-exist and predispose people to pain, acting as an antecedent to symptoms; they can diminish or amplify pain perceptions, be a consequence of chronic pain, and/or serve as maintaining factors (Gatchel et al., 2007).

Thus, chronic pain causes significant changes in the familial, social, and physical roles of individuals, often creating years of suffering for many people. Most studies examining patient experience of chronic note that the subjective emotional experience inevitably tends to be quite negative (Gonzales, Martelli, & Baker, 2000). Evidence shows that depression, anxiety, anger, fear, and hopelessness are prominent comorbid aspects of chronic pain that can serve to maintain or even amplify the degree of disability experienced by the individual (Gatchel, et al., 2007; Williams, Jacka, Pasco, Dodd, & Berk, 2006).

It may be helpful to consider the development of chronic pain in an individual through the following scenario developed by Simon and Folen (2001):

> Often an injury, whether major or minor, causes the initial onset of pain, which leads to an initial decline in physical activity as part of early recovery.

> Lack of physical activity often causes ongoing muscle weakness and likely weight
gain, which can then aggravate muscles spasms, further exacerbating the chronic pain condition. As the limitations caused by the pain begin to take root, concomitant psychological issues may arise as the individual is forced to make undesired changes in work, family, and physical roles. The ongoing pain acts as a stressor, activating the sympathetic nervous system and thereby decreasing the individual’s pain threshold. Lack of medical progress, inability to work, disrupted family relationships, lack of exercise, and isolation all combine to increase anxiety and depression. Increased anxiety and depression often then serve to amplify the perception of pain. Moreover, pain and anxiety disrupt sleep patterns, causing both sleep onset and maintenance problems, often resulting in increased muscle tension, stress, depression, and anxiety. Individuals with severe chronic pain have been provided heavy doses of opiates, which may initially relieve the pain but can eventually increase pain sensitivity and intensify struggles with depression, anxiety, and anger. Anger and frustration continue to build as symptoms persist, the etiology of the pain remains unknown, and treatment failures accumulate.

Considering this scenario, it is evident that chronic pain must be understood not only consider the biomechanisms underlying the pain, but through the cognitive, affective, behavioral, and social factors that further impact both the physical and psychosocial experience of pain. The phenomenon may best understood by comparing different theoretical models of pain, particularly the comprehensive biopsychosocial model.
Theoretical Models of Pain

Historical theories of pain, such as Specificity Theory, presumed that pain always represents ongoing physiological injury and that the intensity is in exact proportion (isomorphic) to the amount of damage being sustained (Abram, 1993; Deardoff, 2003). Though this perception of pain existed since ancient Greece, French philosopher and mathematician Rene Descartes codified a mind/body duality in the 16th century that became the medical and philosophical heuristic of Western though for centuries. Since that time, the isomorphic model was been found overall to be an adequate heuristic for particular injuries and the immediate and short-term pain associated with them, and thus, provides the foundation for most medically-based pain interventions.

The modern biomedical understanding of Specificity Theory proposes that special nerve fibers respond to stimuli such as pain, temperature, pressure, or location and transmit these signals (termed nociception) to the spinal cord and the brain (Siegele, 1974). There, the thalamus is stimulated, arousing the perception of pain, while the cerebral cortex interprets the impulses in terms of location and intensity. The cerebral cortex then sends descending nerve impulses that stimulate autonomic, musculoskeletal, and psychological responses. Analgesics are believed to depress the central nervous system at the thalamus and cerebral cortex, thus interrupting the perception and response to pain.

Over time various limitations to this approach became evident through the observation of widely differential responses to injury and nociception, and the phenomenon of “phantom pain” in which an individual continues to experience pain in an amputated limb. However, as late as the mid-20th century (and still today), chronic pain was conceptualized through a primarily biomedical approach focused on diagnosing and treating underlying pathology, with pain
considered to be in proportion to the severity of the assumed pathology. Treatment focused on identification of underlying pathology and its removal or treatment, which presumably would result in the elimination of pain. And if no specific pathophysiology was localized, then the pain was considered psychogenic (Vlaeyen & Morley, 2005).

With the advent of Gate Control Theory (Melzack & Wall, 1965), there came a dramatic increase in research of the role of psychological moderators and mediators with pain. Gate control theory presented an alternative to traditional biomedical theories such as Specificity Theory, which did not provide explanation for or understanding of the influence of psychological factors on the perception of pain, idiosyncratic responses to injury, persisting pain beyond the expected recovery time, or phenomena such as phantom limb pain. Melzack and Wall integrated certain elements from earlier pain models while accounting for specific observations that had puzzled researchers and clinicians, specifically: 1) the variable relationship between injury and pain; 2) the observation that benign stimuli occasionally cause pain; 3) the inconsistent locations of pain related to injury; 4) the persistence of pain long past an injury’s healing; 5) the changeability of the experience of the pain and its location over time; 6) the multidimensional nature of pain; and 7) the lack of adequate chronic pain treatments (Gatchel et al., 2007). A significant change to the conceptualization of chronic pain took place once it was understood that pain was not simply the isomorphic representation of afferent nociception, but could be significantly altered by descending pathways (Vlaeyen & Morley, 2005).

The distinctive aspect about Gate Control is the proposition that facilitatory neurons (“gates”) in the spinal cord, brain stem, or cerebral cortex can block or intensify the transmission route of a pain signal, resulting in altered pain perception (Deardoff, 2003; Siegele, 1974). Furthermore, the ability to modify the pain signal is greatly influenced by psychological factors
such as anxiety, fear, and depression. Gate Control proposes that the same mechanisms in the cerebral cortex that are responsible for sensory-discriminatory, motivational-affect, and cognitive processes also relay impulses that close or open the gates.

Thus psychological factors can cause facilitatory interneurons on the transmission route to block the pain signal, resulting in little or no pain perception regardless of the amount of tissue damage occurring. Likewise, the same cells can intensify the pain stimulus along the route, resulting in far greater pain perception than warranted by the physical injury. Perhaps the theory’s most important contribution to the field is the way that it changed assumptions about pain perception, asserting that the introduction of central nervous system mechanisms that modulate the experience of pain meant that pain could never again be conceptualized as the sole product of peripheral input (Gatchel et al., 2007). This proved to be a major advancement in pain research and treatment, though its explanatory value continues to be examined.

Melzack (as cited in Gatchel et al., 2007) built upon Gate Control Theory with Neuromatrix theory, which served to provide further explanation for phenomena such as phantom limb pain and chronic pain. Neuromatrix theory proposes that pain is the consequence of an extensive “neural network” within the brain, rather than a isomorphic response to injury. The theory stipulates that the function of this distributed neural network (which Melzack termed the *body-mind neuromatrix*) is to activate sensory-perceptual, behavioral, and homeostatic systems in response to injury or stress. In an elegant arrangement of multiple constructs, Melzack proposed that chronic pain develops through a series of events following the original injury. In this model, ongoing pain creates a homeostatic imbalance, resulting in stress-induced triggering of the autonomic nervous system and eventual hypothalamic-pituitary-adrenal (HPA) axis dysregulation. Similar to the exhaustion phase of General Adaptation Syndrome (Selye, 1950),
consequences of ongoing stress will include eventual muscle atrophy, impaired tissue repair, and immune system suppression (Gatchel et al.). Thus, the biological and psychological sequelae of chronic pain, serve to reinforce and sustain pain perception and physical deconditioning, thus contributing to the chronicity of the condition in a positive feedback loop.

Interestingly, contemporary neuroscience and genetic research has substantiated much of this conceptualization, providing evidence that genetic factors, neurotransmitters, and hormones play a crucial role in the development and maintenance of chronic pain (Gatchel et al., 2007). Thus, Neuromatrix theory continued the trend of shifting the focus of pain research away from sensory mechanisms and towards cognitive, emotional, and behavioral factors capable of producing experiences in the body.

Melzack and Casey (1969) expanded understanding of the role of emotion by proposing that emotional distress is concomitant with nociception rather than its consequence, further integrating the objective physical nociception (“pain”) and subjective emotional experience of pain (“suffering”). This increased understanding of the emotional pain concomitant with nociception has provided insight into a critical area for patient care. Gate Control Theory, considered in the context of the Biopsychosocial model of health (Engel, 1977), led to the development of Biopsychosocial models of pain built around the idea that the experience of pain was not only the product of nociception, but also an individual’s subjective cognitions, affect, behavior, and environment. Key to the model is the realization that the numerous biopsychosocial sequelae of chronic pain are capable of creating a positive feedback loop, with consequences serving as maintaining and aggravating factors (Gatchel, et al., 2007). Thus, the Biopsychosocial model of pain provides a more sophisticated understanding of the complex role that stress and mood play in the perception of pain, and made explicit a comprehensive paradigm
that shows how cognition, behavior, and environment specifically interact with physiology to generate, aggravate, and maintain chronic pain. These comprehensive and multidimensional understandings of chronic pain have largely replaced the simpler biomedical models and have generated a wealth of research (Kerns et al., 2011). The Biopsychosocial model also made way for a wide range of intervention avenues for patients whose pain had otherwise been treatment refractory and forms the basis for multidisciplinary treatment of chronic pain (Kerns et al.,).

The Role of Psychological Factors in the Experience of Chronic Pain

Perception of Pain Intensity

As noted, the nature or severity of an injury does not reliably predict the perception of pain or the suffering endured. The premise of gate control theory is that the pain signal (nociception) can be potentially modulated, blocked, or enhanced, by chemical “gates” that are responsive to stress, anger, and depression, and even the meaning of the pain. Patients’ understanding of their condition and their expectancies for relief, their self-concept, their self-efficacy, and the medico-legal system impacts their perceptions and reports of pain (Tumlin, 2001). By addressing the domains that affect the “gates” through cognitive behavioral interventions, patients’ perceptions and experience of pain may change significantly. Patients may experience unchanged pain intensity levels, in fact, but report reduced life interference, depression, disability, and fear.

Depression

Psychological factors have a direct impact on chronic pain patients’ quality of life and disability (Turk & Okifuji, 2002). Epidemiological studies have identified specific psychological factors that are now recommended to be an important aspect in the experience and treatment of
chronic pain. Depression is one of these primary factors as it interferes with a person’s ability to cope with pain, and negatively impacts motivation and self-efficacy (Gatchel, et al., 2007). There is a great deal of overlap between the somatic and cognitive symptoms of depression and chronic pain (and indeed, many chronic health conditions) including attention and concentration impairment, weight changes, psychomotor agitation or retardation, sleep problems, changes in appetite, fatigue, and difficulty functioning. Changes in occupational and familial roles can lead to feelings of worthlessness, helplessness, and guilt.

Depression is so prevalent within chronic pain populations, that a meta-analysis of 83 studies (Fishbain, Cutler, Rosomoff H., & Rosomoff R., 1997) examined whether depression is an antecedent or consequence of chronic pain. The authors found 5 common hypotheses: 1) depression precedes the development of pain (“antecedent”); 2) depression follows the development of chronic pain (“consequence”); 3) some patients have a genetic predisposition to depression, and within the context of chronic pain, become depressed (“scar hypothesis”); 4) either antecedent or concomitant psychological mediators such as life interference or poor self-efficacy mediate a relationship between pain and depression (“cognitive behavioral mediation model”); and 5) depression and pain share a common pathogenetic / neurochemical mechanism. The results of the study did not present a clear answer, but there was far greater evidence for the consequence and scar hypotheses, particularly as some studies were able to specify the direction of the effect.

Pain Related Anxiety

Several studies have demonstrated a relationship between pain-related anxiety and pain-related impairment. The construct of pain anxiety is comprised of racing thoughts and impaired concentration related to pain, fearful cognitions and negative expectancies due to pain, overt
harm-avoidance behaviors intended to minimize pain, and physiological responses to pain (McCracken & Gross, 1992). Pain-related catastrophizing is a primary element of this domain, and is defined as a “negative cognitive-affective response in pain…characterized by the tendency to magnify and predict the worst concerning a particular experience of pain” (Sharpe, 2012, p. 951). Catastrophizing has been found to play a significant role in the development and maintenance of chronic pain, and seems to predict a number of treatment outcomes including depression, disability, return to work, and healthcare utilization (Sharpe; Turk & Okifuji, 2002). Changes in pain related anxiety are highly associated with, and may be more predictive of, positive treatment outcomes independent of physical capacity performance (McCracken & Gross).

Pain-related anxiety is also characterized by a fear of movement (kinesiophobia) and avoidance. Fear avoidance is primarily manifested in catastrophic thoughts related to pain and future possible injury, in addition to over-awareness of physical sensations and symptoms (Kerns et al., 2011). One study evaluating pain patients, found that those who reported greater pain-related fear-avoidance (as measured by the Pain Anxiety Symptoms Scale) over-predicted the intensity of pain related to an unfamiliar movement while those who reported less pain related anxiety, under predicted pain; further, greater pain anxiety was related to less range of motion overall regardless of actual pain levels reported (McCracken & Gross, 1993). Results also indicated that patients with greater levels of cognitive anxiety related to pain were far more likely to report less ability to control or decrease their pain, and far less likely to demonstrate adequate pain coping skills.
Self-Efficacy

The construct of self-efficacy, defined as the belief in one’s ability to perform certain required behaviors in a specific situation, has become an important factor in chronic pain management. Self-efficacy theory (Bandura, 1977) proposed that if a person has enough motivation to perform a behavior, then it depends on the person’s self-efficacy beliefs whether that behavior is initiated, how much effort is expended, and the degree to which effort is expended and maintained when the behavior is challenged or blocked. Patients with low self-efficacy are more likely to give up or return to old behaviors when challenged that those with high self efficacy. In the context of chronic pain management and functional restoration, the foundational aspects of coping and self-management behaviors are mediated by a patient’s self-efficacy (Turk & Okifuji, 2002). Numerous studies (as cited by Turk & Okifuji), have shown that self-efficacy is strongly related to pain control, healthy emotional functioning, perceived disability, impairment, physical task levels, increase in exercise, medication use, treatment outcomes, and return to work.

The role of self-efficacy in chronic pain cannot be understated. Self-efficacy as a focus of chronic pain treatment is important because so much of treatment is related to ongoing “self-management” of pain. Pain management patients are expected to make significant behavioral and lifestyle changes including reducing stress, increasing physical activity, improving pacing, improving nutrition, and increasing engagement in valued activities despite ongoing nociception. Pain-related self-efficacy beliefs have been shown to be strong predictors of pain severity, interference, and general activity, accounting for up to 45% of the variance in one treatment outcome regression model (Strong, Westbury, Smith, McKenzie, & Ryan, 2002).
Perceived Disability

The American Medical Association defines disability as a change in a person’s capacity to meet social, familial, occupational, and personal demands because impairment resulting from loss of function (as cited in Schoefferman, 2006). Psychosocial factors including perception of disability, inadequate social support, increased psychological distress, job dissatisfaction, workers’ compensation, and occurrence of pain behaviors appear to be more significant predictors of long-term chronic disability than severity of injury or job description (Turk & Okifuji, 2002). Within chronic pain populations, the perception of disability can be the logical outcome resulting from months or years of severe pain, depression, fear, anxiety, and a perceived lack of control over one’s life and ability to manage. Furthermore, a significant diminishment in functionality accompanies years or months of inactivity, including cardiorespiratory deconditioning, decreased muscle strength, and decreased flexibility (Schoefferman). Perceived disability is exacerbated by avoidance behaviors and catastrophic cognitions related to fear of increased pain related to activity, a misapprehension that activity will result in further injury, and the perception that severity of pain is a direct reflection of severity of injury. Importantly, patients who are able reduce depression, catastrophization, fear of movement, and improve self-efficacy in treatment have better outcomes, uniquely predicting improvements in disability (Bergbom, Boersma, & Linton, 2012).

Treatment of Chronic Pain

Biomedical treatment

“Successful treatment of chronic pain produces a patient who describes the pain in mild terms, engages in productive and satisfying activity on a daily basis, is free of excess emotional suffering, and uses medical resources at a prudent level” (McCracken & Turk, 2002).
One can see from the previously described psychosocial sequelae why traditional biomedical treatment might be insufficient to treat many chronic pain patients, with no individual medication, procedure, or treatment able to address the multiple dimensions, symptoms, and consequences of the condition (Gatchel & Okifuji, 2006). Traditional medical interventions for chronic pain focus on identification of the specific cause underlying a symptom, followed by treatment to surgically remove the pathology, or block the pain with medication or an implantable device. Specific single modality interventions include ablative techniques, acupuncture, nerve blocks, joint blocks, epidural steroid injection, intrathecal non-opioid injections, and botulinum toxin injections; electrical nerve stimulation interventions include subcutaneous peripheral nerve stimulation, spinal cord stimulation, and transcutaneous electrical nerve stimulation (TENS); minimally invasive spinal procedures include vertebroplasty, kyphoplasty, and percutaneous disc decompression (American Society of Anesthesiologists, 2010). These solutions address the needs of a significant proportion of the chronic pain population. However they can often prove to be ineffective if not injurious, such as in failed back surgery syndrome, and most of the time they are extremely expensive (Gatchel & Okifuji). There are about 300,000 to 400,000 spine surgeries performed each year, with success rates (defined as a reduction in pain and medication use and improvement in functional ability) measuring from 50-90% depending on the type of surgery (Gatchel, 2001). This approach implies there is a cure for the pain, and patients will often agree to multiple treatments, surgeries, and medications in search of this (Gatchel & Okifuji). Often the “allure of the cure” motivates a client to pursue one failed surgery or injection after another rather than focusing on acceptance and rehabilitation. (Clarke & Iphofen, 2007; DeGood & Tait, 2001; Geisser, 2006).
Unfortunately, patients often embrace the biomedical model of pain and pursue treatment determined to prove the validity of their chronic pain and the extent of their suffering, rather than exploring untried treatment options, and this fundamental misunderstanding of chronic pain management often leads to noncompliance or failure (DeGood & Tait, 2001). Chronic pain patients are often more likely than controls to attribute the degree of pain experienced to physical rather than psychological mechanisms (Allcock, Elkan, & Williams, 2007). Self-management strategies are often regarded with utter disbelief and skepticism, and treatment options such as psychotherapy, biofeedback, and relaxation training appear to imply that the pain is “all in their head,” and therefore not real (Clarke & Iphofen, 2007).

Lastly, traditional biomedical treatment models seek to match an intervention to the medical diagnosis. While this is effective when the symptoms have a known etiology, it is often not appropriate for diagnoses whose cause is unknown which is frequently the case with chronic pain (Turk & Okifuji, 1998). For instance, Waddell asserted that only 15% of patients with low back pain are given a specific medical diagnosis (as cited in Wittink, Nicholas, Kralik, & Verbunt, 2008). However, even when a diagnosis can be made, there is often a sharp discrepancy between the severity of the objective findings and the level of patient disability.

Opioids are a standard treatment option in the management of pain (Tunks, Weir, & Crook, 2008), though the side effects, the potential for abuse, and the complexity in managing tolerance of the analgesic effect makes them a less-than-optimal choice for managing chronic pain (Gatchel et al., 2007). Patients report that opioids lose their effectiveness over time (due to habituation), and require increasingly large doses; with these increasing doses, side-effects such as cognitive impairment and gastro-intestinal issues worsen. The possibility of pain-killer abuse
and dependence cannot be discounted, particularly in patients that are not followed closely by pain management specialists.

Other options for pharmacological management for chronic pain include anti-depressants, anticonvulsants, benzodiazepines, NMDA receptor antagonists, nonsteroidal antiinflammatory drugs (NSAIDs), skeletal muscle relaxants, and topical agents, all of which have also been shown to be helpful in treating several chronic pain conditions, including migraines, fibromyalgia, and neuropathic pain (American Society of Anesthesiologists, 2010). Importantly, however, one qualitative study with chronic pain patients revealed that many patients do not consider pain medications to be treatment but rather a way of being “fobbed off” (Allcock et al., 2007). To that end, frequently patients are simply provided ongoing medication rather than specific treatment for chronic pain.

Due to its nature, chronic pain is best assessed and treated within the broader context of illness, rather than as a disease (though there are efforts to have chronic pain considered a disease in its own right). The distinction between disease and illness lies in the difference between an objective anatomical and physical pathology (disease), while illness encapsulates the subjective experience being sick and how patients and families respond to and cope with symptoms (Gatchel et al., 2007). Thus, like other chronic health conditions, the focus of treatment shifts to symptom management rather than a search for a cure (Main & Spanswick, 2000).

*Chronic Pain Management*

Chronic pain management diverges from the biomedical focus on underlying diagnosis to the impact of the illness on activities of daily living, and strives to identify the behavioral factors that may be operating to aggravate and maintain pain. As standard medical care often provided
inadequate treatment for chronic pain, psychology researchers and clinicians increasingly
focused on cognitive-behavioral assessment and intervention, concentrating on rehabilitation
and behavior-change rather than a cure (McCracken & Turk, 2002). Chronic pain management
goals are a function of the numerous stakeholders involved, and include everything from return
to work and decreased healthcare utilization, to pain relief and improved daily functioning
(Gatchel & Okifuji, 2006). The psychosocial targets of treatment address the multiple domains
of a patient’s life and can include medication, functionality, depression, fear-avoidance, anxiety,
anger, disordered sleep, isolation, disrupted familial and interpersonal relationships, substance
abuse, and other comorbid conditions (Adams, Poole, & Richardson, 2006; Fillingim, 1997;
McCracken & Gross, 2002; Kerns et al., 2011; Simon & Folen, 2001).

Comprehensive assessments gather information about the impact that chronic pain has
had upon the patient’s activities of daily living, hobbies, work, and family relationships. The
implication is that these various areas have been impacted by chronic pain, and that to adequately
self-manage the condition, patients will need to develop ways to make improvements in all these
areas. By gathering valid and reliable measurements of the patient’s level of disability, the daily
amount of physical activity, and the ways that “pain behaviors” (Fordyce, 1977) and
psychopathology are functioning as barrier to recovery, these psychological and behavioral
factors serve as the focus for treatment in order to decrease the impact that pain has upon a
person’s ability to function. (Wittink et al., 2008). These assessments primarily use self-report
instruments that evaluate pain-related psychological status, social and environmental stressors,
pain-related disability, fear of pain, readiness to adopt self-management strategies, belief in
ability to manage pain, types of coping skills, and pain related attitudes. (Bradley & McKendree-
Smith, 2001).
Physical evaluation of chronic pain often includes a quantified assessment of the functional capacity of affected areas such as the lumbar spine (Polatin & Mayer, 2001). Measures such as range of motion, trunk strength, lift/carry capacity, and cardiovascular endurance introduce a degree of objectivity into chronic pain assessment, and are a useful way to measure changes from baseline at the beginning of rehabilitation. These measures are also commonly used to assess for disability compensation because of the self-report assessment limitations that are characteristic of chronic pain.

The physical and medical modalities of chronic pain management include medication management, exercise, heat, cold, massage, transcutaneous electrical nerve stimulations (TENS), and assistive devices. Furthermore, chronic pain patients are often characterized by patterns of pain coping that have emphasized an overly sedentary and avoidant lifestyle. This is addressed by the activity and physical therapy aspects of treatment as these patients are often very de-conditioned and weak; this emphasis on activity and reinforcement for adoption of healthy behaviors has been shown to be predictive of reduced disability and improved emotional functioning (Keefe, Williams, & Smith, 2001). There is limited but positive evidence that physical modalities are effective at managing chronic pain, with the greatest support found for therapeutic exercise (Rakel & Barr, 2003). Not surprisingly, certain traits may decrease effectiveness of physical modalities, including obesity, depression, anxiety, and narcotic use, so they may best be incorporated into comprehensive programs that can address these issues. However, Rakel and Barr note that most studies published in medical or physical therapy journals primarily focus on physical modalities as a monotherapy rather than as an adjunct to comprehensive treatment, so little is known about their role in MCPM.
**Psychological treatment of chronic pain**

While Gate control and Neuromatrix theory theories provide insight into the physical and emotional structures of the biopsychosocial model of pain, much of contemporary understanding of pain’s psychosocial aspects has been built upon behavioral conceptions of chronic pain and illness (Fordyce, 1976; Patterson, 2005). An important development in the evolution of chronic pain treatment came when Fordyce proposed that environmental contingencies and rewards contributed to the maintenance of pain and pain behaviors (overt signs of pain and suffering), while still emphasizing the true suffering experienced by patients. After Gate-control theory, this was the first major advancement in the psychological treatment of chronic pain and it focused on identifying and targeting operant conditioning factors that served to maintain pain behaviors such as excessive dependence on bed rest, overly solicitous family members, over reliance on medications, and an overall lack of activity. The rationale for this approach recognized that pain behaviors could transition over time from physical/biomechanical reinforcement to functional/environmental and by reinforcing “well behaviors,” patients could begin to regain their independence and functionality. Additionally, sufficient physical “evidence” for pain did not negate the likelihood that psychosocial factors mediated the experience of disability associated with pain (Vlaeyen & Morley, 2005). Fordyce described pain behavior as the overt behaviors that people demonstrate when they are suffering, noting that responses to suffering necessarily involved the interpretive function of the brain. He reasoned that reactions to nociception were filtered through expectancies, previous experiences, and anticipated consequences (Fordyce, 1988). Rather than a simple reflexive response, pain behaviors “occur as the complex end-product of the synthesis of signals from various sensory modalities to the central nervous system with other information in the brain” (Fordyce, 1988, p. 279).
Thus, pain behaviors, though indicative of suffering, reveal little about the actual degree of nociception. Fordyce (1976) identified that the acquisition of these behaviors was subject to the same operant processes as other learned behaviors, and that by controlling rewards and contingencies, treatment could perhaps alleviate significant levels of suffering and disability (Patterson, 2005). These contingencies often consist of the unpleasant activities that a patient is able to avoid, such as household chores, or rewards such as disability payments, or solicitous attention from a spouse (Patterson). By identifying environmental and interpersonal reinforcers, an rich area for treatment was developed that emphasized behavioral treatments focused initially on the identification and modification of environmental contingencies that might be operating to maintain pain behaviors.

Patients’ beliefs about their pain have also been shown to have a significant impact upon treatment outcomes, particularly when a patient is focused on finding an organic cause to their pain or is intent on curing the condition rather than managing it (Allcock et al., 2007). Cognitive factors including a perception of pain as uncontrollable, mysterious, and enduring or a perception of pain as necessarily creating disability have been associated with increased depression and disability, and poorer treatment compliance and outcomes. (Allcock et al.; Turk & Okifuji, 1996). One study examining mediators of change in CBT for chronic pain found that pre- to post-treatment changes in patient pain-related beliefs, including reduced catastrophizing, increased perceived ability to control pain, and decreased belief that one is disabled by pain were significantly related to reduced pain intensity and activity interference (Turner, Holtzman, & Mancl, 2007).

Thus, cognitive-behavioral therapy (CBT) provides the crux of most psychological interventions with chronic pain. The four general types of psychological intervention that have
received the most research support are self-regulatory, behavioral, cognitive-behavioral, and
acceptance and commitment therapies (Kerns et al., 2011). Self-regulatory approaches are
focused on increasing a patient’s sense of control over physical and psychological experiences.
The primary interventions in this approach include biofeedback, relaxation training, clinical
hypnosis, and mindfulness (Kabat-Zinn, 1982). Biofeedback studies have shown it to be an
effective intervention for chronic headache, with clinical effects lasting for over a year past
treatment (Kerns et al.; Nestoriuc et al. 2008). These lasting effects are possibly related to the
emphasis placed on the patients learning to voluntarily control physiological processes.

Relaxation training also focuses on patients learning to identify states of tension and
systematically reduce perception of pain and physiological arousal through methods such as deep
breathing, progressive muscle relaxation, or visualization (Kerns et al., 2011). There is a
significant psychoeducation component to relaxation training so that patients can begin to
understand the link between autonomic system arousal and emotional/physiological states.
Studies have shown it to be effective for musculoskeletal pain, headache, and back pain (Kabat-
Zinn, 1982; Kerns et al.; 1991; Strong et al. 1989). Mindfulness meditation is gaining wide
acceptance as a treatment for refractory chronic pain, particularly due to the efforts of Jon
Kabat-Zinn, whose work has shown this to be effective in reducing pain intensity, increasing
physical activity, and increasing adaptive emotional functioning.

Behavioral approaches, as previously discussed, focus primarily on pain behaviors and
their reinforcers. These approaches include operant behavioral therapy interventions for family
and friends who often unknowingly reinforce the verbal or resting behaviors that contribute to
disuse syndrome. Reviews of these operant behavioral interventions show increased activity
levels, decreased disability, and improved quality of life above and beyond that accomplished
through more comprehensive treatment (Kerns et al., 2011). Exposure behavioral techniques have also been directed towards the fear of movement and avoidant behaviors so commonly seen in patients, and which have shown to be predictive of future disability (Asenlof, 2010; Crombez, Vlaeyen, Heuts, & Lysens, 1999; Vlaeyen, 2000). In vivo exposure has been shown to have a significant effect on fear avoidance behaviors, helping patients engage in physical activities while decreasing their fears of further injury (Woods & Asmundson, 2008).

The three primary elements of CBT for pain is psychoeducation to help the patient better understand the role that cognitions and behavior play in the pain experience, the introduction of active coping skills training such as progressive muscle relaxation, pacing, and distraction techniques, and cognitive restructuring to identify and challenge overly negativistic or catastrophic thoughts related to pain (Kerns et al., 2011). Cognitive treatment emphasizes the role that thoughts and beliefs take in attending to physical sensations and emotional and behavioral responses (Vlaeyen & Morley, 2005). Targeted cognitions such as catastrophizing, low pain self-efficacy, and external locus of control have been identified as mediators of treatment outcomes. Lastly, CBT programs emphasize the role that autonomic arousal and muscle tension play in the etiology and maintenance of pain. Incorporating biofeedback and relaxation skills (including mindfulness meditation) is often a key element of MCPM programs (Vlaeyen & Morley).

Common components of both behavioral and CBT treatments include promotion of a self-management perspective; relaxation skills training; cognitive restructuring; behavioral activation including pacing strategies; problem-solving skills training; attentional control strategies; communication skills training; habit reversal; and maintenance and relapse prevention
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(McCracken & Turk, 2002). Cognitive-behavioral intervention has become a standard element for MCPM treatment (Kerns et al., 2011).

Lastly, Acceptance and Commitment Therapy (ACT) (Hayes, Strosahl, & Wilson, 1999) is increasingly used in psychological intervention work with chronic pain. ACT differs from CBT in that rather than focusing on identifying, challenging, and changing their thoughts, patients are taught to observe and experience their thoughts and feelings as they are, without judgment or trying to change them. Some recent studies have indicated that psychological acceptance and flexibility are associated with improved treatment outcomes when compared with coping skills training (Clarke & Iphofen, 2007; McCracken, 2004). Acceptance of chronic pain, which is comprised of both a “willingness” to experience pain and to participate in valued activities in spite of ongoing pain, is associated with significant reductions in patient emotional suffering, improved functioning, and reduced healthcare use (Kerns et al., 2011). Recent studies indicate that pain acceptance (Vowles, McNeil, Gross, McDaniel, Mouse, Bates, Gallimore et al., 2007) and psychological flexibility (McCracken, 2008) have begun to gain traction, with results showing more positive outcome and predictive power than previous treatments focusing on coping skills training or cognitive restructuring.

*Functional restoration*

Rehabilitation of physical and psychological functioning is considered an vital element of chronic pain management. A significant aspect of chronic pain is often a dramatic decrease in activity and physical deconditioning. As previously described, this is mediated by fear-avoidance behaviors and catastrophizing cognitions that focus on the belief that activity will cause more pain, the misunderstanding that the pain associated with activity is an indicator of further injury, and the belief that the severity of pain is a direct reflection of the underlying pathology
(Schoefferman, 2006). Functional restoration has several components including education regarding the nature of chronic pain and targeted towards mistaken beliefs about damage and pathology; exercise targeting increased strength, endurance, and flexibility; and progressive exposure to painful activities so that patients increase their self-efficacy and learn that it safe to engage in valued actions despite pain. Importantly, functional restoration targets a person’s ability to engage in functional behaviors (sitting endurance, standing endurance, lift capacity, carrying capacity, overhead reach flexibility, range of motion flexibility, etc.) with goals that are quota based rather than pain based, and regular reinforcement of progress. Studies have indicated that this approach is effective for helping patients increase self-efficacy to manage their pain, begin to understand the nature of chronic pain (“hurt does not equal harm”), reduce perceptions of disability, and increase return to work (Shoefferman, 2002; Turk & Okifuji, 2002).

*Multidisciplinary chronic pain management*

These different modalities are now often integrated into MCPM programs that can consist of physiatry and other medical oversight, cognitive-behavioral therapies, medication management, physical and occupational therapy, functional restoration, and vocational rehabilitation (Adams, Poole, & Richardson, 2006; Fillingim, 1997; Kerns et al., 2011; Simon & Folen, 2001). Gate control theory (Melzack & Wall, 1965), Fordyce’s behavioral conceptualization of pain (1973), and the development of the biopsychosocial model (Engel, 1977) sparked decades of intervention research that provided the foundation of comprehensive pain management, and MCPM programs (also referred to in the literature as “comprehensive” or “interdisciplinary” treatments) began to flourish during the 1980s (Gatchel & Okifuji, 2006; Kerns et al., 2011).
Programs are typically time-limited, ranging from 20 days to 8 months. Shorter programs tend to be more intensive, some meeting for 8 hours per day, 5 days per week. These are active programs, usually based on the biopsychosocial model of pain and a cognitive-behavioral approach to care. MCPM programs are typically comprised of professionals from multiple disciplines and may include physicians, neurologists, physiatrists, pharmacists, clinical psychologists, physical therapists, occupational therapists, nurses, and vocational counselors all of whom assess different aspects of the patient’s condition and work together to develop an integrated treatment plan addressing the varying dimensions (Simon & Folen, 2001).

The underlying assumption of MCPM programs is that patients will adopt an internal locus of control and learn psychological and functional pain self-management skills, so that they are able to manage their pain and improve their functioning over a lifetime and decrease their reliance on external sources of pain control (Bruehl, 2006). A significant feature of these treatments is the emphasis on reducing sedentary behavior and increasing physical activity. However, comprehensive treatment not only focuses on functional improvements, but in helping patients reduce their suffering and develop the ability to self-manage their pain (Gatchel & Okifuji, 2006.)

Psychoeducation plays a prominent role in these programs as studies have shown that patients need to increase their understanding of the different chronic pain models, so that they can understand that the absence of identifiable pathology does not indicate the pain is psychogenic or imaginary; they also need to understand the role of “pain management,” as their thoughts and beliefs about “adequate” pain treatment often present a barrier to accepting the rationale and engaging with the strategies that form the foundation of MCPM (Alcock et al., 2007). Patients’ understanding and acceptance of the pain-management rationale and their own
self-efficacy are important aspects to treatment success. If patients believe they are not capable of controlling their symptoms, then they will likely put little effort into learning and using self-management practices (Clarke & Iphofen, 2007; Turk & Okifuji, 2002).

Evidence from meta analyses indicate these programs are effective at improving the physical and psychological functioning of its participants (Flor, Fydich, & Turk, 1992; Hoffman et al., 2007; Morley et al., 1999; Vlaeyen & Morley, 2005. Findings show) show CBT and MCPM interventions contribute to significant improvement in pain-intensity ratings, reduced pain behaviors, reduced disability, increased positive coping skills, and improved pain experience. Further, effect sizes calculated for behavioral or cognitive-behavioral treatment often exceed 0.50, indicating sizeable amounts of variance accounted for by the intervention (Gatchel & Okifuji, 2006; McCracken & Turk, 2002).

One of the first major meta-analyses of MCPM programs (Flor et al., 1992) concluded that MCPM programs were more effective than no treatment, wait-list control, and single-discipline therapies on outcomes such as healthcare utilization, medication use, activity levels, return to work, closure of disability claims, and improvements in emotional functioning. This meta-analysis reviewed studies that used an interdisciplinary approach to chronic pain treatment and included empirical data, published from 1960 to 1990. From an initial retrieval of over 300 studies, a total of 65 met criteria for inclusion with 3,089 patients included, ranging from 5 to 467 per study with an average of 61. They noted that significant majorities of the patients were characterized by high rates of unemployment, litigation and unemployment cases, multiple previous surgeries, and medication use. Behavioral and verbal-subjective measures of pain were the most widely used outcome measures and patients showed the greatest improvements on self-
report measures of pain. The authors also found that patients treated in MCPM were nearly twice as likely to return to work (68% v. 36%) than those untreated or within single mode.

In sum, Flor et al., (1992) found that treatment effect sizes were largest between treatment and no treatment, though these were not far more significant than those between treatment and waitlist / medication. The studies also indicated that both the within group and between group effect sizes were large and maintained at short- and long-term follow up. This study also found that at long-term follow-up, patients treated in MCPM centers were functioning better than 75% of samples treated with conventional, single modality treatments. The authors also found that several variables previously associated with poor outcome including chronicity, age, litigation and compensation in fact did not show significant correlations, though they cautioned that these findings were based on a small number of studies.

A more recent meta-analysis employed “state of the art meta-analytic techniques to derive indices of the efficacy” (p. 6) of randomized controlled clinical trials of psychological interventions within outpatient MCPM programs treating non-cancerous chronic low back pain (Hoffman et al., 2007). Data were extracted from 31 separate research studies that met the following inclusion criteria: adults with chronic, nonmalignant pain of at least three months duration; presence of a comparison or control group; random assignment to groups; presence of pain-related outcome measures; and presence of psychological interventions. Outcomes measured included pain intensity, quality of life, and physical and emotional functioning compared to control groups (wait list and treatment as usual), as suggested by recommendations made by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT; Dworkin et al., 2008). The following interventions were considered “psychological”: those characterized as psychological interventions by authors of published
studies and explicitly based upon behavioral, cognitive-behavioral, self-regulatory, or supportive approaches. For instance, a study was not considered to have an adequate psychological component if it provided physical therapy and medication management, whereas a treatment would that included physical therapy, relaxation training, and a pain management psychoeducation group.

The results of the meta-analysis indicated that MCPM programs were superior to active control conditions following treatment at reducing pain interference \( (p < .05, d = .36) \) but not pain intensity \( (p > .10) \). MCPM programs were also found to be superior to active controls at improving percentages of patients who returned to work at both follow up \( (p < .05, d = .36) \) and long-term \( (d = .53) \). MCPM programs were not found to be superior to active controls at follow up on measures of pain intensity or pain interference \( (p > .10) \). Overall, the authors noted that there was little support for comparative efficacy of psychological interventions compared with active controls with the exception of the superiority demonstrated by MCPM programs for behavioral outcomes including return to work when compared with single modality therapies. Furthermore, MCPM programs were found to be superior to other active treatment conditions at improving work related outcomes at both short and long term follow up (Hoffman et al., 2007). Notably, this results of this study also did not find substantive moderator effects, suggesting that patient and study characteristics did not impact treatment, though the authors noted insufficient data to examine factors such as race/ethnicity, education, pain duration, etc.

Critics of MCPM programs have pointed to the possibility that complex and intensive (and expensive) programs are being provided to patients that perhaps could benefit from simpler treatments (Linssen & Spinhoven, 1992). Furthermore, the therapy has not been standardized and differs across different programs (Bruehl, 2006), though others argue that standardized treatment...
fails to target specific interventions to specific patient symptoms (Gatchel et al., 2007). Bruehl (2006) points out that comprehensive treatment could entail any combination of medical, nursing, psychological, physical therapy, occupational therapy, vocational counseling, or recreation therapy, and that some sites are more comprehensive than others. Furthermore, MCPM programs typically treat patients that have a wide range of diagnoses and mechanisms/locations of pain with largely the same offering of interventions (Turk, 2005). Thus, many programs appear to operate under the premise that by providing multidisciplinary treatment (exercise, relaxation training, biofeedback, CBT, medication management, occupational therapy, nerve blocks, etc) to all patients, the appropriate interventions will address each patient’s individual needs. However, as shown, studies indicate a wide variance in treatment response to MCPM, thought to be related directly to chronic pain patient heterogeneity (Gatchel et al., 2007; Turk & Burwinkle, 2005).

Multidisciplinary/Interdisciplinary chronic pain management has been recognized as having the strongest evidence-basis for the treatment of chronic pain (IASP, 2012). However, the IASP reports that in 1999 it was estimated there were over 1000 interdisciplinary pain management programs in the United States, but that number has dropped dramatically to less than 150 as of 2011 (2012). It seems that MCPM is not available to the vast majority of chronic pain patients in the US due to cost and lack of funding. The wide variance in treatment response, plays an important role in insurer’s unwillingness to pay for MCPM (Turk & Burwinkle, 2005). If reliable predictors of treatment response are not identified, the expense of these programs will likely make them unavailable to most patients.
Mediators and Moderators of Chronic Pain Management

While there exist numerous behavioral, pharmacological, and surgical treatments for chronic pain, much of their ability to demonstrate long-lasting relief has not been substantial. Pain patients are a heterogeneous population, and early outcome studies noted that even patients with the same medical diagnosis and physical pathology responded very differently to treatment (Turk & Okifuji, 1998). The variability in outcome increased awareness of the important role that psychosocial factors likely played in treatment. From 1960-1980, many researchers attempted to identify social factors related to pain, including ethnic and culturally-based responses to pain, however these studies resulted in contradictory conclusions (Thorn et al., 2007). In 1982, Cairn, Mooney, and Crane (as cited in Turk & Okifuji, 1998), showed that variables other than medical diagnosis and physical pathology had a significant effect on treatment outcome, noting that initial medical diagnosis had limited prognostic value. In 1990, Zatzick and Dimsdale (as cited in Wade and Price, 2002) concluded that there was little evidence that ethnicity or culture played a role in response to pain.

These early studies seemed to indicate that emotional factors served as mediators and moderators in treatment, predicting potential treatment response, and often contributing to early termination from treatment (Coughlan, Ridout, Williams, & Richardson, 1995; McCracken & Gross, 1998). Therefore, a great deal of research has been directed towards the identification of patient-related psychosocial factors that can be determined at assessment and which may aid in predicting successful treatment outcomes including demographic factors (race, ethnicity, sex, age), worker’s compensation status (Gatchel et al., 2002), physical disability, and medication use (Burns et al., 1998). Scores of psychological constructs have been evaluated including personality, attitudes, coping skills, stage of change, self-efficacy, fear of pain, emotional
distress, and program expectancies (Kreitler, Carasso, & Kreitler, 1989; Turk & Okifuji, 2002). A thorough review of these is beyond the scope of this paper, however a few of these will be reviewed here.

**Pain Intensity and Treatment Outcomes**

Patients prioritize pain reduction, improved sleep, and increased ability to engage in activities of daily living (Allcock et al., 2007). Patients have reported a strong motivation to determine the cause of their pain, indicating that this would provide “hope for a cure.” As noted, patients have strong beliefs in biomedical models of pain (there “must” be a physical cause otherwise the pain is imaginary), thus indicating that psychoeducation regarding the complexities of chronic pain might be an important element of treatment (Clarke & Iphofen, 2007). Patients indicate a desire to make their pain more tolerable but equate this with pain reduction or pain relief. Some patients reported interest in reducing feelings of apathy and depression. Overall, having less pain or some pain-free times was ranked as the highest priority for pain patients and showed that physical improvements were a greater priority than emotional or social improvements.

Since pain is subjective, self-report has been found to be the most valid measure (Melzack & Katz, 2001) and there are no objective measures. Patients are usually asked to quantify their pain using a numerical rating scale (e.g., scale of 1-10). Though this practice is nearly ubiquitous, this requires patients to retrospectively quantify and average their pain across time. Studies report that patients greatly overestimate their pain when recalling previous levels (Turk & Melzack, 2001). Also with few objective descriptions of each pain “level,” there is little standardization or way to discriminate between points on the scale. Despite these limitations,
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pain intensity is the most common measure of pain, and is usually measured by these numerical scales, verbal rating scales, or visual analogue scales.

There is little evidence that there are even divisions or meaningful differences between pain ratings. A patient may experience a two-point reduction in pain from 5-3 to be far more meaningful and impactful than a reduction from 8-6, therefore it is often difficult to ascertain the clinical significance (or, more importantly, the significance to the participant) of changes in pain intensity ratings although this is a primary focus of treatment for many programs (Dworkin et al., 2002).

Depression and Treatment Outcomes

Dworkin, Richlin, Handlin, and Brand (1986), measured treatment outcome differences between depressed and non-depressed chronic pain patients. Patients were assessed on a Likert scale of 1-5 of no improvement to complete improvement, and outcomes were determined by patients’ pain intensity, level of activity, and medication use. The findings indicated that there were no demographic (sex, age, marital status, education level, or compensation status) differences between depressed and non-depressed patients. Depressed patients were more likely to be involved in current litigation and more likely to be unemployed at their initial assessment. The pain-related variables included severity of medical history, number of different modes of prior treatment, and duration of pain, and both depressed and non-depressed patients were very similar. Despite the similarity, the authors found that the two groups differed dramatically in their pain descriptions, with depressed patients far more likely to report higher pain intensity and constancy, while non-depressed patients were likely to report mild or moderate pain that was intermittent. The authors noted however that it was impossible to ascribe causality or direction to these findings. In evaluating response to treatment, it was found non-depressed patients
responded better if they were provided a greater number of treatment visit, did not receive time loss/disability, had not tried multiple types of prior therapy, and were limited to low back pain. A different pattern of treatment response predictors was found for depressed patients, and a who were more likely to benefit when they had been employed prior to treatment and had experienced a shorter duration from injury to treatment. Although there were different treatment response predictors for each set of patients, the groups themselves did not differ in their response to treatment. Further research focused on specific moderating or predictive variables that influenced treatment outcomes such as personality, cognitive traits, and coping skills. Much of this centered on identifying patient subgroups based on psychosocial traits.

*Patient Subgroups*

An early study identified seven variables believed to predict treatment outcomes in 70% of subjects in an inpatient chronic pain program (Maruta, Swanson, & Swenson, 1979). The seven variables were 1) level of pain intensity, 2) duration of pain, 3) number of pain related surgical procedures, 4) dependence on analgesics, 5) loss of work time, 6) MMPI score on hypochondriasis scale and 7) MMPI score on hysteria scale. Response to therapy was evaluated in terms of a) modification of attitude, b) reduction in pain-related medication, and c) improvement of physical functioning. Findings indicated that the ‘treatment failure’ group demonstrated significant differences from the ‘treatment success’ including longer duration of pain and loss of work time, greater number of previous pain related operations, pain levels of 7/10 or above, significantly higher levels of narcotic dependency, and higher elevations on the hypochondriasis and hysteria scales on the MMPI.

In an attempt to replicate these results with an outpatient population, Reich, Steward, Tupin, and Rosenblatt (1985) also added four psychosocial variables to improve the predictive
accuracy of the model. These variables were 1) disruption of close personal relationship within previous 24 months, 2) altered body image due to loss of a visible body part or pain related change in physical activities, 3) history of severe, acute illness or injury before the age of 15, and 4) a history of child abuse. The results found no differences in outcome for age, sex, or level of education between treatment responders and non-responders, while the variables that had been included from the original study were found to be poor predictors of response to therapy. The additional variables were found to add some degree of specificity, but the study was limited by the fact that only 6 patients responded positively to treatment.

Cipher and Clifford (2003) evaluated 66 patients with the McGill Pain Inventory, BDI, and the MBHI and then categorized as them as either “Amplifiers, Repressors, or Social Copers.” These patients underwent treatment at a MCPM program and were evaluated on reductions in pain, functional impairment, and depression levels. While all three groups reported reductions in pain and functional impairment, only “Amplifiers” and “Social Copers” reported reduced levels of emotional distress and depression levels. Thus, the clients who were categorized as “Repressors” appeared not to have responded to the CBT aspects of the program, and thus coping style appeared to differentiate who improved the most in treatment. The authors noted, however that Repressors began treatment with the lowest pre-existing levels of depression and anxiety, and thus might already have had adequately developed coping skills, and thus did not improve “as much.”

However, additional studies have failed to find this patient grouping effect, noting instead that patient subgroups’ response to treatment appeared similar despite significant baseline differences (Gatchel et al, 2007; van der Hulst, Vollenbroek-Hutten, Groothuis-Oudshoorn, & Hermens, 2008). This finding suggested that all patients appear to benefit equally from treatment
in a variety of domains; the authors also suggested that psychological disorders, disability, coping resources, and narcotic medication use would not have to interfere significantly with effective pain treatment “as long as they (were) appropriately managed during the treatment process” (p. 143). While Gatchel et al., did not define how these issues were appropriately managed, they noted that there were no significant differences across subgroups in intensity or type of treatment. Nevertheless, numerous additional studies have proceeded with the MPI-identified patient subgroups, with an eye toward treatment matching. Though subgroups have been reliably reconstructed in further studies, there remains little evidence of treatment matching efficacy or clinical prediction models (Turk, 2005; van der Hulst et al.). Because of this, a growing focus on treatment moderators sought to identify subpopulations differentially affected by treatment and for whom effect sizes may mislead clinical decision making (Kramer, Frank, & Kupfer, 2006).

Coping skills and patient subgroup research continued while other factors came under investigation. Many studies examined the role of a patient’s readiness to change (Gersh, Arnold, & Gibson, 2011; Strong, Westbury, Smith, McKenzie & Ryan, 2002). Built around Prochaska and DiClemente’s (1984) transtheoretical model of behavior change, researchers hoped to identify which patients were more likely to engage and respond to treatment, and perhaps identify which patients were at risk for premature termination. Strong et al. developed the Pain Stages of Change Questionnaire (PSOCQ) in an effort to determine a patient’s readiness to adopt a self-management approach to chronic pain. In a study with 107 chronic pain patients assessed with the PSOCQ, the psychometric properties of the instrument did not prove to be reliable, and the transtheoretical model did not appear to adequately fit with chronic pain patients. Most
patients appeared to be in the contemplation stage prior to treatment, and outcome was better predicted by a patient’s movement through the stages during treatment than at baseline.

Worker’s Compensation Status

An important “subgroup” of chronic pain patients are those who have been injured on the job and are currently receiving disability payments (“time loss”) while they are unable to work and await possible compensation for their injury. Individuals in a MCPM program receiving workers’ compensation reported greater pretreatment interference with daily activities, more intense pain, and greater disability, and demonstrated poor outcomes than non-workers’ compensation patients, particularly if they had a history of pain related surgery (Burns, Sherman, Devine, Mahoney & Pawl, 1995). The authors suggested that the relationship between workers’ compensation status and outcome appeared mediated by the extent to which they believed they would be able to return to their former jobs.

When treating worker’s compensation patients, it is important to consider that issues related to secondary gain are commonly seen as significant barriers to recovery (Gatchel, Adams, Polatin, & Kishino, 2002). In their study with 158 patients participating in a MCPM program, Turk & Okifuji (1996) found that patients who were able to attribute their pain to a specific trauma reported significantly higher levels of pain intensity, emotional distress, and disability than those whose pain had insidious onset or unknown origin, regardless of physical findings. This suggests the context of pain has an impact on its intensity and degree of life disruption. Furthermore, in a study examining the role of beliefs and expectancies in chronic pain treatment, patient groups that attributed the blame for their pain their employer rather than to “no one” or to “other” (according to the authors, no one blamed themselves), reported greater mood distress,
behavioral disturbance, showed poorer response to treatment, and less optimism for future improvement (DeGood & Kiernan, 1996).

However, disability behaviors that are thought to be reinforced by financial, vocational, and emotional reinforcers, do not apparently resolve themselves once these reinforcers are addressed (Gatchel et al., 2002). Some evidence indicates that these factors do not necessarily create a major barrier to treatment as long as they are addressed, while perceptions of conscious malingering related to secondary gain may unfairly label a patient as unmotivated and prevent appropriate recognition of the substantial secondary losses associated with chronic pain. The construct of secondary loss “acknowledges that a major loss can have long term, rippling effects that reach far beyond the initial event and that intensify the trauma” (Gatchel et al., 2002, p. 101). Secondary loss common to chronic pain includes the loss of physical functioning and independence, financial stability, family and social relationships, employment and family roles, self-esteem, self-concept, and general view of the world (Gatchel et al, 2002). An additional challenge in treating worker’s compensation populations is the difficulty in adequately addressing patients’ loss of major social networks and their source of emotional and financial support. As studied by Jahoda (as cited by Gatchel et al, 2002), job loss not only leads to the loss of income, but also to the loss of professional esteem, socialization opportunities, a sense of purpose, and a structured organization to one’s activities and daily living.

Methodological concerns in treatment outcome studies

It is important to acknowledge the problems the field has had in evaluating the treatment efficacy of chronic pain management programs. A primary problem has been the lack of consensus on the definition of outcome criteria (Dworkin, et al., 2008; Thorn et al., 2007; Turk et al., 1999). “Successful” outcomes are differently defined by different stakeholders. Whereas a
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A patient would perhaps consider reduction or elimination of pain to constitute treatment success, a third-party payer (worker’s compensation insurer) might consider this negligible if it is not accompanied by a return to work or elimination of disability (Turk et al., 1993). So too, even a reduction in pain can hold subjective significance, as a 3-point reduction in pain from 6-3/10 might hold far greater importance to a patient and their perceived disability than a 3-point drop from 9-6/10 (Dworkin, et al., 2008).

Treatment outcome studies are limited as it is not clear what components of MCPM account for patient improvement, and thus identifying treatment outcome predictors and moderators can be difficult as the mechanism of change is rarely known. Due to a lack of fidelity to any specified protocol and efforts to “individualize” treatment, meta-analyses of MCPM are hampered by the number of outcome measures, heterogeneity of pain patient characteristics, and varying criteria for success between studies. Meta-analysis authors (Flor et al., 1992; Linssen & Spinhoven, 1992; Morley et al., 1999) report they are limited by treatment studies that have not included means and standard deviations for their reported outcome measures, do not include appropriate control groups, or use treatment drop-outs (sometimes as high as 87%) or those who refused treatment as controls. Few studies included information about the patient sample or study design and analysis. Lastly, this study was limited to multidisciplinary treatment of chronic back pain, and so its findings were not considered generalizable to treatments for other types of pain (Flor et al., 1992).

Chronic pain is a multidimensional, subjective, internal experience, but objective measures considered to be markers of treatment-related improvement are usually limited to simple measures of physical functioning. Furthermore, these measures of physical activity used as an outcome measure during a MCPM program are likely not to reflect the level of activity
characteristic of the patient, and may not transfer to the patient’s “real life” following treatment, thus treatment “gains” are not always maintained (Turk & Okifuji, 1998). In a recent commentary in the journal Pain, the IASP noted that patient outcomes are often not objectively measured and documented as a part of ongoing care, and recommended that the following particular outcomes be assessed using valid measures: patient reported pain intensity, physical and mental functioning, pain interference with mood, sleep, and physical function, and patient satisfaction with care (Ashburn & Witkin, 2012). While outcome studies have shown that use of valid self-report instruments can be effective (Gatchel & Okifuji, 2006) there is wide variability within treatment response. Much of the variability has stemmed the fact that patient’s diagnoses, conditions, and symptoms vary widely across studies (Thorn et al., 2007).

Meta-analyses of outcome studies are hindered by the inconsistent intake measures and differing criteria for success across the various programs (McCracken & Turk, 2002; Vlaeyen & Morley, 2005). Outcome measures of pain treatment programs are typically a combination of performance-based measures (functional tolerances), global ratings by healthcare providers, external variables such as claim closure or return to work, reduction in pain behaviors, utilization of healthcare system, and self-report measures including emotional distress, pain related cognitions, activity levels, perceived disability, coping strategies, and medication use (Turk, Rudy, & Sorkin, 1993; Wittink et al., 2008). The lack of criteria for successful treatment is due to content differences in outcome, the variability between clinicians’ ratings of success versus patients’, vague definitions of outcome, outcomes that were reliant on extraneous, uncontrollable factors (e.g., return to work rates during a recession), and the stringency of definition of successful outcome. The lack of consensus and common measures makes comparisons of results between studies nearly impossible (Turk et al.) and studies are likely limited by data aggregated
from treatment responders with non-responders (McCracken & Turk, 2002; Vlaeyen & Morley, 2005). Meta-analytic studies have typically focused on pain related to musculoskeletal back pain rather than the range of conditions that are present in chronic pain management treatment seekers, thus the generalizability of results is also called into question.

Furthermore, many CBT programs and MCPM programs are “individualized” in order to target a patient’s particular needs. Thus, outcome studies do not often include enough information on the interventions make replication possible. A number of discrete interventions fall under the rubric of CBT and MCPM and there have been no specific techniques or therapeutic mechanisms identified which best predict patient response. There have been a wide variety outcome variables studied, and attempts at determining predictors of CBT and MCPM effectiveness have also been negatively impacted by inconsistent methodology, measures, and outcome variables across studies (McCracken & Turk, 2002; Thorn et al., 2007).

Some additional limitations commonly found chronic pain research literature includes the selective bias in the referral process, lack of randomization and control groups, unacknowledged barriers to treatment, high drop-out rates, non-adherence to treatment recommendations, and strong risk of relapse (Turk & Rudy, 1990). Lastly, the questionable reliability of the self-report measures as the lack of normative data for these measures makes interpretation of outcomes (determination of clinically significant change) difficult to assess (Wittink et al., 2008).

The IMMPACT conference recommended six outcome domains for pain researchers to focus upon during clinical trials: pain intensity, physical functioning, emotional functioning, participants’ own ratings of improvement and treatment satisfaction, symptoms and adverse events, and participant disposition (Dworkin et al., 2008). These recommendations attempt to capture domains important to the wide range of stakeholders involved with chronic pain
treatment in order to improve the clinical utility of findings and implement a common set of standards to improve comparison and meta-analysis studies (Hoffman et al., 2007; Thorn et al., 2007). Additional studies are now investigating treatment mechanisms within CBT and MCPM programs, focusing primarily on processes associated with CBT such as altering negative cognitions that contribute to maladaptive coping behaviors (Day, Thorn, & Burns, 2012).

Obesity and Chronic pain

Highly comorbid with chronic pain, obesity also dramatically affects health-related quality of life. Obesity and overweight are defined by the World Health Organization as “abnormal or excessive fat accumulation that presents a risk to health” (WHO, 2013, p.1). Body Mass Index (BMI) is the standard used to calculate obesity by the WHO and is commonly used in research to calculate obesity, as it is considered to provide the most “useful” population-level measure of overweight and obesity due to its uniformity for both sexes and for adults of all ages (WHO, 2013). BMI is the ratio of weight to height and is calculated as weight in kilograms divided by the square of height in meters. It should be noted that BMI is an imperfect measure of obesity. While it is highly correlated with percentage of body fat, it does not distinguish between fat and lean tissue, so an individual with a significant percentage of muscle mass can be categorized as obese by this measure (Flegal, Carroll, Ogden & Curtain, 2010). Nevertheless, the following weight classifications are determined by BMI: underweight, BMI < 18.5; average weight, BMI = 18.5 – 24.9; overweight, BMI = 25.0 – 29.9; obese (mild), BMI = 30.0 – 34.9; obese (moderate) BMI = 35.0 – 39.9; and obese (severe/morbid) BMI ≥ 40 (Fabricatore & Wadden, 2006). Based on BMI calculations, an individual standing 5’4” (64”) would be considered underweight if they weighed 107.8 pounds or less, would be considered a healthy weight from 107.8 – 145.4 pounds, would be considered overweight from 145.5 – 174.5 pounds,
would be considered obese from 174.6 – 203.6 pounds, moderately obese from 203.7 – 232.8 pounds, and finally, severely obese if over 232.9 lbs.

Obesity has been called a “national health crisis,” and is predicted to be the number one global health problem by the year 2025 (Vaidya, 2006). As of 2008, an estimated 68.0% of adult US residents were overweight or obese; the prevalence of obesity averaged 32.2% among adult men and 35.5% among adult women, and varied by age group and racial/ethnic group for both men and women (Flegal et al., 2010). In 2005, the International Obesity Task Force (as cited in Haslam & James, 2005) estimated that approximately 1.1 billion adults met criteria for being overweight, including 312 million meeting criteria for obesity. In 2009 – 2010, the age-adjusted mean BMI among US adults was 28.7 for men, and 28.7 for women, while the median BMI was 27.8 for men and 27.3 for women (Flegal, Carroll, Kit, & Ogden, 2012). Rates of obesity increase across the lifespan particularly during middle to older age, which has been associated with several psychosocial and behavioral variables including cohabitation (hypothesized to be related to increased meal portion sizes for women), repeated pregnancies, and the progressive decrease in physical activity (Haslam & James, 2005). Prevalence varies according to socioeconomic status (SES), and in developed countries such as the US, there is a far higher prevalence of obesity in lower SES groups (Vaidya, 2006). Changes in BMI across the US have mirrored changes in rates of obesity, and though these increased dramatically from 1980 – 1999, recent data suggest a leveling off of this trend (Flegal et al., 2012). The negative impacts of obesity are wide ranging and include financial and economic costs, public health consequences, and individual physical and psychosocial consequences. Furthermore, it appears that obesity interacts with other conditions such that it aggravates and exacerbates their severity, or possibly acts as a moderating factor in treatment.
From a financial perspective, the steep health-care costs related to the multitude of health conditions due to obesity are predicted to significantly increase an individual’s Medicare costs compared to normal weight individuals (Cai, Lubitz, Flegal, & Pamuk, 2010). Previous research by van Heuvel, Boshuizen, & Hildebrandt (as cited by Kyrolainen, Santtila, Nindl & Vasankari, 2010) has shown that physically active (and comparatively less likely to be obese) employees had significantly reduced absenteeism. Additional studies have shown that high BMI, poor muscle strength, and impaired endurance were risk factors for reduced productivity (Kyrolainen, Hjäkkinen, Kautianinen et al., 2008). Additionally, high BMI and smoking have been shown to be the strongest predictors of sickness-related absenteeism, particularly for medically-confirmed absence, in fact doubling the risk in male employees, and nearly doubling the risk for absenteeism in female employees (Laaksonen, Piha, Martikainen, Rahkonen, & Lahelma, 2009).

From a public health perspective, the New England Journal of Medicine noted that morbidity related to obesity increases so significantly, that overall life-span of the US population could potentially decline over the 21st century (Olshansky, Passaro, Hershow, Layden et al., 2005). Obesity is a significant public health concern associated with numerous health conditions including chronic pain, type 2 diabetes, heart disease, hypertension, metabolic syndrome, breast cancer, polycystic ovary syndrome, dyslipidemia, stroke, gout, gallstone disorder, osteoarthritis, and significant psychological disorders (Centers for Disease Control, 2011; Haslam & James, 2005). The respiratory effects of obesity are significant and have a strong effect on health related quality of life, and numerous studies indicate that obesity is highly correlated with reduced physical fitness and activity and increased sedentary lifestyles (Kyrolainen et al., 2010; Marcus, 2003). The greatest impairments associated with obesity are found in social functioning, general health status, physical functioning, vitality, and the role limitations caused by physical disability.
and limited mobility (Barofsky, Fontaine, & Cheskin, 1997). In addition to physical disability that impairs in activities of daily living, obesity has been linked to depression, anxiety, and psychological disability. Successful treatment of obesity has been shown to improve all these physical and psychological aspects of life (Marcus, 2003; Sullivan, Karlsson, Sjostrom, et al., 1993).

It can be difficult to discriminate between the social and pathophysiological consequences of obesity and weight gain when evaluating the relationship between psychological health and obesity. However, obesity has been well-established as having a negative impact on physical well-being, though results have inconsistently demonstrated its relationship to psychological disturbance (Doll, Petersen, & Stewart-Brown, 2000). Findings from the 2005 Behavioral Risk Factor Surveillance System, a large-scale study of US adults using ongoing, random-digit telephone survey of the U.S. adults, indicated that persons who were “dissatisfied/very dissatisfied” with their lives were 1.5 times more likely than those very satisfied to be obese, and 2.2 times more likely to be physically inactive (Strine, Chapman, Balluz, Moriatry, & Mokdad, 2008). Large epidemiological studies have found that two obese subgroups, women and the severely obese, are at increased risk of depression (Fabricatore & Wadden, 2006). While the prevalence of psychiatric comorbidity in community-based obese populations is similar to non-obese groups, there is a significant increase in psychiatric problems in obese patients seeking treatment.

There are no studies that show a specific pattern of personality traits associated with obesity (Davin & Taylor, 2009; Vaidya, 2006). However, it is noted that slimness is idealized in most developed societies so overweight individuals, particularly women, are often subject to discrimination and stigma, and may suffer from feelings of poor self-esteem, anxiety, and
depression in relation to their excess weight (Haslam & James, 2005), though these effects are found to be moderated by gender (Vaidya, 2006). Obesity increases the risk of a diagnosis of major depression in women by 37% while obese men have a 37% reduced risk of depression in comparison to healthy weight men. As noted, aspects of psychological distress are mediated by treatment seeking for obesity (bariatric surgery), as bariatric surgery candidates often have far greater rates of depression and anxiety than obese individuals not seeking treatment (Fabricatore & Wadden, 2006). The relationship between BMI and depression is likely mediated by the impact of obesity on health-related quality of life (Fabricatore & Wadden, 2006.)

While numerous epidemiological and clinical studies have noted the association between being overweight/obese and psychopathology in obesity treatment seeking populations, obesity has also been found to impact treatment outcomes in psychiatry research directed towards mood disorders. Several studies have noted that obesity is related to poorer outcome in the treatment of bipolar I disorder (Fagiolini, Kupfer, Houck, Novick, & Frank, 2003; Faitha, Calamaroa, Dolana, & Pietrobelli, 2004; Hainsworth, Davies, Khan, & Weisman, 2009) with findings indicating that obesity is associated with poorer response to antidepressants and mood-stabilizers. Fagiolini et al. (2003) evaluated the demographic and clinical characteristics and treatment outcomes of 175 patient with bipolar I disorder who were being treated for an acute affective episode and then monitored through a period of maintenance treatment. Results indicated that obese patients ($n = 62, 35.4\%$) experienced a greater number of lifetime affective episodes, presented with more severe and less-responsive to treatment index affective episodes, and were at greater risk to experience depressive recurrences. Specific results showed that in comparison to non-obese participants, obese participants were less educated, reported greater numbers of previous manic episodes, more previous depressive episodes, and higher baseline Hamilton depression scale
scores. Further, obese patients required increased treatment time in order to attain stable remission and showed a significantly shorter maintenance phase between acute affective episodes. Additionally, obese patients showed significantly greater numbers of depressive recurrences.

It appears that a similar response was found with unipolar depression studies. Results from the Munich Antidepressant Response Signature Study indicated that patients with high BMI (≥25) showed slower clinical response, less improvement in neuroendocrinological (plasma cortisol and adrenocorticotropin (ACTH)) levels, and less improvement in cognitive speed and attention than patients with normal BMI (<25) during antidepressant treatment (Kloiber, Ising, Reppermund, Horstmann, et al., 2007).

The rates of comorbidity between obesity and chronic pain are primarily gathered from community based studies and obesity research, as BMI appears rarely reported in chronic pain research. Findings from the community based University of Washington Twin-Registry showed that overweight and obese twins were significantly more likely to report low back pain, headache, fibromyalgia, abdominal pain, and chronic widespread pain more than normal-weight twins after adjusting for age, gender, and depression (Wright et al., 2010). Previous findings have also demonstrated that black patients with chronic pain are at higher risk for obesity and morbidity than white patients with chronic pain (Caldwell, Hart-Johnson, & Green, 2009).

In another study looking at the association between obesity and health-related quality of life, found that out of nearly 8,600 respondents, approximately 33% were overweight (n = 2682, 31%) and or obese (n = 921, 11%); and more than 55% of overweight and obese respondents suffered from at least one co-morbid chronic illness, most frequently back pain or joint pain (Doll et al., 2000).
The impact of chronic pain upon obesity patients is frequently studied though the corollary may not be true. It appears that obese persons with chronic pain are significantly more impaired across multiple domains than obese persons without pain (Barofsky, Fontaine & Cheskin, 1997; Caldwell et al., 2009; Doll et al., 2000; Hainsworth, et al., Marcus, 2004). In a sample of obese patients seeking university-based weight management, over one-half presented with chronic pain, including low back pain, arthritis, chronic headache, and chest pain (Barofsky et al., 1997). Even when controlling for BMI, these patients were significantly impaired in all areas of health-related quality of life, sociodemographic factors, and depression, indicating that pain played an important role beyond the limitations imposed by obesity.

In a community sample, participants reporting co-morbid obesity and chronic illness (particularly back and joint pain), reported particularly poor physical and emotional well being when compared with individuals that were normal weight/health, or who suffered from chronic illness or obesity independent of one another (Doll et al., 2000). While studies show that obesity itself is not a reliable, independent risk factor for emotional disturbance, (Wadden & Stunkard, 1987), the Doll et al. study provided additional evidence that obesity likely compounds the physical and emotional disability often associated with chronic pain. Obesity impairs walking ability and exacerbates pain when walking; further, difficulty with walking is an early predictor of pain related disability (Vincent et al., 2011). Obesity alone contributes to lower back pain due to lumbar weakness, poor quality skeletal muscle, poor balance, and strength deficits in back muscle; it is a prime contributor to joint pain and arthritis in the knees and hips (Haslam & James, 2005).

Obesity is also common in fibromyalgia patients and has been associated with greater pain sensitivity, reduced physical strength, reduced flexibility, and poorer sleep quality (Okifuji,
Donaldson, Barck, & Fine, 2010). Furthermore, a positive linear relationship has been found between BMI and levels of inflammatory markers such as interleukin-6 and C-reactive protein and levels of stress indicators such as cortisol and epinephrine in fibromyalgia patients, factors likely related to the poorer sleep quality and greater pain sensitivity (Okifuji, Bradshaw, & Olson, 2009). Okifuji, Bradshaw et al. also found that obese patients demonstrated reduced degrees of fitness as evidenced by shorter treadmill walking distance accompanied by higher maximum heart rate. The implication of this is that obesity may present a barrier to activating and physically-based therapies, which are common components of pain management.

Further, people with higher BMI appear to be more sensitive to chronic pain (Wood, Goodnight, Haig, and Nasari, 2011). In a study examining the relationship between obesity, hypertension, and chronic pain, Wood et al. found no significant correlation between pain rating and BMI as a continuous variable (though they reported that it “approached significance”). However, ANOVA results indicated a statistically significant difference in BMI for patients with low pain ratings (0-3/10) versus those with high pain ratings (7-10/10), with those reporting high pain ratings having significantly higher BMIs though the effect size was small \( F(2,172) = 3.63, p = .03, \eta^2 = 0.04 \). Obese patients in this study also reported a higher incidence of narcotic use for pain despite similar pain intensity ratings, which the authors interpreted as a possible lower tolerance for pain.
Obesity, Depression and Health-Related Quality of Life

Obesity is also believed to increase pain severity and disability also due to increased psychological distress. Further, obesity is strongly related to disability for engaging in activities for daily living and occupational functioning, which in turn diminishes quality of life. Marcus (2003) found that in a sample of chronic pain patients seeking treatment, obese patients were significantly more likely to endorse severe levels of depression, though there were no differences in anxiety between obese and non-obese patients. Further, pain severity was not related to weight, but increased weight was significantly associated with increased disability and impaired physical functioning. Based on these findings, the author suggested that obesity serves as a marker of disability and depression in patients seeking treatment for chronic pain, thus patients’ weights should be included in pain assessment as patients with obesity might benefit from a greater intensity of treatment, targeting both depressive symptoms and disability.

While the relationship between pain and obesity is typically attributed to the mechanical problems caused by obesity, studies suggest that obese persons with pain have significantly higher levels of pain catastrophization (Vincent et al., 2011). In a study evaluating the impact of obesity on physical therapy, fear of movement and pain catastrophizing was found to be far more severe in obese adults than non-obese adults with chronic low back pain, despite similar pain ratings and depression severity.

Barofsky et al. (1997) investigated the impact of pain in a population of obese patients on health related quality of life. In a sample of 312 patients seeking treatment for obesity at the Johns Hopkins Weight Management Clinic, 47.7% reported moderate to severe chronic pain at the start of treatment. There were no differences between obese patients with pain and obese patients without pain on demographic measures or levels of depression. However, they found
that obese persons reporting pain were significantly negatively impacted on all health related quality of life domains on the SF-36, including physical functioning, physical role, general health, vitality, social functioning, emotional roles, and mental health. Based on the lack of differences on demographic measures, the authors reported that chronic pain appeared to be associated with significant impairment in health-related quality of life independent of obesity, depression, or socio-demographic factors.

However, in a community-based study, BMI was significantly associated with health-status, but the effect varied according to whether the measure reflected well-being in a physical or emotional domain (Doll, Petersen, & Stewart-Brown, 2000). 9,000 adults were surveyed using the Multidimensional Short Form (SF-36), a self-report, 36-item, Likert scale or forced choice measure that evaluates health related quality of life in the following domains: Physical Functioning, Role Limitations due to Physical Functioning, Bodily Pain, General Health, Vitality, Social Functioning, Role Limitations due to Emotional Functioning, and Mental Health. Lower scores are indicative of reduced health-related quality of life. This measure has been shown to have excellent construct validity, high internal consistency, and high test-retest reliability (Yancy, Olsen, Westman, Bosworth, & Edelman, 2002). In this study, 31% \((n = 2,682)\) of respondents were overweight, and an additional 11% were either moderately obese \((10\%, n = 852)\) or severely obese \((1\%, n = 69)\). Increasing BMI was found to be linearly associated with reporting a long-standing illness and frequency of health service utilization. The association of increased BMI with pain also resulted in reduced scores in physical well-being independent of the number of chronic illnesses reported. Overweight and obesity were strongly associated with diminishing levels of physical and emotional well-being, though the effect was far more pronounced in measures of physical well-being. The authors stated that these result
supported previous findings that the consequences of obesity were found to be primarily physical rather than emotional.

Numerous studies have demonstrated the positive relationship between weight and depression in women, thought to be mediated by impairments in health-related quality of life (Fabricatore & Wadden, 2006). In their research looking at the relationship between obesity and health-related quality of life in men, Yancy et al. (2002) found an strong relationship between BMI and the physical domains of health-related quality of life. Of the 1,168 male participants in the study, 43% \( (n=507) \) were overweight, 25% \( (n=292) \) were obese, 8% \( (n=98) \) were moderately obese, and 3% \( (n=30) \) were morbidly obese. Overall, 79.3% were overweight or obese. After controlling for age, race, comorbid illness severity, depression, and physical activity, individuals with morbid obesity (BMI \( \geq 40 \)) scored significantly worse than average-weight participants on 5 of 10 subscales: Physical Functioning, Role-Physical, Bodily Pain, Vitality, and Physical component Summary. Even overweight individuals (BMI from 25 to 29.9) reported significantly greater problems with bodily pain compared with normal weight individuals.

\textit{Chronic Pain and Treatment for Obesity}

Chronic pain is considered one of the primary barriers to the diet and exercise components of obesity treatment (Mauro, Taylor, Wharton, & Sharma, 2008). In a qualitative study investigating the experience of patients with comorbid obesity and chronic pain, Janke and Kozak (2012) found that patients perceived depression as amplifying their comorbid somatic symptoms and interfering with treatment in numerous ways. Patients reported that hedonic hunger (eating for pleasure) and emotional/binge eating were triggered by physical pain and resulted in additional guilt and shame. Patients also reported making unhealthier food choices in
response to pain. Importantly, patients reported experiencing far lower self-efficacy for physical activity due to pain and far greater levels of disability.

Obesity and Treatment for Chronic Pain

One important study examined the effects of obesity on pain coping skills training (PCST) and lifestyle behavioral weight management (BWM) (Somers, et al., 2012). In this study, overweight or obese participants with osteoarthritis were randomly assigned to a six-month treatment program examining the differences between PCST + BWM, PCST-only, BWM only, or treatment as usual. Results indicated that patients who were provided PCST + BWM demonstrated significantly better outcomes in numerous domains including pain, disability, and self-efficacy. The authors noted that the emphasis in reducing catastrophizing and anxiety, and improving self-efficacy, as found in the combined condition, may have contributed to the better outcomes. The authors of this study recommended that overweight or obese patients with osteoarthritis be provided concurrent training in pain coping skills and weight management in order to provide greater long-term benefits.

Obesity and Cognitive-behavioral Treatment for Chronic Pain

The literature indicates that obese patients with chronic pain often experience greater levels of depression and impaired self-efficacy related to their weight, reduced health related quality of life, and greater perceived disability. With obesity’s impact on so many dimensions related to chronic pain, it seems unsurprising that a recent study evaluating cognitive-behavioral treatment of chronic pain patients found that obesity was a significant moderator of patients’ ability to benefit from treatment (Sellinger et al., 2010). The authors compared the treatment outcomes of obese and non-obese chronic pain patients and found that non-obese participants showed greater improvement following treatment than obese participants on measures of
disability \( (p<.05) \), physical aspects of quality of life \( (p<.01) \), and emotional functioning \( (p<.05) \).

It is unknown to what degree this treatment included a multidisciplinary component that would also emphasize physical conditioning, occupational therapy, and operant behavior shaping, but the entire treatment was delivered by psychologists rather than an integrated treatment team. It appeared that there were few differences found in emotional functioning post-treatment aside from depression scores, but this may be related to small sample size \( (N = 74) \) and a lack of statistical power.

Based on this review of the literature, one might hypothesize several possible reasons why obesity may function as an added emotional and physical stressor that negatively impacts MCPM. In particular, important psychosocial chronic-pain management outcomes related to pain intensity, emotional functioning, fear of movement, self-efficacy, and perceived disability have been shown to be also responsive to obesity, such that participants with these co-morbid conditions will present as more severe treatment cases and show impaired response to treatment. Additionally, an increase in exercise provides a multitude of benefits, both psychological and physical, and previously sedentary adults derive benefit from even modest exertion (Haslam & James, 2005). It is possible that the increase in physical activity that is a component of MCPM is highly associated with patient improvements in emotional and physical functioning. If a patient is unable to fully engage with this increased physical activity due to obesity, they may derive less benefit from the treatment overall.

As of 2013, there appear to be no published outcome studies evaluating the role obesity plays in treatment outcomes for MCPM programs. These programs have been shown to provide effective treatment for chronic pain, but response to treatment is impacted by a wide variety of variables, which have inconsistent interactions with treatment effects. While MCPM has been
shown to be as effective as biomedical interventions (Turk & Okifuji, 2006), it is important to know what factors impact treatment outcomes. With estimates of obesity ranging from 35-50%, particularly for chronic pain patients, and based upon the numerous ways in which chronic pain and obesity interact, it is worth considering that obesity may contribute to the variance found in treatment outcomes, thus playing a moderating role in showing what treatments work for whom. MCPM is considered an expensive treatment and there have been no published studies looking at the impact of obesity on a patient’s ability to benefit from MCPM. If there is an interaction between treatment and obesity, MCPM programs can begin to develop targeted treatments to this specific subset of patients and outcome studies can better control for this possibly influential patient characteristic.

Thus, the literature shows that obesity is linked with greater impairments in emotional functioning, a differential treatment response to anti-depressant medications, lower levels of physical activity, greater deconditioning, reduced response to physical therapy, an increased fear of movement, and greater levels of perceived disability. It may be expected that individuals with comorbid chronic pain and obesity would also be characterized by these impairments. Furthermore, chronic pain has been shown to act as a barrier to treatment for obesity, often functioning as a stressor that contributes to reduced physical activity and increased hedonic/emotional eating. While obesity is a well-known risk factor for chronic pain, and chronic pain has been shown to moderate obesity treatment outcomes, there has been little published research examining the role of obesity in chronic pain treatment. Although it has been demonstrated that obesity and chronic pain are often comorbid and have an interdependent relationship with one another, there is little research that has shown what role obesity plays in
comprehensive treatment for chronic pain. This study attempted to address this gap in the literature.
Hypotheses

The purpose of this study was to compare the treatment outcomes of obese versus non-obese participants in a 20-day chronic pain management program. By retrospectively examining two years of treatment records and comparing treatment outcomes of different participant groups, this study sought to provide insight into the demographics of overweight and obese patients within a chronic pain population, and examine the potential for differential emotional functioning and response to treatment. The following hypotheses were tested:

1. It was expected that weight status as measured by body mass index (BMI) would predict differences in response to treatment, such that participants with BMIs equal to or greater than 30 (obese) would have higher rates of treatment drop-out than participants with BMIs lower than 30.

2. It was expected that participants in a MCPM program would show statistically significant change (improvement) from pre-treatment (Time 1) to post-treatment (Time 2) on measures of pain intensity (Pain), depression (BDI-II), anxiety (PASS), self-efficacy (PSEQ) and perceived disability (PDQ).

3. It was expected that improvements on measures of pain, depression, anxiety, self-efficacy, and perceived disability would vary according to whether a participant was classified as Obese or Non-Obese.

4. It was expected that obese participants would report higher levels average pain intensity, in comparison to non-obese participants, as measured by numerical pain-rating scores (NRS).
5. It was expected that obese participants would report more severe depressive symptoms in comparison to non-obese participants, as measured by scores on the Beck Depression Inventory (BDI-II).

6. It was expected that obese participants would report greater pain-related fear and anxiety in comparison to non-obese participants, as measured by scores on the Pain Anxiety Symptom Scale (PASS).

7. It was expected that obese participants would report less ability to self-manage their pain in comparison to non-obese participants as measured by scores on the Pain Self-Efficacy Scale (PSEQ).

8. It was expected that obese participants would report greater levels of perceived disability than non-obese participants, as measured by scores on the Pain Disability Questionnaire (PDQ).

9. It was expected that weight status (obese versus non-obese) would predict reductions in pain intensity, such that the mean change in pain intensity, from pre to post-treatment, would be less for obese participants than non-obese participants.

10. It was expected that weight status would predict reductions in depressive symptoms, such that the mean change on the BDI-II, from pre to post-treatment, would be less for obese participants than non-obese participants.

11. It was expected that weight status would predict reductions in pain related fear and anxiety, such that the mean change on the PASS, from pre to post-treatment, would be less for obese participants than non-obese participants.
12. It was expected that weight status would predict improvements in ability to self manage pain, such that the mean change on the PSEQ, from pre to post-treatment, would be less for obese participants than non-obese participants.

13. It was expected that weight status would predict improvements in perceived disability, such that the mean change on the PDQ, from pre to post-treatment, would be less for obese participants than non-obese participants.
Method

Design

In order to evaluate whether obesity moderated MCPM treatment outcomes, this study conducted retrospective analyses of patient medical records and outcome data from a community based comprehensive pain-management clinic. The total data set was comprised of 221 participants who consecutively completed a 20-day pain-management program over a period of 2 years. Data consisted of demographic and health-related data, in addition to results from pre- and post-treatment assessments of pain intensity, depression, pain-related anxiety, perceived disability, and self-efficacy in managing pain. These assessment data were captured by 5 self-report measures provided at intake and discharge. At the time of treatment, there was no consideration of the participants’ BMI, nor was this information incorporated into treatment in any way. The retrospective data were divided into obese and non-obese groups using BMI for comparison, using specific criteria described below. The data were analyzed for both within-group change related to treatment and between-groups change in relation to weight status.

Participants

Participants were patients treated consecutively through the Progressive Rehabilitation Associates multidisciplinary chronic pain management program during the years 2009 to 2011. Patients were referred to treatment by their physicians or by their worker’s compensation systems. The primary goal of the program is to restore functional tolerances so that a patient may return to the workforce at an adequately assessed level; another goal is to evaluate the patient’s history and condition in order to rate them as stable (“Maximally Medically Improved”) or in need of further treatment. Upon treatment completion and stable rating, participants who were referred by their worker’s compensation system were usually provided a return-to-work rating,
and their worker’s compensation cases settled and closed. Upon closure of a worker’s compensation case, there is often a financial settlement and a patient’s “time-loss” (paid benefits) are concluded. The exclusion criteria for the program were limited to inability to pay, active psychosis, recent surgery requiring further recovery prior to rehabilitation, and an absolute refusal to enter the program. There was no information available regarding patients who were evaluated by the program but were not admitted or chose not to enroll.

The sample consisted of 221 individuals, the majority of whom were male \( n = 144, 65.15\% \) and Caucasian \( n = 187, 84.61\% \). One patient was African American, while 33 \( (15.2\%) \) patients were Latino; of these Latino participants, many were Spanish-speaking only (exact number and percentage unknown) and were accompanied at all times by an interpreter provided by the clinic. The mean age of the sample was 44.89 years \( (SD = 9.98) \), with a range of 20 – 81 years old. The majority of patients \( n = 210, 95.02\% \) were referred by worker’s compensation systems primarily within Oregon and Washington State. All patients had at least a 3-month history of pain unresponsive to medical treatment prior to entering the program, though the majority had experienced considerably longer injury-treatment intervals, with an average of 44 months \( (SD = 50.09) \) between injury and treatment intake. 43\% \( (n = 95) \) of the sample met criteria for obesity \( (BMI \geq 30) \), with a BMI range of 19 – 52, and a mean BMI of 30.07 \( (SD = 6.39) \).

The participants had varying types of mechanism of injury, including headache, musculoskeletal injury, osteoarthritis, neuropathic deterioration, and non-specific (i.e., fibromyalgia). The majority \( (47.5\%) \) were related to lumbar pain and dysfunction \( (n = 105) \). A significant portion of the participants had previously sustained severe injuries related to industrial accidents (e.g., run over by a tractor-trailer), and there were varying degrees of
disability, pain, and psychopathology as measured at intake. See Table 1 for more demographic information of the total sample.

**Table 1**

*Total Sample Demographic Characteristics*

<table>
<thead>
<tr>
<th>Demographic</th>
<th>N</th>
<th>Mean (SD) / %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>221</td>
<td>44.89 (9.98)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>144</td>
<td>65.15%</td>
</tr>
<tr>
<td>Female</td>
<td>77</td>
<td>34.84%</td>
</tr>
<tr>
<td>Race</td>
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<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>187</td>
<td>84.61%</td>
</tr>
<tr>
<td>Other</td>
<td>34</td>
<td>15.38%</td>
</tr>
<tr>
<td>Education</td>
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<td></td>
</tr>
<tr>
<td>&lt;High school</td>
<td>29</td>
<td>13.2%</td>
</tr>
<tr>
<td>High school Graduate/GED</td>
<td>79</td>
<td>35.7%</td>
</tr>
<tr>
<td>Some College / Certificate</td>
<td>88</td>
<td>39.8%</td>
</tr>
<tr>
<td>College graduate</td>
<td>25</td>
<td>11.3%</td>
</tr>
<tr>
<td>Referral Source</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worker's Compensation</td>
<td>210</td>
<td>95.02%</td>
</tr>
<tr>
<td>Private Clinician</td>
<td>11</td>
<td>4.08%</td>
</tr>
<tr>
<td>Type Employment at Injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heavy labor</td>
<td>81</td>
<td>36.65%</td>
</tr>
<tr>
<td>Low skilled labor</td>
<td>100</td>
<td>45.25%</td>
</tr>
<tr>
<td>Skilled professional</td>
<td>40</td>
<td>18.10%</td>
</tr>
<tr>
<td>Employment Status at Intake</td>
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<td></td>
</tr>
<tr>
<td>Time loss / Worker's compensation</td>
<td>160</td>
<td>72.40%</td>
</tr>
<tr>
<td>Unemployed / no income</td>
<td>17</td>
<td>7.69%</td>
</tr>
<tr>
<td>Employed</td>
<td>17</td>
<td>7.69%</td>
</tr>
<tr>
<td>Permanent Disability / Retired</td>
<td>27</td>
<td>12.22%</td>
</tr>
<tr>
<td>Pain duration (months)</td>
<td>221</td>
<td>44.09 (50.09)</td>
</tr>
<tr>
<td>Morphine equivalency (pretreatment)</td>
<td>153</td>
<td>67.53 (71.67)</td>
</tr>
<tr>
<td>Average pain intensity (pretreatment)</td>
<td>221</td>
<td>6.35 (1.47)</td>
</tr>
<tr>
<td>Location of Pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper Body</td>
<td>59</td>
<td>26.7%</td>
</tr>
<tr>
<td>Lower Extremity</td>
<td>19</td>
<td>8.6%</td>
</tr>
<tr>
<td>Lumbar</td>
<td>106</td>
<td>48.0%</td>
</tr>
<tr>
<td>Multiple</td>
<td>37</td>
<td>16.7%</td>
</tr>
</tbody>
</table>
Obesity was assessed according to the World Health Organization’s standard BMI ≥ 30. Of the 221 participants, 96 (45%) met criteria for obesity with a BMI ≥ 30, while 125 (55%) did not meet criteria for obesity. The mean BMI of the sample was 30.01. Further breakdown revealed that 22.17% (n = 49) of the total sample had a BMI within a normal-weight range (BMI ≤ 24.9). The greatest proportion of patients (34%, n = 76) were classified as overweight based on a BMI between 25 and 29.9. “Mildly” obese participants (BMI = 30.0 – 34.9) comprised 21.27% (n = 47) of the total sample, while the remaining 22.17% (n = 49) consisted of severely obese participants (BMI ≥ 35); see Figure 1.

Figure 1. Proportions of weight status based on BMI
Measures

Weight Status. For the purposes of this study, BMI was used to calculate obesity as this is the measurement standard employed by the World Health organization, which defines underweight, BMI < 18.5; normal weight, BMI = 18.5 – 24.9; overweight, BMI = 25.0 – 29.9; obese (mild), BMI = 30.0 – 34.9; obese (moderate/severe) BMI ≥ 35.0. BMI is the ratio of weight to height and is calculated as weight in kilograms divided by the square of height in meters. BMIs above the cutoff of 30 are associated with an increased risk for weight-related health concerns (Fabricatore & Wadden, 2006). For this study, BMIs were calculated from the height and weight gathered at the patient’s initial medical evaluation by the clinic, retrieved from the patient’s electronic medical record, and converted to BMI and appropriate weight category for analysis.

Average Pain Intensity. Patients were asked to rate their least, worst, and average pain intensity over the previous week using a self-report Numerical Pain Rating (NRS) that ranged from 0 (“no pain”) to 10 “worst pain imaginable.” As pain is a subjective experience, self-report has been found to be the most reliable and valid measure (Melzack & Katz, 2001) and NRS is a standard instrument used in research and clinical contexts. For the purpose of this study, average pain ratings provided at intake and discharge interviews were used in order to best understand the participant’s overall pain experience. A reduction in average pain intensity by 20% is generally considered clinically significant (Dworkin et al., 2008).

Depression. The Beck Depression Inventory, 2nd edition (BDI- II; Beck, 1996) was used to assess patients for cognitive, somatic, and emotional components of depression. The items on this measure also relate to somatic complaints such as sleep disturbance, lack of energy, and weight change that can also be directly linked to chronic pain, and thus may inflate scores
somewhat. It has been found to discriminate well between patients with and without depression (Harris & D’Eon, 2008). The BDI-II is a 21-item self-report measure designed to assess symptoms of depression in adolescents and adults. It provides a single overall score ranging from 0 to 63. Test takers rate various depressive symptoms from 0 (not present) to 3 (severe), based on their status during the previous two weeks. General guidelines for patients diagnosed with major depression suggest the following cut-off scores of depressive severity: minimal (0–13); mild (14–19); moderate (20–28); and severe (29–63). Convergent validity with the original Beck Depression Inventory (BDI-I; Beck, 1961) is reported at $r = .93$ and $r = .71$ with the Hamilton Psychiatric Rating Scale for Depression; its internal consistency is excellent, $\alpha = .91$ among psychiatric outpatients, $\alpha = .93$ among undergraduate students (Harris & D’Eon, 2008). Clinically significant change is generally considered to be reflected by a change in scores by nine points (Jacobson & Truax, 1991).

**Pain-related Fear and Anxiety.** Patients were provided a self-administered instrument designed to assess pain-related anxiety and fear. The Pain Anxiety Symptom Scale (PASS) is a 40-item scale that measures the fear of pain across cognitive, behavioral, and physical domains (McCracken, Zayfert, & Gross, 1992). The items load onto four subscales: Fearful appraisal (a measure of fearful cognitions and negative expectancies related to pain), Cognitive anxiety (racing thoughts and impaired concentration related to pain), Physiological anxiety (somatic responses to pain), and Escape and avoidance (overt behavioral responses intended to reduce or eliminate pain). Questions are answered on a 7-point Likert scale ranging from never (0) to always (6), and the internal consistency is good with a Cronbach’s alpha for the entire instrument at .94. Its construct validity has been established by its moderate correlations with other anxiety scales. PASS subscale scores have been found to significantly correlate with measures of trait
anxiety (McCracken, Zayfert, & Gross, 1992). PASS scores have also been shown to be negatively correlated with physical assessments of range of motion and positively correlated with ratings of fear during these assessments (McCracken & Gross, 1995). PASS scores have also be found to be related to behavioral measures of disability and patient reports of fear of movement. Higher scores indicate greater anxiety responses. While clinical use is built around patients’ particular subscale scores as treatment targets, for the purpose of this study, scores reported are patients’ overall percentile ranks (compared with normative data provided by the test publisher). As a patients’ levels of pain related anxiety and fear of movement can be compared to that of other pain patients, scores above the 90th percentile have been characterized as a “significantly unhelpful level of anxious responding (McCracken & Gross, 1993).

Perceived Ability to Self-manage Pain. Patients were assessed for their confidence in ability to perform specific tasks, engage in meaningful activities, and cope in spite of their pain, using the Pain Self Efficacy Questionnaire (PSEQ) (Nicholas, 2007). The PSEQ is a 10 item scale that reflects an individual’s beliefs about their capacity to manage their pain. It is considered useful as a screening instrument to gauge a person’s ability to benefit from pain management programs and high PSEQ scores are strongly associated with clinically significant functional gains in treatment (Nicholas, 2007). Scores cut-offs of 40 have been found to be associated with maintained treatment gains at 6-12 months, while scores of 30 or below suggest difficulty in maintaining treatment gains and greater likelihood of ongoing disability. Questions are answered on a 7-point Likert scale ranging from “not at all confident” (0) to “complete confident (6), and include such questions as “I can enjoy things despite the pain,” and “I can do most household chores despite the pain.” Internal consistency is excellent, with Cronbach’s alpha calculated at 0.92. Validity was assessed through analysis of the factor structure and
examination of the PSEQ's correlation with other validated measures and was found to be
strong. The PSEQ has been found to be a strong predictor of functional outcomes from chronic
pain treatment (Strong, 2002).

Perceived Disability. Participants were provided a functional assessment to evaluate the
degree of disability they experience related to their pain. The Perceived Disability Questionnaire
(PDQ) (Anagnostis, Gatchel, and Mayer, 2004) is a 15 item instrument that yields a score from
0 – 150, and is designed for a wide range of musculoskeletal disorders rather than simply chronic
low back pain. Test-retest reliability coefficients range from .94 to .98, and internal consistency
is measured through Cronbach’s alpha of .96. Construct validity is also regarded to be excellent
as the measure is highly correlated with two other highly validated measures, including the
Oswestry and SF-36 (Anagnostis et al., 2004). Higher intake scores on the PDQ are associated
with decreased return-to-work retention, and higher scores post treatment are associated with
decreased return to work rates, decreased work retention (staying at the job), and increased
percentages of seeking healthcare from a new provider (Gatchel et al, 2006). Additionally, PDQ
scores can be broken down into three categories: Mild/Moderate (0-70); Severe (71-100); and
extreme (101-150). This breakdown has been found to be predictive of work and health-related
outcomes such as return to work, work retention, and healthcare utilization (Gatchel et al, 2006).

It should be noted that the measures used to evaluate treatment outcome herein were not
chosen by the clinic for their research, but rather their clinical value. The clinic in which the
treatment is conducted has determined that the measures they use to assess their patients at intake
and discharge (aside from BMI) provide them the most useful clinical data regarding the
emotional status of their patients, potential targets for treatment, suitability for treatment, and
prediction of functioning post-treatment (C. Buist, Clinic Director, personal communication, July
The particular measures utilized in this study were chosen by this researcher because they contained the most complete data (pre and post treatment) on each participant, as some other measures related to attitudes and coping skills were only provided at intake. It was originally proposed that measures of cardiovascular fitness, flexibility, and functional capacity would be used to objectively evaluate physical outcomes of treatment. However, the data collected by the program simply noted “Improved fitness” and “Improved flexibility” as part of its outcome data. There was no information provided to indicate what “improved” ratings were based upon or to what amount of clinical change differentiated improved from not improved. Further, there was little variance in these scores in the treatment outcomes data, as almost all patients that had completed the program were rated as having improved their flexibility and functional capacities (lift/carry, sitting and standing tolerances, etc.). This data was considered to add little interpretable data, and was therefore not considered for analysis.

**Intervention**

Prior to treatment, patients underwent a rigorous one-day assessment evaluation that included a medical and neurological evaluation, including medication review performed by registered nurses and physicians; physical ability testing performed by licensed physical therapists; functional capacity evaluation performed by licensed occupational therapists; vocational consultation by a vocational counselor; and psychological assessment performed by licensed clinical psychologist. As part of this intake evaluation, participants completed self-report measures that assessed the various dimensions of their subjective and objective response to chronic pain.

The specific patient demographic and medical variables collected through clinical interview and intake questionnaires included age, gender, ethnicity, height/weight, mechanism of
injury, morphine dose equivalence (MSEQ), date of injury, injury-treatment interval, and insurer/referral source (see Appendix A for patient intake packet). Specific pain and disability information were gathered through a modified McGill Pain Inventory (Melzack, 1975), which evaluated the intensity, location, affective, and sensory qualities of their pain. Patients were also evaluated with a psychosocial assessment interview that focused on current symptoms, the history of treatment, psychosocial background, and the impact of chronic pain on the client’s life. The psychological battery consisted of symptom inventories and pain specific measures, including the Beck Depression Inventory (BDI-II), Pain Anxiety Symptom Scale (PASS), Chronic Pain Coping Inventory (CPCI), Survey of Pain Attitudes (SOPA), Pain Self-Efficacy Questionnaire (PSEQ), and Perceived Disability Questionnaire (PDQ). Treatment success is partially gauged by the amount of change from pre- to post-treatment on these self-report measures.

The discharge process consisted of a medical review, physical examination, tests of functional tolerances and physical abilities, and re-admittance five of the specific self-report measures (NRS, BDI-II, PASS, PSEQ, and PDQ). Following the completion of discharge reports, treatment outcome information is compiled and entered into medical records by clinical and administrative staff, and also entered into an outcome database by the quality assurance director.

Treatment consisted of an intensive 20-day program multidisciplinary pain-management program led by a team of physicians, nurses, vocational counselors, physical therapists, occupational therapists, clinical psychologists, and graduate students. Patients were treated on an outpatient basis, with local patients staying at home and patients from over 25 miles away staying at a nearby hotel arranged by the program and paid for by their worker’s compensation
system. All patients attended an 8-hour per day structured treatment program, and any absences were made up for by adding additional treatment days.

While treatment plans were individualized based upon the patients’ particular injuries, the focus was upon functional restoration rather than elimination of pain. Daily treatment components included: one-hour group therapy sessions for instruction in pain management, stress management, and coping skills; one-hour psychoeducation sessions related to physiological and psychological bases of pain, medication use, nutrition, worker’s compensation systems, and behavior change principles. Additional group sessions were related to improved body mechanics, mastering activities of daily living, increasing functional tolerances, and relaxation training. All patients received ongoing individual biofeedback training, cognitive-behavioral therapy, and vocational counseling. Patients using narcotics had their medications evaluated on an individual basis, and were tapered off (or lowered) during the course of the program. Patients also participated in 3 hours of daily stretching, physical therapy, and occupational therapy classes that included individualized exercise quotas, therapeutic massage, thermal modalities, TENS, and assistive or orthotic devices. Functional restoration focused upon improving patients’ sitting and standing tolerances, walking endurance, lift and carry capacity, and improved range of motion, balance, and ergonomic skills.

**Procedures**

Prior to participating in the intake assessment, patients were provided an informed consent document requiring their signature to allow any information gathered at intake, treatment, discharge, and follow-up to be used for any future research. Patients were not required to sign the informed consent as a requirement for treatment and only clients who provided consent had data available for evaluation in this study. Following approval of this study’s
proposals, permission was obtained from both the pain clinic’s Clinical Director and Privacy Officer, and Pacific University’s Institutional Review Board to retrospectively gather BMI data from the electronic patient medical record.

After IRB approval was granted, a copy of the company’s outcomes database was created, and patient height and weight data gathered from the medical records was added to each patient’s record. The height and weight of each patient was the only variable of interest not typically extracted but was gathered from EMR and added to the data set for each patient sampled, serving as the calculation for patient BMI. All other variables of interest (scores on intake/outcome variables) had previously been compiled within a database maintained by the Quality Control department at PRA. These scores were verified against original clinical assessment reports, and several corrections were made to the scores reported in the company database. Following completion of the database with all variables of interest, each patient record was assigned a unique identifying number and subsequently all identifiable information was deleted (name and birthday). This de-identified database was saved as a password-protected encrypted file to a laptop for the researcher’s analysis.

Data Screening and Preliminary Analysis

Of 272 individual patients who were admitted to the clinic during the time period under consideration (2009 – 2011), 34 patients (12.5%) did not complete at least 17 days of the 20-day program, and these records were eliminated from consideration as it was not considered that they would have adequately engaged in the full program. 9 (3.3%) patient records did not include height and/or weight information, making BMI calculation impossible, and these records were also eliminated from the sample. Lastly, 8 (2.9%) patient records did not include scoring data from intake or discharge measures, and these records were also eliminated from the sample, for a
total of 51 records (18.8%), leaving 221 patient records for primary analysis. Of the 34
treatment dropouts, BMI was available from 26 participants (76%). Mean BMI of this treatment
dropout sample was 31, and 65% \((n = 17)\) met criteria for obesity with BMIs \(\geq 30\). Of the total
sample, 14.4% of obese participants dropped out of treatment whereas 6.6% of non-obese
participants failed to complete treatment (see Results section for analysis of this dropout rate).

A review of the data was completed to ensure accuracy and completeness, and a
preliminary analysis screened all variables of interest using the SPSS 21.0 statistics program to
ensure accuracy of data entry, screen for missing values and outliers, and evaluate normality of
distributions. All scores on variables of interest were verified against the original clinical intake
and discharge reports, and several corrections were made to the scores originally recorded in the
clinic’s outcome database. The accuracy of data entry was completed by visually inspecting the
data file to determine if any overtly inconsistent scores were present on any of the variables, in
addition to reviewing the SPSS descriptive output for minimum and maximum scores. In order to
address the assumptions of repeated measures ANOVA, the distribution of outcome scores at
pre-and post-treatment were grouped according to weight status (Obese and Non-Obese) and
examined for the presence of outliers through visual inspection of histograms and box-plots for
pre-and post-treatment Pain, BDI, PASS, PSEQ, and PDQ raw scores. In the event an extreme
outlier was determined by interquartile range, the score was evaluated for the number of standard
deviations it lay from the mean.

There were six extreme outliers found in the Non-Obese group pre-treatment Pain scores
based on interquartile range. An examination of the distribution of these scores indicated that all
scores fell within three standard deviations. Examination of the participants’ raw data verified
that these outlier scores were not the result of entry errors and that the participants who provided
these scores were valid members of the participant population. Therefore, these outliers were included in the data analysis. There were no extreme outliers found for Obese group Pain pre-treatment scores based on interquartile range and all scores fell within three standard deviations of the mean.

There were no extreme outliers found for Non-Obese group post-treatment Pain scores based on interquartile range and all scores fell within three standard deviations of the mean. There was one extreme outlier found in the Obese group post-treatment Pain scores based on interquartile range, and an examination of the distribution indicated that all scores fell within three standard deviations of the mean. Examination of this participant’s raw data verified that this score was not the result of entry error and that the participant who provided this score was a valid member of the participant population, and thus was included in the data analysis.

There were no extreme outliers found for Non-Obese group BDI pre-treatment scores based on interquartile range and all scores fell within three standard deviations of the mean. Two extreme outliers were present within the Obese group BDI pre-treatment scores based on interquartile range and an examination of the distribution indicated that these scores fell within three standard deviations of the mean. Examination of these participants’ raw data verified that these scores were not the result of entry errors and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis.

There were three extreme outliers found for Non-Obese group BDI post-treatment scores based on interquartile range; an examination of the distribution indicated that these scores ($z = 3.29, z = 3.39, z = 3.39$) fell slightly outside of three standard deviations of the mean. Examination of these participants’ raw data verified that these scores were not the result of entry
errors and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis. In addition, inclusion of these scores was deemed relevant to the study’s assumption that chronic pain patients would show a wide variance in response to treatment.

Two extreme outliers were found within the Obese group BDI post-treatment scores based on interquartile range and an examination of the distribution indicated that these scores \( z = 3.29, z = 3.39 \) fell slightly outside of three standard deviations of the mean. Examination of these participants’ raw data verified that these scores were not the result of entry errors and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis.

There were no extreme outliers on PASS pre- or post-treatment scores for either group based on interquartile range. An examination of the distribution indicated that all scores fell within three standard deviations of the mean.

Two extreme outlier scores were found on the PSEQ pre-treatment scores for the Non-obese group based on interquartile range, however an examination of the distribution indicated that all scores fell within three standard deviations of the mean. There were no extreme outlier scores found for the Obese groups PSEQ pre-treatment scores. There were no extreme outliers on PSEQ post-treatment scores for either group based on interquartile range. An examination of the distribution indicated that all scores fell within three standard deviations of the mean.

There were two extreme outlier scores found on the PDQ pre-treatment for the Non-obese group and one extreme outlier found in the PDQ pre-treatment for the Obese group, based on interquartile range; an examination of the distribution indicated that all scores fell within three standard deviations of the mean. Examination of the patients’ raw data indicated that these scores
were not the result of data entry and these participants were valid members of the patient population and were thus included for analysis. There were no extreme outliers found in the PDQ post-treatment scores for the Non-obese group. There was one extreme outlier found in the PDQ post-treatment scores for the Obese group, based on interquartile range, however an examination of the distribution indicated that all scores fell within three standard deviations of the mean. Examination of the patient’s raw data indicated that this score was not the result of data entry and this participant was a valid member of the patient population and was thus included for analysis.

Further preliminary analysis of each dependent variable within each group (Obese/None Obese) was conducted to ensure that scores met assumptions for normality (See Table 2 for Skewness and Kurtosis of pre-treatment and post-treatment scores on all variables). Though some distributions of the variables (e.g., BDI post-treatment) were moderately skewed and/or kurtotic, normality was assumed as the values were not +/- 2.0. Tests for equality of error variance and homogeneity of variance-covariance matrices will be addressed within the Results section.

**Table 2**

*Distribution of Scores on Pre and Post-Treatment Dependent Variables*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-Treatment</th>
<th>Post Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obese</td>
<td>Non-Obese</td>
</tr>
<tr>
<td></td>
<td>Skewness</td>
<td>Kurtosis</td>
</tr>
<tr>
<td>Pain</td>
<td>.017</td>
<td>-.600</td>
</tr>
<tr>
<td>BDI</td>
<td>.348</td>
<td>.014</td>
</tr>
<tr>
<td>PASS</td>
<td>-.172</td>
<td>-1.112</td>
</tr>
<tr>
<td>PSEQ</td>
<td>.215</td>
<td>-.136</td>
</tr>
<tr>
<td>PDQ</td>
<td>-.562</td>
<td>.264</td>
</tr>
</tbody>
</table>
To examine mean change scores between groups, 5 additional variables (e.g., *Change in Pain, Change in Depression, etc.*) were created by subtracting the pre-treatment raw score from the post-treatment raw score on each variable of interest. In order to address the assumptions of Independent samples *t*-test, the distribution of change scores were grouped according to weight status (Obese and Non-Obese) and examined for the presence of outliers through visual inspection of histograms and box-plots for Change in Pain, Depression, Anxiety, Self-efficacy, and Disability. In the event an extreme outlier was determined by interquartile range, the score was evaluated for the number of standard deviations it lay from the mean, and whether the score was a result of error.

There were eleven extreme outliers found for Non-Obese group *Change in Pain* scores based on interquartile range; an examination of the distribution indicated that only one of these scores (*z* = 3.28) fell slightly outside of three standard deviations of the mean. Examination of this participant’s raw data verified that this score was not the result of entry error and that the participant who provided this score was a valid member of the participant population, and thus was included in the data analysis. In addition, inclusion of this score was deemed relevant to the study’s assumption that chronic pain patients would show a wide variance in response to treatment.

There were six extreme outliers found for Obese group *Change in Pain* scores based on interquartile range; an examination of the distribution indicated that only one of these scores (*z* = 3.31) fell slightly outside of three standard deviations of the mean. Examination of this participant’s raw data verified that this score was not the result of entry error and that the participant who provided this score was a valid member of the participant population, and thus was included in the data analysis. In addition, inclusion of this score was deemed relevant to the
study’s assumption that chronic pain patients would show a wide variance in response to treatment.

There were four extreme outliers found for Non-obese group Change in Depression scores based on interquartile range; an examination of the distribution indicated that none of these scores fell outside of three standard deviations from the mean. Examination of these participants’ raw data verified that these scores were not the result of entry error and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis.

There was one extreme outlier found for Obese group Change in Depression scores based on interquartile range; an examination of the distribution indicated that this score \( z = 3.62 \) fell slightly outside of three standard deviations from the mean. Examination of this participant’s raw data verified that this score was not the result of entry error and that the participant who provided this score was a valid member of the participant population, and thus was included in the data analysis.

There were six extreme outliers found for Non-obese group Change in Anxiety scores based on interquartile range; an examination of the distribution indicated that none of these scores fell outside of three standard deviations from the mean. Examination of these participants’ raw data verified that these scores were not the result of entry error and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis. There were no extreme outliers found for the Obese group’s Change in Anxiety scores and all scores fell within three standard deviations from the mean.

There were no extreme outliers found for the Non-obese group’s Change in Self-Efficacy scores and all scores fell within three standard deviations from the mean. There was one extreme
outliers found for the Obese group’s Change in Self-Efficacy scores based on interquartile range; an examination of the distribution indicated that this score ($z = 3.08$) fell slightly outside of three standard deviations from the mean. Examination of these participants raw data verified that these scores were not the result of entry error and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis.

There were two extreme outliers found for Non-obese group Change in Disability scores based on interquartile range; an examination of the distribution indicated that none of these scores fell outside of three standard deviations from the mean. Examination of these participants’ raw data verified that these scores were not the result of entry error and that the participants who provided these scores were valid members of the participant population, and thus were included in the data analysis. There were no extreme outliers found for the Obese group’s Change in Disability scores and all scores fell within three standard deviations from the mean.

Further preliminary analysis of the change scores within each group (Obese/None Obese) was conducted to ensure that scores met assumptions for normality (See Table 3 for Skewness and Kurtosis of change scores on all variables). Though some distributions of the variables (e.g., BDI post-treatment) were moderately skewed and/or kurtotic, normality was assumed as the values were not +/- 2.0. Equality of variances was addressed through Levene’s Test, and none were found to be significant. Thus, although the Obese and Non-obese groups were of unequal size, results are reported with equal variances assumed.
Table 3

Distribution of Scores on Change Variables

<table>
<thead>
<tr>
<th>Measure</th>
<th>Obese Skewness</th>
<th>Obese Kurtosis</th>
<th>Non-Obese Skewness</th>
<th>Non-Obese Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in Pain</td>
<td>.061</td>
<td>1.483</td>
<td>-.205</td>
<td>1.068</td>
</tr>
<tr>
<td>Change in BDI</td>
<td>-.445</td>
<td>.350</td>
<td>-.514</td>
<td>.946</td>
</tr>
<tr>
<td>Change in PASS</td>
<td>-.304</td>
<td>-.397</td>
<td>-.565</td>
<td>-.047</td>
</tr>
<tr>
<td>Change in PSEQ</td>
<td>.493</td>
<td>-.091</td>
<td>.588</td>
<td>-.093</td>
</tr>
<tr>
<td>Change in PDQ</td>
<td>-.175</td>
<td>-.531</td>
<td>-.786</td>
<td>.436</td>
</tr>
</tbody>
</table>

Though some of the score distributions were moderately skewed or kurtotic, normality was assumed because none of the values were greater than ± 2.0; as normality could be assumed, it was determined that an independent samples t-test would be appropriate for statistical analysis of these change scores. Scores on the different measures were independent from one another and therefore the assumption of independence was met.

To test the hypothesis (1) that obesity would predict differences in response to treatment, such that participants with BMIs ≥ 30 (obese) would have higher rates of treatment drop-out than participants with BMIs < 30 (non-obese), a chi-square test of independence was performed to compare the proportion of cases that occurred in each group.

Primary clinical analyses examined the effects of treatment and obesity on MCPM outcomes, specifically measures of pain intensity, depression, anxiety, self-efficacy, and disability. To investigate these issues, the sample of participants was grouped according to their weight status: obese or non-obese; this between-subjects factor was labeled Obesity. In addition, each participant was measured before and after treatment for their level of pain intensity, creating a single within-subjects factor, labeled Time.

To evaluate Hypothesis 2, a series of mixed-model repeated-measure ANOVAs were conducted to evaluate the within-subjects main effect for Time to determine if treatment
influenced the participants’ levels of pain intensity, depression, anxiety, self-efficacy, and disability (i.e., did the mean pain rating change from pre-treatment to post-treatment). To address Hypothesis 3, the interaction effect from the mixed model ANOVA, Time x Obesity, was examined to determine if group differences on the outcome measures varied across time.

To address Hypotheses 4 – 8, the between-subjects main effects for Obesity, the mixed-model repeated-measure ANOVAs were evaluated to determine if obesity influenced participant levels of pain intensity, depression, anxiety, self-efficacy, and disability (i.e., did obese participants have different mean pain intensity ratings than non-obese participants).

To examine mean change scores between groups (Hypotheses 9 – 13), 5 additional variables (e.g., Change in Pain, Change in Depression, etc.) were created by subtracting the pre-treatment raw score and the post-treatment raw score on each variable of interest. A series of independent-samples t-tests were conducted to evaluate whether the mean amount of change from pre-treatment to post-treatment differed between obese and non-obese participants on each outcome variable. The independent variable was the weight status of each group, with two levels Obese and Non-obese. The dependent variables were the participants’ change scores on measures of pain, depression, anxiety, self-efficacy, and disability. To reduce the probability of committing a Type I error due to multiple pairwise comparisons within the mixed ANOVA and Independent t-tests, a Bonferroni correction was performed to each set of analyses. As there were 5 pairwise comparisons within each analysis, the alpha was set at 0.01 (\( \alpha = 0.05/5 \)). The Bonferroni was chosen in this instance as it is a general, though conservative, method of evaluating multiple pairwise comparisons (Green & Salkind, 2008).
Results

Preliminary Analyses

Demographics comparisons using chi-square and independent samples $t$-test results showed no statistically significant differences between obese and non-obese groups. See Table 4 for more information related to demographic comparisons.

**Table 4**
*Demographics Comparisons Between Obese And Non-Obese Groups.*

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Obese (N = 96)</th>
<th>Non-obese (N = 125)</th>
<th>$P^t$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>45.27 (10.03)</td>
<td>44.45 (10.05)</td>
<td>0.55</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>68.8%</td>
<td>63.2%</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>31.3%</td>
<td>36.8%</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>84.4%</td>
<td>84.8%</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>15.6%</td>
<td>15.2%</td>
<td></td>
</tr>
<tr>
<td>Pain duration (months)</td>
<td>40.59 (39.74)</td>
<td>46.78 (56.79)</td>
<td>0.36</td>
</tr>
<tr>
<td>Morphine equivalency (pretreatment)</td>
<td>58.30 (71.86)</td>
<td>76.40 (70.81)</td>
<td>0.12</td>
</tr>
<tr>
<td>Average pain intensity (pre treatment)</td>
<td>6.37 (1.51)</td>
<td>6.33 (1.44)</td>
<td>0.85</td>
</tr>
<tr>
<td>Mechanism of Injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper Body</td>
<td>30.2%</td>
<td>24.0%</td>
<td>0.58</td>
</tr>
<tr>
<td>Lower Extremity</td>
<td>9.4%</td>
<td>8.0%</td>
<td></td>
</tr>
<tr>
<td>Lumbar</td>
<td>46.9%</td>
<td>48.8%</td>
<td></td>
</tr>
<tr>
<td>Multiple</td>
<td>13.5%</td>
<td>19.2%</td>
<td></td>
</tr>
</tbody>
</table>

$^t$ Chi square analyses and independent samples $t$-tests

As previously noted, 14.4% of obese participants dropped out of treatment whereas 6.6% of non-obese participants failed to complete treatment. Drop-out rates between groups were compared using a Chi-Square Test of Independence (with Yates Continuity Correction), which indicated no connection between obesity and attrition, $\chi^2 (1, N = 239) = .99, p = .32, \Phi = .08$. It was expected that obese patients would have higher treatment drop out rates than non-obese patients. This finding failed to support this hypothesis (1).
**Primary Clinical Outcomes.**

The following results are presented according to each outcome domain tested. The means and standard deviations of each Pre- and Post-test variable are presented in Table 5. The main and interaction effects from the mixed-model ANOVA are presented in Table 6.

### Table 5
**Means and Standard Deviations on Outcome Variables Pre- and Post- Treatment by Obesity**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-Treatment</th>
<th></th>
<th>Post Treatment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obese</td>
<td>Non-Obese</td>
<td>Obese</td>
<td>Non-Obese</td>
</tr>
<tr>
<td></td>
<td>N = 96</td>
<td>M (SD)</td>
<td>N = 125</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Pain</td>
<td>6.37 (1.51)</td>
<td>6.33 (1.45)</td>
<td>5.57 (1.74)</td>
<td>5.58 (1.54)</td>
</tr>
<tr>
<td>BDI</td>
<td>22.42 (10.78)</td>
<td>20.78 (11.29)</td>
<td>14.11 (9.89)</td>
<td>13.90 (10.17)</td>
</tr>
<tr>
<td>PASS</td>
<td>48.28 (23.89)</td>
<td>43.02 (25.90)</td>
<td>35.08 (25.19)</td>
<td>33.06 (24.89)</td>
</tr>
<tr>
<td>PSEQ</td>
<td>24.88 (10.97)</td>
<td>25.34 (11.04)</td>
<td>37.04 (13.21)</td>
<td>36.66 (12.17)</td>
</tr>
<tr>
<td>PDQ</td>
<td>99.12 (19.07)</td>
<td>97.90 (19.00)</td>
<td>83.68 (23.87)</td>
<td>86.00 (25.13)</td>
</tr>
</tbody>
</table>

**Pain Intensity**

A mixed-model ANOVA was conducted to evaluate the effect of treatment and obesity on a measure of average pain intensity to determine if changes in pain intensity following treatment varied as a function of weight status. 221 participants rated their average pain intensity on a numerical rating scale of 0 - 10 at both pre-treatment and post-treatment; these scores were grouped according to the participants’ weight status, **Obese** and **Non-Obese**. The dependent variable was participants’ pain ratings, while the two factors were **Time** and **Obesity**. An interaction effect (*Time x Obesity*) examined whether or not changes in pain ratings varied as a function of obesity (Hypothesis 2). A within-subjects main effect for **Time** was examined to see if participants’ mean pain ratings changed from pre-treatment to post-treatment while a between
subjects main-effect for *Obesity* evaluated whether obese participants had different (greater) mean pain intensity ratings across time periods than non-obese participants (Hypothesis 3). The results of Levene’s test for the pretreatment pain rating, $F(1,219) = .51, p = .475$ and post-treatment pain rating $F(1,219) = .24, p = .236$ were not significant, indicating that the error variance in the dependent variable was equivalent across groups. Box’s Test for Equality of Covariance Matrices was not significant ($p = .423$), therefore homogeneity of variance-covariance was assumed.

The *Time x Obesity* interaction effect was not significant, Wilks’ $\Lambda = 1.00, F(1, 219) = .05, p = .817$, partial $\eta^2 < .001$, indicating that less than .1% of the variance could be accounted for by an interaction between time and obesity, a very small effect size. The *Time* main effect was significant, Wilks’ $\Lambda = .81, F(1, 219) = 51.42, p < .001$, partial $\eta^2 = .19$, with both groups showing reductions in pain intensity, and indicating that 19% of the variance could be accounted for by time, a large effect size. The results indicated participants showed significant reductions from pre- to post-test on pain ratings scores ($M = -.78, SE = .11, 95\% \text{ CI} [-.99, -.56]$). The main effect comparing groups of participants was not significant, $F(1,219) = .01, p = .944$, partial $\eta^2 < .001$, suggesting no difference in the pain intensity ratings between obese and non-obese participants. It was expected that participants would demonstrate significant reductions in pain intensity from pre- to post-treatment, and this finding supports that hypothesis (2). It was expected that these reductions would vary according to weight status, and this finding failed to support that hypothesis (3). Lastly, it was expected that obese and non-obese groups would differ in average pain intensity, and this finding failed to support that hypothesis (4) (see *Figure 2*).
An independent-samples $t$-test was conducted to evaluate the hypothesis (9) that non-obese participants would show greater change in pain scores from pre-to post-treatment than obese participants. The test was not significant, $t(219) = .23, p = .817$. Obese participants’ Change in Pain scores ($M = -.80, SD = 1.55$) were comparable with Non-obese participants Change in Pain scores ($M = -.75, SD = 1.63$). The 95% confidence interval for the difference in means ranged from -.38 to .48. The eta square effect size was very small ($\eta^2 < .001$), indicating that less than .1% of the variability in change in pain ratings was accounted for by whether or not the participant was obese. Based on these results, obese participants’ Change in Pain from pre to
post-treatment do not differ from those of Non-obese participants, and thus this hypothesis was not supported.

As noted, the overall mean change in pain was less than one point ($M = -0.77, SD = 1.59$, 95% CI [-.99, -.56]), though Change in Pain ratings ranged from a reduction of 6 points to an increase of 4.5 points. The most frequent change score was zero ($n = 48, 21.7$%), and 57% of patients experienced change of 1 point or less on the 10 point scale; 48 participants (21.7%) reported a reduction in pain ratings by two points or more. 65% of participants rated their pain as a 6/10 or worse at intake, while 50% rated their pain as a 6/10 or worse at discharge. At discharge, 13 participants (5.9%) reported their pain had increased by two points or more. 10% ($n = 22$) of patients completed treatment with pain intensity ratings of 3.5/10 or less.

**Depression**

A mixed-model ANOVA was conducted to evaluate the effect of treatment and obesity on a measure of average depression scores to determine if changes in depression following treatment varied as a function of weight status. 221 participants completed the BDI – II, rating their depression on a numerical rating scale of 0 - 63 at both pre-treatment and post-treatment; these scores were grouped according to the participants’ weight status, Obese and Non-Obese. The dependent variable was participants’ depression scores, while the two factors were Time and Obesity. An interaction effect (Time x Obesity) examined whether or not changes in depression scores varied as a function of obesity (Hypothesis 2). A within-subjects main effect for Time was examined to see if participants’ mean depression scores changed from pre-treatment to post-treatment while a between subjects main-effect for Obesity evaluated whether obese participants had different (greater) mean depression scores across time periods than non-obese participants (Hypothesis 3). The results of Levene’s test for the pretreatment depression score, $F(1,219) =$
2.041, \( p = .154 \) and post-treatment depression score \( F(1, 219) = .03, \ p = .866 \) were not significant, indicating that the error variance in the dependent variable was equivalent across groups. Box’s Test for Equality of Covariance Matrices was not significant (\( p = .582 \)), therefore homogeneity of variance-covariance was assumed.

The \( Time \times Obesity \) interaction effect was not significant, Wilks’ \( \Lambda = 1.00, F(1, 219) = 1.09, \ p = .297, \) partial \( \eta^2 = .005 \), indicating that less than .5% of the variance could be accounted for by an interaction between time and obesity, a very small effect size. The \( Time \) main effect was significant, Wilks’ \( \Lambda = .64, F(1, 219) = 124.72, \ p < .001, \) partial \( \eta^2 = .36, \) with both groups showing reductions in depression, and indicating that 36% of the variance could be accounted for by time, a large effect size. The results indicated participants showed significant reductions from pre- to post-treatment on depression scores (\( M = -7.59, SE = .68, 95\% \ CI [-8.93, -6.25] \)). The main effect comparing groups of participants was not significant, \( F(1, 219) = .53, \ p = .467, \) partial \( \eta^2 = .002, \) suggesting little difference in the depression scores between obese and non-obese participants. It was expected that participants would demonstrate significant reductions in depression from pre- to post-treatment, and this finding supports that hypothesis (2). It was expected that these reductions would vary according to weight status, and this finding failed to support that hypothesis (3). Lastly, it was expected that obese and non-obese groups would differ in average pain intensity, and this finding failed to support that hypothesis (5) (see Figure 3).
An independent-samples $t$-test was conducted to evaluate the hypothesis (10) that non-obese participants would show greater change in depression scores from pre- to post-treatment than obese participants. The test was not significant, $t(219) = 1.20, p = .231$. Obese participants’ Change in Pain scores ($M = -8.30, SD = 10.60$) were comparable with Non-obese participants Change in Pain scores ($M = -6.66, SD = 9.70$). The 95% confidence interval for the difference in means ranged from -1.06 to 4.35. The eta square effect size was very small ($\eta^2 < .001$), indicating that less than .1% of the variability in change in depression scores was accounted for by whether or not the participants were obese. Based on these results, obese participants’ Change
in Depression from pre to post-treatment do not differ from those of Non-obese participants, and thus this hypothesis was not supported.

As noted, the overall mean reduction in BDI score was 7 points \((M = -7.37, SD = 10.11, 95\% \text{ CI } [-8.71, -6.03])\), with changes in depression ratings ranging from a decrease of 44 points to an increase of 21 points. The most frequent change score was zero \((n = 26, 11.8\%)\), though 65.2\% \((n = 144)\) of participants experienced a change of 5 points or more. 36 participants (21.7\%) reported a reduction in depression scores of at least 10 points, while 8 participants (3.6\%) reported their depression scores had increased by 10 points or more.

**Pain-Related Fear and Anxiety**

A mixed-model ANOVA was conducted to evaluate the effect of treatment and obesity on a measure of Pain-related fear and anxiety (PASS) scores to determine if changes in anxiety following treatment varied as a function of weight status. 221 participants completed the PASS, which rates their anxiety on a scale of 1 – 100 at both pre-treatment and post-treatment; these scores were grouped according to the participants’ weight status, Obese and Non-Obese. The dependent variable was participants’ anxiety scores, while the two factors were Time and Obesity. An interaction effect \((\text{Time } \times \text{Obesity})\) examined whether or not changes in anxiety scores varied as a function of obesity (Hypothesis 2). A within-subjects main effect for Time was examined to see if participants’ mean anxiety scores changed from pre-treatment to post-treatment while a between subjects main-effect for Obesity evaluated whether obese participants had different (greater) mean anxiety scores across time periods than non-obese participants (Hypothesis 3). The results of Levene’s test for the pretreatment anxiety score, \(F(1,219) = 1.245, p = .266\) and post-treatment anxiety score \(F(1,219) = .02, p = .901\) were not significant, indicating that the error variance in the dependent variable was equivalent across groups. Box’s
Test for Equality of Covariance Matrices was not significant \((p = .631)\), therefore homogeneity of variance-covariance was assumed.

The \(Time \times Obesity\) interaction effect was not significant, Wilks’ \(\Lambda = 1.00, F(1, 219) = 1.08, p = .299\), partial \(\eta^2 = .005\), indicating that less than .5\% of the variance could be accounted for by an interaction between time and obesity, a very small effect size. The \(Time\) main effect was significant, Wilks’ \(\Lambda = .80, F(1, 219) = 55.14, p < .001\), partial \(\eta^2 = .20\), with both groups showing reductions in anxiety, and indicating that 20\% of the variance could be accounted for by time, a large effect size. The results indicated participants showed significant reductions from pre- to post-treatment on anxiety scores \((M = -11.58, SE = 1.60, 95\% CI [-14.65, -8.50])\). The main effect comparing groups of participants was not significant, \(F(1,219) = 1.46, p = .229\), partial \(\eta^2 = .007\), suggesting little difference in the anxiety scores between obese and non-obese participants. It was expected that participants would demonstrate significant reductions in anxiety from pre- to post-treatment, and this finding supports that hypothesis (2). It was expected that these reductions would vary according to weight status, and this finding failed to support that hypothesis (3). Lastly, it was expected that obese and non-obese groups would differ in anxiety across time, and this finding failed to support that hypothesis (6) (see Figure 4).

An independent-samples \(t\)-test was conducted to evaluate the hypothesis (11) that non-obese participants would show greater change in anxiety scores from pre- to post-treatment than obese participants. The test was not significant, \(t(219) = 1.04, p = .299\). Obese participants’ \textit{Change in Anxiety} scores \((M = -13.20, SD = 21.69)\) were comparable with Non-obese participants’ \textit{Change in Anxiety} scores \((M = -9.95, SD = 23.91)\). The 95\% confidence interval for the difference in means ranged from -2.90 to 9.39. The eta square effect size was very small \((\eta^2 = .005)\), indicating that .5\% of the variability in change in anxiety was accounted for by
whether or not the participant was obese. Based on these results, obese participants’ Change in Anxiety from pre to post-treatment do not differ from those of Non-obese participants, and thus this hypothesis was not supported.

![Graph showing pre and post-treatment anxiety for obese and non-obese groups](image.jpg)

**Figure 4.** Reductions in pain-related fear and anxiety scores from pre- to post-treatment between Obese and Non-obese participants.

As noted, the mean reduction in PASS scores was approximately 11.5 percentile points ($M = -11.36$, $SD = 22.98$, 95% CI [-14.41, -8.32]), with changes in anxiety ratings ranging from a decrease of 75 percentile points to an increase of 40 percentile points. The median and modal change were -5.00 ($n = 28$, 12.7%) though 107 participants (48.4%) reported a reduction in PASS scores of 10 percentile points or more. 39 participants (17.6%) reported their PASS scores increased by 10 percentile points or more. Though not previously hypothesized, it was noted that at intake 10 participants (4.5%) met the criterion for “problematic” extreme anxiety of 90th
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percentile or higher, and at discharge, 3 participants (1.4%) met this criterion. Due to the small number of participants, a Fisher’s Exact Test was conducted rather than a McNemar’s. The result indicated that this was a significant reduction in the number of participants with extreme ratings of PASS ≥ 90 following treatment, \( p < .001 \).

**Self-efficacy**

A mixed-model ANOVA was conducted to evaluate the effect of treatment and obesity on a measure of Pain-related self-efficacy ratings (PSEQ) to determine if changes in self-efficacy following treatment varied as a function of weight status. 221 participants completed the PSEQ, rating their self-efficacy on a numerical rating scale of 0 to 60 at both pre-treatment and post-treatment; these scores were grouped according to the participants’ weight status, *Obese* and *Non-Obese*. The dependent variable was participants’ self-efficacy scores, while the two factors were *Time* and *Obesity*. An interaction effect \( (Time \times Obesity) \) examined whether or not changes in self-efficacy scores varied as a function of obesity (Hypothesis 2). A within-subjects main effect for *Time* was examined to see if participants’ mean self-efficacy scores changed from pre-treatment to post-treatment while a between subjects main-effect for *Obesity* evaluated whether obese participants had different (lower) mean self-efficacy scores across time periods than non-obese participants (Hypothesis 3). The results of Levene’s test for the pretreatment self-efficacy score, \( F(1,219) > .001, p = .994 \) and post-treatment self-efficacy score \( F(1,219) = 1.25, p = .265 \) were not significant, indicating that the error variance in the dependent variable was equivalent across groups. Box’s Test for Equality of Covariance Matrices was not significant \( (p = .848) \), therefore homogeneity of variance-covariance was assumed.

The *Time x Obesity* interaction effect was not significant, Wilks’ \( \Lambda = 1.00, F(1, 219) = .27, p = .608 \), partial \( \eta^2 = .001 \), indicating that .1% of the variance could be accounted for by an
interaction between time and obesity, a very small effect size. The *Time* main effect was significant, Wilks’ $\Lambda = .52$, $F(1, 219) = 203.56$, $p < .001$, partial $\eta^2 = .48$, with both groups showing increases in self-efficacy, and indicating that 48% of the variance could be accounted for by time, a very large effect size. The results indicated participants showed significant increases from pre- to post-treatment on self-efficacy scores ($M = 11.74$, $SE = .82$, 95% CI [10.12, 13.36]). The main effect comparing groups of participants was not significant, $F(1,219) > .01$, $p = .978$, partial $\eta^2 = .002$, suggesting no difference in the self-efficacy scores between obese and non-obese participants. It was expected that participants would demonstrate significant increases in self-efficacy from pre- to post-treatment, and this finding supports that hypothesis (2). It was expected that these increases would vary according to weight status, and this finding failed to support that hypothesis (3). Lastly, it was expected that obese and non-obese groups would differ in average self-efficacy, and this finding failed to support that hypothesis (7) (see Figure 5).

An independent-samples *t*-test was conducted to evaluate the hypothesis (12) that non-obese participants would show greater change in self-efficacy scores from pre- to post-treatment than obese participants. The test was not significant, $t(219) = -.51$, $p = .608$. Obese participants’ Change in Self Efficacy scores ($M = 12.17$, $SD = 12.38$) were comparable with Non-obese participants scores ($M = 11.32$, $SD = 11.94$). The 95% confidence interval for the difference in means ranged from -4.09 to 4.35. The eta square effect size was very small ($\eta^2 = .001$), indicating that .1% of the variability in change in self-efficacy was accounted for by whether or not the participant was obese. Based on these results, obese participants’ Change in Self Efficacy from pre to post-treatment did not differ from those of Non-obese participants, and thus this hypothesis was not supported.
Figure 5. Reductions in Pain-related self-efficacy scores from pre to post-treatment between Obese and Non-obese participants.

As noted, the mean Change in Self-efficacy was approximately 11 points ($M = 11.69$, $SD = 12.11$, 95% CI [10.08, 13.29]), with change in self-efficacy ratings ranging from a decrease of 13 points to an increase of 49 points. The most frequent change was 0.00 ($n = 35$, 15.8%), and 34.8% ($n = 74$) reported changes of 5 points or less. 111 participants (52.5%) reported an increase in PSEQ scores of 10 points or more, while 5 participants (2.3%) reported their self-efficacy had decreased by 10 points or more. At intake, approximately 70% of participants ($n = 155$) scored 30 or less on the PSEQ, while only 10% ($n = 22$) scored 40 or above. After treatment, approximately 44% ($n = 97$) of participants scored at 40 or above, while 33% ($n = 73$) remained below the cutoff score of 30.
**Perceived Disability**

A mixed-model ANOVA was conducted to evaluate the effect of treatment and obesity on a measure of perceived disability (PDQ) scores to determine if changes in disability following treatment varied as a function of weight status. Only a subset of participants completed the PDQ as this measure was introduced towards the end of the sampling period \( n = 54, 24\% \).

Participants completed the PDQ, rating their disability on a numerical rating scale of 0 - 150 at both pre-treatment and post-treatment; these scores were grouped according to the participants’ weight status, *Obese* and *Non-Obese*. The dependent variable was participants’ disability scores, while the two factors were *Time* and *Obesity*. An interaction effect \( (\text{Time} \times \text{Obesity}) \) examined whether or not changes in disability scores varied as a function of obesity (Hypothesis 2). A within-subjects main effect for *Time* was examined to see if participants’ mean disability scores changed from pre-treatment to post-treatment while a between subjects main-effect for *Obesity* evaluated whether obese participants had different (lower) mean disability scores across time periods than non-obese participants (Hypothesis 3). The results of Levene’s test for the pretreatment disability score, \( F(1,52) > .01, p = .988 \) and post-treatment disability score \( F(1,52) = .32, p = .574 \) were not significant, indicating that the error variance in the dependent variable was equivalent across groups. Box’s Test for Equality of Covariance Matrices was not significant \( (p = .706) \), therefore homogeneity of variance-covariance was assumed.

The *Time x Obesity* interaction effect was not significant, Wilks’ \( \Lambda = .99, F(1, 52) = .53, p = .471, \) partial \( \eta^2 = .010 \), indicating that 1% of the variance could be accounted for by an interaction between time and obesity, a very small effect size. The *Time* main effect was significant, Wilks’ \( \Lambda = .62, F(1, 52) = 31.34, p < .001 \), partial \( \eta^2 = .37 \), with both groups showing reductions in disability, and indicating that 37% of the variance could be accounted for
by time, a large effect size. The results indicated participants showed significant reductions from pre- to post-treatment on disability scores ($M = -13.67, SE = 2.44, 95\% CI [-18.58, -8.77])$. The main effect comparing groups of participants was not significant, $F(1,52) = .53, p = .471$, partial $\eta^2 = .010$, suggesting little difference in the disability scores between obese and non-obese participants. It was expected that participants would demonstrate significant reductions in disability from pre- to post-treatment, and this finding supports that hypothesis (2). It was expected that these reductions would vary according to weight status, and this finding failed to support that hypothesis (3). Lastly, it was expected that obese and non-obese groups would differ in average perceived disability, and this finding failed to support that hypothesis (8) (see Figure 6).

Figure 6. Reductions in perceived disability scores from pre- to post-treatment between Obese and Non-obese participants.
An independent-samples \( t \)-test was conducted to evaluate the hypothesis (13) that non-obese participants would show greater change in disability scores from pre- to post-treatment than obese participants. The test was not significant, \( t(52) = .73, p = .47 \). Obese participants’ \textit{Change in Disability} scores (\( M = -15.44, SD = 15.56 \)) were comparable with Non-obese participants \textit{Change in Disability} scores (\( M = -11.90, SD = 19.67 \)). The 95% confidence interval for the difference in means ranged from -6.26 to 13.34. The eta square effect size was very small (\( \eta^2 = .01 \)), indicating that less than 1% of the variability in change in disability was accounted for by whether or not the participant was obese. Based on these results, obese participants’ \textit{Change in Disability} from pre to post-treatment did not differ from those of Non-obese participants, and thus this hypothesis was not supported.

As noted the mean reduction in PDQ rating was approximately 14 points (\( M = -13.54, SD = 17.81 \), CI 95% [-18.40, -8.68]), with change in disability ratings ranging from a decrease of 61 points to an increase of 23 points. The most frequent change was 0.00 (\( n = 5 \), 9.3%) and 22% (\( n = 12 \)) reported changes of 5 points or less. However, 35.2% (\( n = 10 \)) patients reported a decrease in PDQ scores of 20 points or more, while only 1 participant (2.3%) reported an increase in perceived disability by 20 points or more.

Though not related to an original hypothesis, PDQ scores for the entire sample were further partitioned into distinct theoretical categories: \textit{Mild/moderate} (scores 0-70); \textit{Severe} (scores 71 – 100), and \textit{Extreme} (scores 101-150), modeled after cut-off scores that have been shown to be predictive of work and health related outcomes (Gatchel et al., 2006). At intake, out of 54 participants, 6 (11.1%) scored within the \textit{Mild/moderate} range, 22 (40.7%) scored as \textit{Severe}, and 26 (48.1%) scored in the \textit{Extreme} range. At discharge, 16 (29.6%) participants
scored within the *Mild/moderate* range, 23 (42.6%) within the *Severe*, and 15 (27.8%) participants continued to score within the *Extreme* range of perceived disability.
### Table 6
*Effects Of Time And Obesity On Multidisciplinary Chronic Pain Management Treatment Outcomes.*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-Treatment</th>
<th>Post Treatment</th>
<th>Main Effect (Time)</th>
<th>Main Effect (Obesity)</th>
<th>Interaction (Time x BMI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>F (df)</td>
<td>p</td>
<td>η²</td>
</tr>
<tr>
<td>Pain</td>
<td>6.35 (1.47)</td>
<td>5.58 (1.63)</td>
<td>51.42 (1,219)</td>
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<td>0.19</td>
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<td>BDI</td>
<td>21.49 (11.07)</td>
<td>14.00 (10.03)</td>
<td>124.72 (1,219)</td>
<td>&lt;.001</td>
<td>0.36</td>
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<tr>
<td>PASS</td>
<td>45.30 (25.13)</td>
<td>33.94 (24.98)</td>
<td>55.14 (1,219)</td>
<td>&lt;.001</td>
<td>0.20</td>
</tr>
<tr>
<td>PSEQ</td>
<td>25.14 (10.99)</td>
<td>36.82 (12.60)</td>
<td>203.56 (1,219)</td>
<td>&lt;.001</td>
<td>0.48</td>
</tr>
<tr>
<td>PDQ</td>
<td>98.46 (18.82)</td>
<td>84.93 (24.35)</td>
<td>31.34 (1,52)</td>
<td>&lt;.001</td>
<td>0.38</td>
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</tbody>
</table>

* Mixed model ANOVA
Discussion

This study sought to examine whether obesity moderated treatment outcomes from a multidisciplinary chronic pain management (MCPM) program. It had previously been shown that obesity predicted treatment outcomes in a cognitive behavioral chronic pain treatment program such that participants with a body mass index (BMI) over 30 did not show improvement comparable with non-obese participants following treatment (Sellinger et al., 2010). By examining pre- and post-treatment outcomes on measures of pain intensity, emotional functioning, ability to self-manage pain, and perceived disability, this study would show if obesity interacted with multidisciplinary treatment in such a way that differential outcomes were observed based on a patient’s BMI.

Results supported the hypothesis that, overall, participants would show significant improvements on measures of pain intensity, depression, anxiety, self-efficacy, and perceived disability following a 20-day MCPM program. Repeated measures ANOVA showed main effects for time on all outcomes, indicating that the MCPM interventions were effective. However, the findings of this study do not support the hypothesis that treatment outcomes would vary according to whether a participant met criteria for obesity or not. More specifically, it appears that obesity did not moderate MCPM treatment outcomes, and obese individuals with chronic pain benefitted from MCPM to the same degree as non-obese individuals on all outcomes. There was no support found for the hypothesis that obesity would predict treatment dropout. Likewise, there was no support found for the hypothesis that obese participants would score significantly worse than non-obese participants on measures of pain intensity, depression, anxiety, self-efficacy, and disability. As there were no significant differences found between obese and non-
obese groups on any measures, these findings suggest that obese participants are able to benefit from MCPM to the same degree as non-obese participants.

It was expected that, consistent with other studies reporting patient BMI (Okifuji et al., 2009; Sellinger et al., 2010; Vincent et al., 2011), obese participants would make up a sizable proportion of the sample of chronic pain patients. Not only did obese (BMI $\geq 30$) participants comprise 45% of the total sample (compared with a 35% US adult average), but healthy weight (BMI $\leq 24.9$) participants made up only 22% of the total sample, in nearly the same proportion (21%) as severely obese (BMI $\geq 35$) participants. Over 78% over the total sample was overweight or obese, compared with the US adult average of 69% (Flegal et al., 2012). Whether this larger proportion is representative of the chronic pain population is unknown as participant BMI is rarely reported in chronic pain literature, however it is similar to the few previous studies that have examined the relationship between chronic pain and obesity. This high rate provides additional support for the growing evidence (Fabricatore & Wadden, 2006; Vaidya, 2006; Wright et al., 2010) of the comorbidity of chronic pain and obesity, and it is likely such percentages are not unusual. The implication is that any possible impact of obesity on chronic pain treatment outcomes could be present for a significant percentage of patients.

Due to the wide range of negative impacts that obesity poses to physical and emotional health (Fabricatore & Wadden, 2006) and consistent with the literature showing greater disability related to comorbid obesity and pain (Doll et al., 2000; Yancy et al., 2002), it was hypothesized that obese patients would present with greater symptom severity than non-obese patients. Furthermore, due to neuroendocrine changes associated with obesity in fibromyalgia patients (Okifuji, 2009) it was considered that a high BMI would function similarly to maintain inflammation and therefore, pain intensity. It was also believed that cardiovascular and muscular deconditioning would prevent obese participant from engaging fully and benefitting from the
exercise and flexibility components of treatment (Rakel & Barr, 2003). Thus, there were several possible paths that might block obese participants from benefitting from treatment to the same degree as non-obese participants.

As expected, this sample of patients reported a wide range of negative biopsychosocial sequelae related to the impact of chronic pain on their health and quality of life beyond that of pain intensity, regardless of their weight status. Consistent with results reported in previous studies (Kerns & Haythornwaite, 1988; McCracken & Turk, 2002; Sellinger et al., 2010), these patients experienced significant disruption and distress in numerous domains, including moderate to severe depression, anxiety, and disability. At intake, 70% of patients reported a profound lack of ability to self-manage their pain or engage in valued activities despite their pain (PSEQ < 30). Unexpectedly, obese and non-obese participants reported similar amounts of dysfunction at intake and discharge, suggesting that the obese participants were not disproportionately burdened due to the added stressor of weight. It seemed possible that the severity in presentation of the overall sample of participants, comprised mainly of workers’ compensation patients, was increased to the degree that healthy weight participants appeared similarly dysfunctional to obese participants and were more severely impaired than the participants in the Sellinger et al. study. However, a comparison between this study’s non-obese participants’ pre-treatment mean BDI scores versus the non-obese participants’ pre-treatment mean BDI scores reported by Sellinger et al. found no significant differences ($p = 0.38$).

Notably, there was a wide variance in treatment response, with some patients reporting changes of over two standard deviations on some measures from pre to post-treatment while a significant number of participants reported little to no change on each of the measures. While there was a significant change in pain intensity from pre- to post- treatment ($p < .001$), the mean change in pain rating was less than one point and the modal change was 0. While the effect size
of variance explained was large, there was little clinically meaningful change in pain intensity, with nearly 60% of participants reporting changes of one point or less. Patients have rated reduction in pain as the most important aspect of treatment for them and identify a minimum of 3 points as meaningful change (Dworkin et al., 2008). Overall, less than 25% of participants experienced pain reductions of 2 points or more, and 50% of participants continued to experience average pain of 6/10 or higher following treatment. These results are consistent with literature showing that there is often little dramatic change in pain intensity associated with MCPM as the focus is upon functional restoration and improved quality of life despite pain rather than elimination of pain; thus dramatic reductions in pain are neither the intent nor expected outcome of treatment (Gatchel & Okifuji, 2006).

It was expected, however, that obese participants would experience more severe pain at treatment intake and discharge based on a previous finding showing a positive correlation between BMI and level of pain in persons with chronic pain (Wood et al., 2001). Thus it was expected that higher rates of pain and less responsiveness to pain treatment would be reflected in highly maintained NRS scores for obese patients. As noted, the entire sample maintained high pain intensity ratings following treatment. Possible explanation for the lack of differences in pain intensity between obese and non-obese participants may be related to the biases in referral to treatment and sample characteristics. A previous study has shown that there is a negative relationship between receiving worker’s compensation benefits and treatment outcomes (Burns et al., 1995) and that patients who are able to identify a specific trauma responsible for their pain report greater pain intensity, emotional distress, and disability (Turk & Okifuji, 1998). This particular sample was comprised almost entirely of worker’s compensation patients (95%), many of whom had been severely injured in industrial accidents. It is possible that the intensity of pain
and disability in this treatment group is more severe and resistant to treatment overall than other chronic pain populations.

A majority (55%) of patients presented themselves with moderate to severe symptoms of depression at intake. As depression is a frequent comorbidity with chronic pain, this high percentage of participants is consistent with the literature and thus rightly targeted by MCPM. A significant portion of patients saw a dramatic drop in depression ratings, with 65% reporting a change of 5 points or more. The remainder of patients, however, saw little to no change in their depression ratings. These patients typically had mild to low-moderate scores on the BDI at intake, and their lack of change likely reflects a “floor” associated with symptoms associated with continuing high levels of pain. Some patients, however reported an increase in their rates of depression. These increases may have been associated with extraneous circumstances outside of treatment, but consistent with the literature (Gatchel et al., 2007), may be related to patients’ unhappiness related to the realization of the chronicity of their pain following treatment. Furthermore, many patients engaged in worker’s compensation rehabilitation programs are faced with difficult life circumstances upon treatment completion as they continued to face unemployment, the closure of their worker’s compensation claim, an uncertain future with regards to their occupation and career, and continuing difficulties with spouses, families, and employers (Burns et al., 1995). It may be there is also a certain “floor” to the reduction in BDI scores in such circumstances.

Pain related anxiety was measured by the Pain Anxiety Symptoms Scale, a well-validated measure that captures the catastrophization and fear-avoidance behaviors that characterize many chronic pain patients. As fear-avoidance behaviors have been found to also additionally impact obese individuals with chronic pain, presenting a barrier to physical activity, it was considered that obese patients with pain would have greater difficulty engaging with the physical modalities
of MCPM, including exercise, flexibility, and behavioral activation. Overall patients scored in approximately the 45th percentile range of PASS scores, indicating that fear-avoidance and catastrophizing cognitions played a low to moderate role participants’ pain. Nearly 50% of patients reduced their scores by 10 percentile points or more, indicating a robust response to treatment.

On the PSEQ, scores of 30 or below are associated with low probability of benefitting from treatment and maintaining treatment gains, and greater likelihood of ongoing disability; scores of 40 or higher are associated with greater benefit from treatment and maintained treatment gains at 6-12 months. As noted, following treatment there was a significant increase (from 10% to 44%) in the percentage of participants scoring greater than the cutoff of 40/60 on the PSEQ, a measure of an individual’s self-efficacy in their ability to engage in valued behaviors in spite of ongoing pain. Scores above 40 have been associated with maintained treatment gains at 6-12 months (Nicholas, 2007). Despite a very large effect size ($\eta^2_p = .48$), indicating that treatment accounted for 48% of the variability in scores (Nandy, 2012), a significant percentage of participants showed little change on this measure, with nearly 35% reporting changes of 5 points or less. Again it is possible that the heavily skewed sample of worker’s compensation patients may provide explanation for the lack of response.

Perceived disability was evaluated with the Pain Disability Questionnaire (PDQ), although this was only given to a small subset of patients ($n = 54$) towards the end of the sampling period. This measure provided a great deal of useful, possibly predictive information that is valuable particularly in light of the lack of physical outcome measures included within this study. Relapse is a common problem in chronic pain treatment (Turk & Burwinkle, 2005) and the PDQ, perhaps more than any other measure, has been shown to be highly correlated with return to work, stay at work, and healthcare utilization rates at one year (Gatchel et al., 2006).
This outcome is highly important to the third party referral sources (worker’s compensation) whose primary goal is to see their patients return to work. Furthermore, with treatment scores correlated with patient outcomes at one year, the PDQ may be useful for consideration in this data set, which is lacking in follow-up measures with participants.

While the mean reduction in PDQ rating was approximately 13 points, changes ranged from a decrease of 61 points to an increase of 23 points. However, as with many other measures, the modal change was 0. When PDQ scores were partitioned into previously determined theoretical categories (Gatchel et al., 2006), the number of participants scoring in the Extreme disability range was reduced by approximately 20% following treatment. Overall, however, approximately 70% of participants remained in the Severe or Extreme disability categories at discharge suggesting a significant majority of participants continued to view their pain as presenting a significant negative impact on their ability to function in everyday life.

It was expected that obese participants would show differential treatment outcomes when compared with non-obese participants as greater rates of self-report disability have been reported in obese patients with chronic pain when compared with non-obese (Vincent et al., 2011). As the PDQ captures patients’ perceptions around their ability to engage in activities of daily living and physical activity, it provided this study with possible indicator of (subjectively reported) physical outcomes, which may be more reactive to obesity based on previous results primarily found on self reported physical health measures (Sellinger et al., 2010). However, there was no differential treatment response by obese participants. The lack of an interaction effect on this particular measure may indicate that obese participants benefitted from MCPM similarly to non-obese participants on measures of physical health, which appears to contradict previous findings by Sellinger et al.
In this study, obese participants showed similar improvements on measures of depression (BDI), pain related fear and anxiety (PASS), pain intensity (NRS), ability to self-manage pain (PSEQ), and perceived disability (PDQ) as healthy weight individuals. Though these findings appear to conflict with a previous study showing that obese participants did not benefit as much as non-obese subjects from cognitive behavioral treatment for chronic pain, the absence of significant differences on measures of emotional functioning may be consistent with previous findings by Sellinger et al. (2010) and Doll et al. (2000), both of whom found significant differences on the physical measures of the SF-36 subscales, but not the emotional subscales. However, in the previous study, the BDI was used as an additional measure of emotional functioning and did show differential treatment outcomes unlike this study. While this limitation might have been predictable based on these previous findings, it was considered likely that the BDI, PASS, and PSEQ were more extensive instruments that captured a more complete picture of each construct, and thus might be more sensitive to differential treatment outcomes. Nevertheless, in a sample of chronic pain patients that completed a 20-day MCPM program, there were significant improvements seen in five different outcome variables, and most importantly, these improvements were seen regardless of obesity.

Limitations to Study and Suggestions for Future Research

There were several limitations to this study. Though the overall results of this program were positive, as the treatment components were not precisely defined nor uniformly implemented, it is impossible to ascertain the mechanism of change, the essential components, or how the treatment was conducted. Furthermore, there were no control group data with which to compare treatment outcomes. Thus, the internal and external validity are questionable and generalizing the results of this program to other MCPM programs is unwise (e.g., Turk, Rudy, & Sorkin, 1993). Furthermore, a significant aspect in chronic pain treatment is also relapse. There
were not complete data for follow up evaluations with patients, and the rate of attrition from follow-up is typically quite high. Participant loss to follow-up remains a difficult barrier to assessing treatment outcomes (particularly with treatment drop-outs or those who were disappointed with their treatment outcomes), but remains an essential aspect of measuring the benefits of MCPM.

Also, previous studies have provided evidence of patient subgroups showing differential treatment outcomes. As this particular pain program does not subgroup patients, nor provide assessment measures that could make possible a post-hoc subgrouping, it is possible that uncontrolled patient heterogeneity contributed to the wide variance in group outcome means, reducing the ability of obesity to discriminate between treatment outcomes. Furthermore, treatment outcomes are the result of the interaction between a multitude factors, so a single factor such as obesity may not explain a great deal of the variance in outcome. However, it should previously be noted that in the study previously documenting differentiated treatment effects (Sellinger et al., 2010) there were no treatment outcome differences found on pain intensity scores (NRS) or the Mental Component score of the SF-36V, in which scores also remained relatively static from pre- to post-treatment. With the exception of the PDQ, the measures evaluated on this study were limited to pain intensity, and self-report psychosocial functioning measures, similar to measures on the previous study that were not differentially affected by obesity. While the PDQ does capture outcomes related to physical experience, they are not entirely comparable with the physical component, physical function, physical role, bodily pain, and vitality scores of the SF-36, areas previously found to indicate differentiated treatment outcomes for obese patients. It is possible that the domain of experience captured in this previous study was not adequately captured by the measures employed by this pain management clinic.
A significant critique of MCPM is the lack of presumed treatment fidelity, as it is unknown the extent to which treatment is conducted in a specified and consistent manner (Turk, Rudy, & Sorkin, 1993). With the variance seen in mechanism of injury, degree of disability, and degree of psychopathology seen at intake, it can be difficult to state that patients received uniform treatments, to specify the exact treatment process, or to what comprised the effective components of treatment. Future studies looking at this population will benefit from including a reliable source of objective physical treatment outcome measures related to functional improvement, such as measures of physical endurance and physical health quality of life. It would also be important to gather more sensitive and specific measures of response to treatment rather than “improved/not improved endurance.” The lack of findings may also be related to the focus on psychological self-reports measures, despite the wide range of behaviors and experiences encompassed. It is possible that if there had been appropriate objective physical and self-report physical-health measures, a differential response to treatment may have been detected in these domains.

An important aspect to bear in mind is the fact that workers’ compensation patients are required to attend treatment by their claims adjuster or risk losing their benefits. It is often believed that many of workers’ compensation patients (Burns et al., 1995) are not necessarily motivated to perform at the best of their abilities as this could have a negative impact on any eventual disability amount they are awarded. Considering the number of patients that showed little to no change on psychosocial measures related to treatment, it may be worth considering whether this particular patient population was consistently highly motivated to engage in a highly physical and intensive treatment that required attendance away from home for 20 days, did not focus upon a reduction in pain, and often required participants to reduce or eliminate their current medications. Moreover, as noted, many of these workers’ compensation patients
consider themselves to be at “treatment of last resort” before being declared maximally medically improved, so the amount of clinically significant change may be influenced by this factor.

In considering the applicability of these results to other chronic pain treatment programs, it is noted that this study consisted of a heavily skewed convenience sample (95%) of participants referred by worker’s compensation programs. These patients are often “at the end of the line” of treatment options and likely not representative of chronic pain patients in general. Furthermore, there were few exclusion criteria to the program, thus the participants are a heterogeneous sample with numerous mechanisms of injury including headache, crush injuries, musculoskeletal injury and degeneration, CNS/spinal injury, peripheral neuropathy, arthritis, lower extremity injury, upper extremity injury, etc. Additionally, the sample consisted of a wide range of ages, severity of pain, degree of emotional distress, and disability. Based on these characteristics, it is unlikely that the results could be generalized to other MCPM management programs.

As noted, a significant proportion of patients showed little change on outcome measures (although obesity did not seem to be a significant factor to this), which appears consistent with literature on treatment outcomes with workers’ compensation patients (Burns et al., 1995). As additional research continues to explore the mechanisms of action within MCPM, findings may be able to determine if there are particular treatment components within the program that addressed possible limitation posed by obesity that therefore made it possible for patients to benefit in similar ways. Further analysis should investigate the factors that contribute the most to overall treatment outcome variance to determine if any differences on baseline and patient characteristics can be found that predict the greatest degree of improvement. Lastly, this particular MCPM program might benefit from employing the SF-36 as outcome measure due to
this measure’s frequent use by other chronic pain research programs, thus facilitating comparisons of treatment outcomes in the future.

In summary, the findings from this dissertation indicate that obese and non-obese chronic pain patients experience similar levels of disability and distress in multiple pain-related domains, and show a similar positive response to MCPM. These equivalent responses were found in the domains of pain intensity, depression, fear and anxiety, self-efficacy, and perceived disability, domains that have been previously shown to also be negatively impacted by obesity. The literature makes clear that the comorbidity of obesity and chronic pain not only serve to exacerbate one another, but may, in some instances, present a barrier to treatment. For instance, it has been suggested that obesity may interfere with the physical modalities of chronic pain treatment, including exercise, flexibility, and behavioral activation (Rakel & Barr, 2003). A previous finding (Sellinger et al., 2010) suggested that obese participants showed less improvement on depression scores and measures of physical health quality of life following CBT for chronic pain. While countless previous studies have examined the role of patient characteristics in chronic pain treatment outcomes, few had explored the role of obesity despite the conditions’ common comorbidity. This study addressed this gap in the literature by examining the role of obesity in treatment outcomes of a multidisciplinary chronic pain management (MCPM) program.

While the undifferentiated response to treatment failed to support the proposed hypotheses, it appears that obese participants, even those with BMIs over 40, are able to benefit significantly from MCPM programs. As chronic pain has been shown to present a significant barrier to weight-loss treatment (Mauro et al., 2008; Okifuji et al., 2010), it may be worth in the future investigating whether providing MCPM to obese individuals prior to or concurrent with weight loss treatment would improve engagement and outcomes. Although the relationship
between the experience and treatment of comorbid chronic pain and obesity have not been made fully explicit, it has been proposed that obese patients respond to chronic pain in ways that promote or aggravate their obesity, such as engaging in less activity and increased hedonic eating. It is believed that these behaviors are mediated by an increased fear of movement, less self-efficacy for activity, and a greater perception of disability (Vincent et al, 2011). Many of these factors contribute substantially to the development and maintenance of chronic pain and are targeted specifically by MCPM. As this barrier has been identified, it seems that the results of this study indicate that there are possible ways to increase patient success by incorporating MCPM prior to obesity treatment, as has been encouraged by previous studies in both chronic pain and obesity treatment (Mauro et al., 2008; Sellinger et al., 2011). By addressing this comorbidity directly, not only would physical factors be better addressed, but improvements in emotional functioning, self-efficacy, fear avoidance, and perceived disability could increase patients’ long term success. Lastly, due to the high prevalence of overweight and obesity within chronic pain populations, the field may benefit from additional qualitative information gathered from obese patients so we might better understand the impact of comorbid pain and obesity upon their lives.
References


