PRE-TREATMENT LEVEL OF OPIOID USE AS A PREDICTOR OF

CHRONIC PAIN REHABILITATION OUTCOME

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The completion of this work is dedicated with love and gratitude

to the memory of my best friend,

Lara Kathleen Barnett, M.D.

May 28, 1970 – September 2, 2006

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The current study examines the relationship between pre-treatment opioid use and treatment outcomes among 1,226 chronic pain patients who participated in a functional restoration program. Patients were divided into five categories based on self-reported pre-treatment level of opioid use. Patients received an initial evaluation prior to treatment, which included a physical examination, medical history, disability assessment interview, quantitative functional capacity evaluation, and psychological intake interview. During the initial weeks of treatment, patients consented to and were weaned from all opioid medications. Assessments were repeated at program completion, and a structured telephone interview was conducted at one-year post-treatment to evaluate socioeconomic outcomes. Nearly half of the patients (596/1226) reported opioid use upon admission. Pre-treatment opioid dose, though, was not associated with clinically

significant differences in pre-treatment socioeconomic variables, pain report, self-reported disability, or health-related quality of life. At pre-treatment, only patients taking the highest opioid doses showed greater self-reported depressive symptoms. Clinician-rated depressive symptoms did not differ significantly based on opioid dose. Opioid use was associated with pre-treatment health variables, with patients taking opioids being one and a half times more likely to report a prior work-related injury and a pre-treatment surgery. Higher levels of opioid use were associated with more severe psychopathology, as demonstrated in less desirable MMPI profiles. Contrary to expectation, level of pre-treatment opioid use did not play a significant role in post-treatment outcomes related to gains in physical functioning, pain report, self-reported disability, or health related quality of life. In general, opioid users showed similar gains relative to non-opioid users from completing functional restoration. However, opioid users showed significantly lower work return and work retention rates, and higher rates of new surgery and healthcare utilization at a one-year follow-up. Pre-treatment opioid dose was also inversely related to program completion rates. Results suggest that compared to non-opioid users, patients who discontinue opioid use show similar post-treatment benefits from functional restoration, but poorer socioeconomic outcomes. Thus, level of pre-treatment opioid use could be a useful guide for identifying patients who are at risk, and targeting treatment interventions to improve the likelihood of program completion and positive long-term treatment outcomes.

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LIST OF ABBREVIATIONS

ANCOVA	Analysis of Covariance
ANOVA	Analysis of Variance
APA	American Psychiatric Association
BDI	Beck Depression Inventory
CES-D	Center for Epidemiologic Studies Depression Scale
CI	Confidence Interval
CNP	Chronic Nonmalignant Pain
COX	Cyclooxygenase
DAWN	Drug Abuse Warning Network
DEA	Drug Enforcement Agency
DEF	Defended
DEPR	Depressed
DIST	Distressed
DSM-III-R	Diagnostic and Statistical Manual of Mental Disorders-3rd Edition Revised
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders-4th Edition
F	F Ratio (Test Statistic)
FDA	Food and Drug Administrations
HAM-D	Hamilton Rating Scale for Depression
IPTAs	Intractable Pain Treatment Acts
IRB	Institutional Review Board
JCAHO	Joint Committee on Accreditation of Health Care Organizations
M	Sample Mean
MANOVA	Multivariate Analysis of Variance
MHE	Mental Health Evaluation
MMPI	Minnesota Multiphasic Personality Inventory
MVAS	Million Visual Analog Scale
N	Total Number in Sample
n	Number in Subsample

NDIC	National Drug Intelligence Center
NHSDA	National Household Survey on Drug Abuse
NSAID	Non-steroidal Anti-inflammatory Drug
OR	Odds Ratio
OSW	Oswestry Disability Questionnaire
PMQ	Pain Medication Questionnaire
PRIDE	Productive Rehabilitation Institute of Dallas for Ergonomics
QPD	Quantified Pain Drawing
SD	Standard Deviation
SF-36	Short-Form 36
SF-36MHS	Short-Form 36 Mental Health Scale
SF-36PHS	Short-Form 36 Physical Health Scale
SOAPP	Screener and Opioid Assessment for Patients with Pain
SOM	Somatoform
SSDI	Social Security Disability Insurance
SSI	Supplemental Security Income
WAIS-R	Wechsler Adult Intelligence Scale-Revised
χ^2	Chi-square

CHAPTER ONE

Introduction

Chronic pain is a major public health problem in the United States today. An estimated one-third of the population experiences some form of chronic pain, to varying degrees of intensity, which exacts a serious socioeconomic toll in terms of personal suffering, interpersonal distress, and widespread financial losses (Miotto, Compton, Ling, & Conolly, 1996). Fifty million Americans are affected, leading to decreased earnings, decreased productivity, and increased health utilization costs and disability benefits (Gatchel & Turk, 1996). Patients with chronic pain are five times more likely than patients without chronic pain to utilize health-care services, and over 80% of physician visits are pain-related (Becker, et al., 1997; Gatchel & Turk, 1996). In 1995, an estimated 2.9 million Americans (1.1% of the population) were treated by healthcare professionals who specialize in the treatment of chronic pain (Marketdata Enterprises, 1995). This estimate does not include individuals treated by primary care physicians, non-pain specialists, complimentary/alternative medicine practitioners, or self-treatment with over-the-counter pain remedies (Turk, 2002). Treatment costs, lost productivity, and social security disability insurance costs attributable to low back pain alone have been calculated at $15 billion to $60 billion annually (Frymoyer, 1991; Frymoyer & Durett, 1997). One study of chronic pain estimated the cost of medical charges and hospitalization to exceed $125 billion annually (Frymoyer & Durett, 1997). Additionally, the impact of chronic pain reaches further than the

individual lives of the sufferer. Chronic pain impacts the lives of significant others and family members, leaving few individuals unaffected by chronic pain at some point in their lives (Turk, 2002). In light of the enormous economic and personal costs associated with chronic pain, the health-care community is faced with the continuing challenge of developing more therapeutically effective and financially efficient treatment modalities.

Recognizing that pain is a major public health problem, the Joint Committee on Accreditation of Health Care Organizations (JCAHO, 2002) introduced new standards for the assessment and treatment of pain. These new standards acknowledge pain as a condition that can co-exist with many injuries and diseases. Under these new standards, pain is treated as a "5th Vital Sign," joining pulse, blood pressure, core temperature, and respiration. Physicians are now required to assess and document pain severity on a pain scale for all patients during each contact. Additionally, JCAHO mandates that health-care organizations comply with several other standards in order to acquire and maintain accreditation. Health-care providers must perform an initial assessment which includes, in the patient's own words, a description of the pain, its location, duration, and its impact on the patient. This initial assessment also includes the patient's pain goal, information on aggravating and alleviating factors, a pain management regimen, and measures of treatment effectiveness. Furthermore, the American Pain Society (2000) has issued a "Pain Care Bill of Rights" that informs patients of the JCAHO standards and their rights to expect proper assessment and treatment of any pain condition. These initiatives have fostered a

new mandate for health-care professionals to sufficiently assess and manage all types of pain (Gatchel, 2001).

Effective treatment of chronic pain conditions must target a host of complex variables that contribute to the experience of pain. Accordingly, interdisciplinary pain management programs have empirically demonstrated therapeutic- and cost-effectiveness in the treatment of chronic pain (Gatchel & Turk, 1999). Regardless of the success of these programs, one common component of interdisciplinary pain management, the use of opioid medications, continues to come under scrutiny. Opioid medications are the most potent and effective analgesics available (Turk, 1996; Polatin & Gajraj, 2002), and their use in the management of acute and malignant pain conditions has long been the accepted standard of care (Portenoy, 1996). However, the use of opioids in the treatment of chronic nonmalignant pain (CNP) is surrounded by controversy due to concerns about the potential for abuse and addiction, and questions regarding long-term effectiveness (Portenoy, 1996; Turk, 1996). Additionally, the debate over the use of opioids for CNP has been heightened by evidence that chronic opioid use alters pain modulatory systems, possibly increasing pain sensitivity and aggravating the underlying pain condition (Covington, 2000; Basbaum, 1992; Mao, Price, & Mayer, 1994; Mao, Sung, Ji, Lim, 2002; Angst & Clark, 2006).

The term “opiophobia,” coined by Morgan in 1986, refers to an irrational degree of fear regarding the addictive potential of opioid medications. Unfortunately, this fear has been reinforced by patient-initiated litigation against

physicians for allegedly fostering opioid addiction (Gatchel, 2001). "Opiophobia" likely results in inadequate treatment of pain for some patients with moderate to severe pain (Sees & Clark, 1993). The immediate suffering of these patients might be compounded by additional consequences of chronic pain and stress, which can impair immune function and enhance tumor growth (Liebeskind, 1991).

Concerns regarding addiction potential, however, appear to be exaggerated. In light of the variability in patient samples and a lack of consensus regarding the definition of addiction, reliable estimates of opioid addiction in chronic pain populations have been difficult to establish (Kirsh, Whitcomb, Donaghy & Passik, 2002; Miotto, et al., 1996; Strain, 2002; Weaver & Schnoll, 2002). Epidemiological data suggest that between 3% and 16% of the American population develop an addictive disorder of some type (Regier, Meyers, & Kramer, 1984). Given the documented risk for individuals with one addictive disorder to develop another (i.e., cross addiction), an estimated 3% to 16% of the population is also at risk of developing an addiction to opioids, especially with long-term use (Savage, 1996). Based on observational data, some researchers have suggested that iatrogenic opioid addiction (physician-induced addiction as a direct result of treatment) is probably rare among patients with no prior history of substance abuse (Portenoy, 1996; Porter & Jick, 1980; Perry & Heindrich, 1982). Several studies of long-term opioid therapy for chronic pain have shown a low risk for addiction in the absence of a prior substance abuse history or a severe

personality disorder (Nedeljkovic, Wasan & Jamison, 2002; Portenoy & Foley, 1986; Strain, 2002).

Other researchers, however, have suggested disproportionately high rates of substance abuse disorders among chronic pain patients (Atkinson, Slater, Patterson, Grant, & Garfin, 1991; Fishbain, Rosomoff, & Rosomoff, 1992; Fishbain, Goldberg, Meagher, Steele, & Rosomoff, 1986; Katon, Egan & Miller, 1985). According to Savage (2002), reviews of substance use disorders in chronic pain patients have concluded that these patients show higher than expected rates of these disorders compared to the general public. In a sample of patients with chronic disabling occupational spinal disorders, nearly 11% of patients met criteria for substance abuse or dependence (excluding alcohol) in the past month (Dersh, Gatchel, Mayer, Polatin, & Temple, 2006). More specific data on the rate of iatrogenic opioid addiction, however, are not yet available due to the relatively small number of controlled studies examining long-term opioid treatment for chronic pain. Thus, the risk of inducing addiction through opioid treatment regimens cannot be discounted altogether, which underscores the importance of monitoring medication compliance and the need for more long-term studies (Savage, 1996).

Setting aside concerns of iatrogenic addiction, the literature remains unclear whether opioids are effective in treating chronic non-malignant pain, especially over the long-term. Also, whether or not treatment is deemed successful depends largely on one's definition of success. At the center of this

dispute is whether the appropriate goal of treatment is rehabilitation or palliation (Covington, 2000). The literature on pharmacological interventions for chronic pain focuses on pain relief and adverse events, to the near exclusion of functional outcomes (Turk, 2002).

Finally, clinical reports have long supported the possibility that long-term opioid use diminishes an individual's natural physiological capacities to modulate pain (Brodner & Taub, 1978; Finlayson, Maruta & Morse, 1986; Terman & Loeser, 1992). Evidence is mounting that long-term use leads to changes in opioid receptors, as well as, in the serotonin, norepinephrine, dopamine, and GABA neurotransmitter systems (Collin & Cesselin, 1991; Savage, 1993). More recent research indicates that these changes lead to opioid tolerance and opioid-induced hyperalgesia (Mao, Price, & Mayer, 1994; Basbaum, 1992; Lim, Wang, Zeng, Sung, and Mao, 2005). Furthermore, previous exposure to opioids might lead to long-term, cumulative decreases in their efficacy. Researchers suggest that the risk of hyperalgesia might have serious implications for, and limit the clinical utility of opioid therapy in chronic pain (Lim, Wang, Zeng, Sung, and Mao, 2005; Chu, Clark, and Angst, 2006).

CHAPTER TWO

Literature Review

History of Opioid Use

An extract of the poppy flower, *Papaver sominiferum*, opium has been used for recreational and medicinal purposes dating back to the ancient Sumerians in 4000 BCE (Gold & Johnson, 1998). Deriving its name for the Greek word "opos," meaning juice, opium has been valued for its ability to induce sleep, analgesia, and a state of euphoria. Throughout history, the use of this substance has involved the ingestion or smoking of raw opium. In the 16th century, Paracelsus formalized opium's place in medicine by developing "laudanum," a tonic of alcohol and opium used to induce analgesia and sleep. By the end of the 19th century, opioids were prescribed widely in many countries for a variety of physical ailments, including cough, headache, pain, and diarrhea (Savage, 1996). Opium tonics enjoyed widespread popularity among artists and intellectuals, such as Elizabeth Barrett Browning and Samuel Taylor Coleridge. Writer Thomas DeQuincy, who was dependent on opium by the age of 20 after taking laudanum to treat a toothache, described his 20 gram-per-day opium habit in "Confessions of an English Opium Eater" in 1812 (Benedetti & Premuda, 1990). An opium-induced dream served as inspiration for Berlioz's *Symphonie Fantastique* (Robinson, et al., 2000). An 1888 survey of a Boston pharmacy revealed that 15% of all prescriptions and 78% of prescriptions refilled more than three times

contained opium. At that time, one in 400 Americans was believed to be dependent on opium (Benedetti & Premuda, 1990).

Three key events during the late 1800s promoted the use and abuse of opium (Gold & Johnson, 1998). The invention of the hypodermic syringe, the isolation of specific alkaloids from raw opium, and the synthesis of heroin from morphine allowed for the development of purer, more potent drugs that can be administered directly into the blood stream, bypassing digestion and first pass metabolism. By the early 20th century, an estimated 300,000 Americans were abusing opioids, and most of these were Civil War veterans and white-middle class females who were introduced to opium for medicinal purposes (Robinson, et al., 2000; Savage, 1996). Opium addiction peaked again in the 1910s and 1920s among urban immigrants who were seeking relief from the miseries of overcrowding, poverty, and bigotry (Savage, 1996). Because of its association with poverty, the view of addiction as a medical problem shifted to that of a social and criminal problem (Savage, 1996). During the 1960s, social stigmas surrounding illicit drug use had lessened and opioid addiction peaked once again, with heroin-related deaths becoming the leading cause of mortality among 15 to 35 year olds (Strain & Stoller, 1999).

The 1990s brought a problematic increase in the misuse and abuse of prescription opioids. The National Household Survey on Drug Abuse (NHSDA) reported increases in the non-medical use and abuse of prescription opioids in the United States. According to the 2001 incidence data, approximately two million

individuals, aged 12 years and older, reported using a prescription opioid for non-medical purposes for the first time during 2000, a five-fold increase from the 1980s (Zacny, et al., 2003). The National Drug Intelligence Center (NDIC) reported that illegal diversion, distribution, and abuse of oxycodone products, especially OxyContin, represents a serious public health problem in the United States (NDIC, 2001). OxyContin is the longest lasting form of oxycodone available. Paired with its reliable strength and dosage levels, OxyContin 12-hour controlled release makes it a viable substitute for heroin. Many pharmacies have ceased stocking OxyContin, because its street value of approximately one dollar per milligram makes it a target for theft and fraud (Kalb, 2001). The manufacturer of OxyContin discontinued the distribution of its strongest 160 mg tablets, in response to Food and Drug Administrations (FDA) reports detailing the illegal misuse, abuse, and diversion of the substance. Additionally, the manufacturer added a FDA box warning in 2001 outlining the appropriate indications for use, the abuse liability, and emphasizing the targeted patient population (FDA, 2001). Thus, the misuse and abuse of prescription opioids, like OxyContin, have serious implications for the legitimate use of opioids in the treatment of chronic nonmalignant pain, including opiophobia, stigmatization of patients, and inadequate treatment of pain conditions (Zacny, et al., 2003).

Legislation of Opioid Use

Early legislation. Acknowledging the increasing problem of opium abuse, U.S. legislators passed the Federal Harrison Narcotics Act of 1914, which implemented a tax on all opioids and mandated the monitoring of opioid trafficking through careful recording of all transfer points, including dispensing medication to patients (Savage, 1996). In 1919, as a result of decisions in two pivotal cases ("Webb, et al. v. the United States and "The United States v. Doremus"), the Supreme Court ruled that the treatment of addiction fell outside the bailiwick of medicine. As a result, physicians could no longer prescribe opioids in the treatment of opioid addiction legally. Consequently, physicians faced the challenge of differentiating between addiction-related distress and legitimate medical uses before prescribing opioids, under penalty of having their medical licenses limited or revoked. Physicians had no legal provision for using opioids in the treatment of addiction until the 1960s when federal agencies, under recommendation of the American Medical Associate, approved the study of methadone as a maintenance treatment for opioid addiction (Savage, 1996). Under the Kennedy Administration, legislation permitted the opening of methadone clinics for this purpose. Decades later, however, major limitations still exist in the availability of these clinics and the services they provide (Savage, 1996).

Recent federal legislation. The Harrison Narcotic Act was updated in 1970, by the Comprehensive Drug Abuse Prevention and Control Act, which

categorized all opioids and other drugs with abuse potential into five schedules based on abuse potential and medical purpose (Clark & Sees, 1993). Abuse potential is based on risk for both physical and psychological dependence. Within this classification system, abuse potential decreases with each increasing schedule. Schedule I drugs, including cocaine and heroin, have the greatest potential for abuse and no accepted medical use. Schedules II, III, and IV include opioid medications, while schedule V includes those drugs with the lowest abuse potential. While this classification system can be helpful, Clark and Sees (1993) suggest that parameters for drug classification are not an exact science and fail to reflect actual prescribing practices or street demand for any given drug.

Despite shortcomings of the classification system, federal regulations mandate that physicians fulfill several requirements in administering controlled substances. For example, federal code requires that "A prescription for a controlled substance to be effective must be issued for a legitimate medical purpose by an individual practitioner acting in the usual course of his professional practice," (as cited in Clark & Sees, 1993, p. 299). Thus, regardless of the physician's intention, a prescription written for purposes outside the usual course of practice is unlawful. If a practitioner's behavior comes under investigation, the definition of "legitimate medical purpose" is determined by expert witnesses acting on behalf of the government. Furthermore, federal legislation directs that a physician before administering opioids, must attempt to determine if the patient is already an addict. Federal law defines an addict as, "any individual who

habitually uses any narcotic drug so as to endanger the public moral, health, safety, or welfare, or who is so far addicted to the use of narcotic drugs as to have lost the power of self-control with reference to his addiction" (as cited in Clark & Sees, 1993, p. 299). Aside from being circular, this definition has received criticism for being too broad to assist practitioners in decision-making, leaving well-intentioned physicians vulnerable to civil and criminal charges. Also, the definition inadequately distinguishes between physical and psychological forms of abuse and dependence (Joranson, 1990). A major criticism of the definition is the inclusion of physical dependency, which is an expected and predictable outcome of long-term opioid treatment (Savage, 2002). According to Savage (1996), these laws have fostered an environment in which many physicians treating pain conditions are excessively fearful of prescribing opioids and patients with legitimate needs may be inadequately treated or undeservingly labeled as addicts.

Intractable pain treatment acts. In a united effort to address physicians' concerns and to protect the needs of chronic pain patients, Texas and California passed legislation, called "intractable pain treatment acts" (IPTAs), to shield physicians who prescribe controlled substances in the treatment of intractable pain (Clark & Sees, 1993). The legal definition of intractable pain is that which cannot be removed or cured through reasonable and accepted medical practices. IPTAs grant physicians prescribing opioids to patients with intractable pain, immunity from disciplinary action by state medical boards. The protection granted by

IPTAs is significant because the Drug Enforcement Agency (DEA) does not automatically permit physicians to use opioids in the treatment of intractable pain, particularly in outpatient settings. However, there are exceptions in two cases to the protection provided by the intractable pain statutes: 1) when a physician is treating a patient for chemical dependency; and 2) when a physician knows a patient is using drugs for non-therapeutic purposes. Consequently, if a legal inquiry is made into the appropriateness of a patient's opioid prescription, the prescribing physician who knows her patient is smoking marijuana may not be protected by the intractable pain law. Additionally, physicians in certain states, including Texas, are not protected legally if they fail to maintain complete and accurate records of their dispensing of controlled substances (Clark & Sees, 1993). Although no legislation prohibits the use of opioids in treating known addicts for genuine medical conditions, the physician bears the responsibility of establishing and demonstrating the legitimacy of the medical condition and the appropriateness of the treatment plan (Adams, 2004). This process is complicated by the subjective nature of pain and unclear etiologies of some pain disorders, making it difficult to differentiate legitimate complaints of pain from distress signals of opioid addiction (Savage, 1996).

Clinical Pharmacology of Opioids

Opioid medications produce analgesic effects through their binding action with opioid receptors located throughout the central nervous system, with higher concentrations in the limbic system, thalamus, striatum, hypothalamus, midbrain, and spinal cord (King & Miller, 1998). Opioid medications in use today act predominately through agonistic action on the μ-opioid receptor, but most also act on the kappa- and delta-opioid receptors (Brookoff, 2000). When stimulated, peripheral opioid receptors in primary afferent nociceptive neurons may increase the pain threshold and inhibit the release of pain-producing inflammatory substances from these neurons (Polatin & Gajraj, 2001). Some opioid receptors also have stimulatory functions that may be responsible for some adverse side effects. Animal studies suggest that the reinforcing properties of opioids associated with addiction (i.e., euphoria) are mediated by the mesolimbic dopamine system, and appear to be distinct from the analgesic properties mediated by supraspinal spinal system (Gutstein & Akil, 2001). Opioid analgesics are divided into three classes based on their interactions with receptors: 1) pure agonists; 2) partial agonists; and 3) mixed agonist-antagonists (Bannwarth, 1999). Partial and mixed classes of opioids are characterized by a ceiling effect for their analgesic properties, despite further dosage increases. Pure agonists bind primarily to μ-opioid receptor and with continued dosage increases, produce increasingly higher levels of analgesia until, theoretically, complete analgesia is achieved. In practice, however, side effects experienced at higher

doses limit the analgesic effects of pure agonists. For example, codeine is classified as a pure agonist; however, its analgesic effectiveness is limited by adverse side effects that manifest at high doses (Bannwarth, 1999, Inturrisi, 2002).

For clinical purposes, opioid medications are typically classified as either "weak" or "strong," based on the intensity of pain for which they are effective. Codeine, dihydrocodeine, and tramadol are considered "weak" opioids and are typically prescribed for moderate pain. Morphine, methadone, oxycodone, hydrocodone, and pentazocine are considered "strong" opioids and are usually prescribed for severe pain, or for moderate pain, in low doses (Bannwarth, 1999). Opioids with shorter half-lives, such as meperidine, are usually not recommended for the treatment of chronic pain because they require frequent dosing. Furthermore, short-acting opioids are believed to pose a higher risk for abuse because of the initial "high" or state of euphoria associated with administration (Bannwarth, 1999).

Although the mechanisms are not well-understood, individual differences exist in response to equivalent doses of opioids (Bannwarth, 1999; Brookoff, 2000). Individuals can vary widely in how they metabolize opioids, and differences in response, may also be determined by genetic factors or influenced by interactions with other drugs. Gender differences in response also exist, with opioids that act extensively on the kappa-opioid receptor having a greater analgesic effect in women than in men (Brookoff, 2000). Treatment guidelines

recommend that the choice of analgesic proceed from a weaker to a stronger agent. Thus, initial treatment might begin with a non-opioid analgesic, such as a non-steroidal anti-inflammatory drug (NSAID). If the response is inadequate, treatment might progress to a weak opioid and, if necessary, a strong opioid (Polatin & Gajraj, 2001).

Non-opioid analgesics. In addition to opioids, two broad categories of medication, with different mechanisms of action, are available to physicians for pain management (Portenoy, 2000). The first category includes aspirin, acetaminophen, and the NSAIDs. The major analgesic effect of these medications is at the site of pain. Although these medications have a ceiling effect for their analgesic properties, continued increases in dose may increase the duration of pain relief (Polatin & Gajraj, 2001). Their mechanism of action involves the inhibition of cyclooxygenase (COX), which has two main variants. COX-1 is required for normal physiological functioning of the stomach, kidneys, and platelets. COX-2 is involved in inflammation.

NSAIDs do not produce the physical dependence or tolerance found with opioids, but they are not without risk of potentially serious side effects. For example, individuals on high doses of NSAIDs are at risk for massive gastrointestinal hemorrhage. In hopes of minimizing this risk, newer NSAIDs target the COX-2 variant, while limiting the action on COX-1, thus decreasing the risk for gastrointestinal effects. These COX-2 selective NSAIDS are more expensive, however, so physicians must identify patients at risk for toxicity and

contrast the benefit of decreased risk of side effects with the additional cost (Portenoy, 2000). Still, use of these medications is not without risk. In September, 2004, pharmaceutical company Merck announced the voluntary worldwide withdrawal of Vioxx (rofecoxib) from the market after the data safety and monitoring board overseeing a long-term study of the drug found increased risk of serious cardiovascular events, including heart attacks and strokes among patients taking Vioxx compared to placebo. The U.S. Food and Drug Administration concluded that both COX-2 selective and non-selective NSAIDS have similar cardiovascular risk profiles (Merck & Co., Inc., 2004; FDA Consumer magazine, 2004).

Adjuvant analgesics. "Adjuvant analgesics" represent the second broad category of alternative analgesics. Adjuvant analgesics include an array of medications that have primary indications for something other than pain, which may function as analgesics under specific circumstances (Portenoy, 2000). Physicians have long recognized that antidepressants, for example, are potentially efficacious in the treatment of various kinds of chronic pain, including headache, lower back pain, and cancer pain. Neuroleptics, anticonvulsants, and GABA agonists are prescribed to treat neuropathic pain, while muscle relaxants and certain benzodiazepines are given for musculoskeletal pain (Portenoy, 2000; Polatin & Garjaj, 2002). Like opioid and non-opioid analgesics, these "adjuvant analgesics" have potential risks that physicians must weigh against their possible benefits when developing a medication regimen (Adams, 2004).

Physician Attitudes and Beliefs about Opioid Use

Opiophobia. Given the availability of alternative analgesics, some physicians prefer to avoid the perceived risks associated with prescribing opioids for the treatment of chronic pain. Clark and Sees (1993) reported that some physicians believe that patients with a chronic pain condition with no known or identifiable etiology should never be prescribed opioids. A 1990 survey of Minnesota physicians supported the notion of opiophobia. Nearly one-third of the physicians in the survey were generally unwilling to prescribe opioids for the management of chronic pain. Furthermore, one-third of internists and one-fourth of family practitioners reported they were unlikely to accept new patients with pre-existing prescriptions for controlled substances (Minnesota Medical Association, as cited in Clark & Sees, 1993).

A more recent survey of 386 physicians, representing 234 counties throughout the State of Texas, highlights the enduring quality of this skepticism (Weinstein, et al., 2000). Weinstein and colleagues reported that a significant number of respondents endorsed "opiophobia," defined as a prejudice against the use of opioids in the treatment of pain. This bias against opioids entailed limited knowledge about pain and its treatment, negative views of chronic pain patients, and fears regarding potential legal ramifications. More specifically, 42% of respondents endorsed the belief that addiction is a common result of opioid

treatments for pain, and 54% believed that psychological addiction is a common consequence of legitimate opioid prescriptions. Furthermore, 26% of responding physicians thought it likely that prescribing opioids for chronic pain would trigger a drug enforcement agency investigation, and 24% admitted limiting their prescribing of opioids to avoid such investigation. The survey found that "opiophobia" is more common in small communities, and among physicians specializing in surgery and anesthesiology. To the contrary, psychiatrists held more favorable attitudes about chronic pain patients and had fewer reservations about prescribing opioids. The survey raised concerns that "opiophobia" contributes to the under-management and mismanagement of pain, and called for increased educational strategies to reverse these attitudes (Weinstein, et al., 2000).

Support among pain specialists. Specialists in pain management appear to have more positive attitudes regarding the use of opioids in the treatment of CNP and are less restricted by fears, than are specialists in other fields of medicine. The American Pain Society, in a survey of 100 physician members, found that only 13% reported having no patients for whom they prescribed chronic opioid therapy, while 6% reported having more than 50 patients on long-term opioid therapy. Some respondents expressed concerns about tolerance and physical dependence, but respondents did not believe that fear of regulatory pressure significantly impacted their prescribing practices. However, respondents tended to agree that addiction was overemphasized, and that opioids were under-utilized in the treatment of patients with noncancer pain (Turk & Brody, 1992).

Therapeutic Effectiveness of Opioids

Numerous studies have supported the utility of opioids in the treatment of acute and cancer pain patients (Zech, Grond, Lynch, Hertel, & Lehmann, 1995; Portenoy, 1996; Nedeljkovic, et al., 2002). These studies indicate that opioid therapy is effective in relieving pain in 70-90% of cancer patients, and results in increased functioning and improved quality of life (Jorgensen, Mortensen, Jensen, & Eriksen, 1990; American Pain Society, 1992). The success of opioids in the treatment of acute and cancer-related pain has fostered optimism, and has lead to the exploration of the use of these substances for the treatment of CNP. However, use in this population is surrounded by controversy due to concerns about physical dependence, psychological dependence, addiction, tolerance, risk of litigation, adverse side effects, and long-term therapeutic efficacy (Turk, 1996). Many physicians believe that the potential risks outweigh benefits, but a growing contingent of the medical community believes that many CNP patients may respond well to long-term treatment with opioid medications (Portenoy, 1996; Nedeljkovic, et al., 2002; Savage, 2002).

Uncontrolled studies. Much of the literature on the efficacy of opioid therapy with CNP patients has come from uncontrolled studies. Many early studies support the effectiveness of opioids, while dispelling concerns about the risk of addiction and adverse side effects (Taub, 1982; Jamison, Anderson,

Petters-Asdourian, & Ferrante, 1994; Urban, France, Steinberger, Scott, & Maltbie, 1986; Green & Coyle, 1989). In one of the earliest published studies, Taub (1982) reviewed the medical charts of 313 CNP patients who were treated with opioids on a long-term basis. Patients had been receiving average daily doses of opioid medications equivalent to 10-20 mg of oral methadone. In the author's judgement, overall efficacy of treatment was good. Only, 13 (4%) of the 313 patients were identified as abusing their opioid medication, and of these 13 patients, 8 had a prior substance abuse history. In a retrospective survey of 112 patients, most of whom had chronic back pain, Jamison and colleagues (1994) found that 83% reported moderate or better pain relief on opioid maintenance. Additionally, 82% reported minimal or no side effects, and 60% reported no or minimal need to increase dose over time. Similarly, two small case reports offer supporting evidence for the therapeutic efficacy of opioid treatment in chronic pain patients. Urban, et al. (1986) treated 5 patients with phantom limb pain who had failed several other treatments. With opioid therapy, four of the five patients reported a 50% reduction in pain. The authors noted no problems with dose escalation, addiction, or side effects. In another small study, Green and Coyle (1989) reported that 5 of 7 CNP patients experienced good or excellent results when treated with methadone.

Beyond pain relief, opioid therapy has been associated with positive outcomes such as decreased health utilization and increased functioning (Tennant & Uelman, 1983; France, Urban, & Keefe, 1984). Over a 2-year period, Tennant

and Uelman (1983) prescribed opioids to 22 chronic pain patients who had failed to benefit from pain clinic programs. Survey data revealed decreases in the number of medical visits and hospitalizations for all patients relative to the beginning of opioid treatment, and 15 (68%) patients were able to return to work. France and colleagues (1984) gathered survey data from a sample of 16 chronic pain patients who received opioid medication as part of an inpatient interdisciplinary pain treatment program. At discharge, 13 of the 16 patients reported 75-99% relief from pain and the remaining 3 patients reported 50-74% relief. Additionally, 12 (75%) of these patients showed increases in their activity level or returned to work. The authors noted, however, that 25% of patients reported marked decreases and 38% reported slight decreases in the effectiveness of opioid medications several months after initial treatment.

Still, the results of these early uncontrolled studies have not been uniformly positive. Tennant and colleagues (1988) surveyed 52 CNP patients who were treated with various opioids at doses ranging from the equivalent of 10-240 mg of methadone. Although treatment duration was unspecified, average opioid use was greater than 12 years. The authors found that 88% of these patients reported adequate pain relief and 12% reported partial pain relief, without dosage elevations. Unfortunately, 17% of patients in the study evidenced abuse behaviors. Patients also experienced significant side effects including constipation, edema, and adrenal insufficiency.

Other studies have failed to demonstrate adequate pain relief with opioid therapy, and have yielded mixed results in terms of other outcomes (Portenoy & Foley, 1986; Zenz, Strumpf, & Tryba, 1992). Portenoy & Foley (1986) published outcome data for 38 chronic pain patients treated with opioids. Reported pain relief was adequate for 11 (29%) patients and partial for 13 (34%) patients. But, pain relief for the remaining 14 (37%) patients was inadequate. Abuse was noted in 2 (5%) patients, both of whom had a history of drug abuse. Zenz and colleagues (1992) monitored 100 patients receiving long-term opioid therapy for CNP. Over a 7-month average course of treatment, 51 patients reported good pain relief and 28 patients reported partial pain relief. These patients also reported significant increases in functional ability. However, opioid therapy was discontinued in 10 patients due to medication non-compliance and in 21 patients due to declines in physical functioning. An additional 20 patients were switched to alternative therapies. Nine patients showed abuse behaviors, and 33 patients experienced side effects including constipation, edema, and adrenal insufficiency.

Survey studies have suggested that long-term opioid treatment may provide significant pain relief for some CNP patients (Nedeljkovic, et al., 2002). However, a number of these studies have failed to demonstrate adequate pain control for large proportions of patients (Portenoy & Foley, 1986; Zenz, et al., 1992). Results for other outcomes were likewise mixed. Evidence of abuse behaviors ranged from 4% (Taub, 1982) to 17% (Tennant, et al., 1988). Several studies reported improvements in activity level and return to work (Tennant &

Uelman, 1983; France, et al., 1984). Zenz and colleagues (1992) reported improvements in functional ability for 51 patients, but 21 patients in the same study showed declines in physical functioning. Several survey studies documented significant side effects, including constipation, edema, and adrenal insufficiency.

Controlled studies. A growing number of controlled studies have investigated the efficacy of opioids in treating CNP, many with positive outcomes (Arkinstall, et al., 1995; Watson & Babul, 1998; Moulin, et al., 1996). One of the first randomized studies, conducted by Arkinstall and colleagues (1995), compared controlled-release codeine to placebo in a sample of patients with mostly rheumatic and back pain. Thirty chronic pain patients completed a 7-day protocol and the codeine treatment group reported significantly lower overall pain intensity scores than the placebo control group. In another randomized, placebo-controlled study, Watson and Babul (1998) found controlled-release oxycodone to be superior to placebo in reducing pain intensity among a sample of elderly chronic pain patients. Moulin and colleagues (1996) reported that oral morphine significantly decreased pain relative to placebo among patients with chronic pain. While the morphine treatment group showed greater pain control than the placebo group, similar improvements in psychological functioning and quality of life were not found.

Several studies comparing patients taking opioids to patients not taking opioids have yielded mixed outcomes on a variety of measures. From a tertiary

care multidisciplinary pain program, Harden and colleagues (1997) randomly selected 100 patients who were taking daily opioids and matched them to 100 patients who were not taking opioids. The two groups did not differ in pain type, duration, location, or surgical history. No significant differences were found in pain, psychological status, or functional measures (Harden, Bruehl, Siegler, & Cole, 1997). In a telephone survey of 89 patients with CNP who attended either a university pain or rehabilitation clinic, Adams (2003) found that 63% consistently used opioid and 37% did not use opioids. A comparison of the two groups revealed that patients on opioid therapy had significantly more bodily pain, more physical dysfunction, and greater role limitations. Opioid users also reported more severe life-interfering chronic pain and lower quality of life. However, the opioid users and non-users did not differ in Mental Component Summary scores on the SF-36, nor did they differ in rates of full-time work or school.

Other studies have compared varying dosages of opioids on several outcome measures, in addition to pain relief (Rowbotham, Twilling, Davies, Reisner, Taylor, & Mohr, 1996; Jamison, Raymond, Slawsby, Nedeljkovic, & Katz, 1998). Rowbotham and colleagues (1996) randomized a cohort of 81 chronic neuropathic pain patients to either low-strength (0.15 mg) or high-strength (0.75 mg) levorphanol. Dose was titrated by the patient to a maximum of 21 capsules per day. The low-strength group averaged 2.7 mg per day, while the high-strength group averaged 8.9 mg per day. Both groups showed improvements in sleep, functioning, and level of affective distress. The authors concluded that

higher doses of opioids lead to significantly greater reduction in pain intensity, but higher doses produced more side effects without significant additional benefit in terms of other outcomes.

In a randomized study, Jamison and colleagues (1998) compared an NSAID (naproxen) to two opioid regimens, the first involving a set-dose of oxycodone, and the second involving titrated oxycodone and morphine. A total of 36 chronic back pain patients completed the 1-year protocol, with periodic monitoring of pain relief, activity levels, and sleep. Results of the study indicated that patients in the titrated opioid regimen experienced significantly less pain and emotional distress relative to patients in the other two regimens. Further, patients in the set-dose oxycodone regimen reported significantly less pain and emotional distress than did those in the NSAID regimen. Few differences were found in activity levels and quality of sleep among the three regimens, and only one patient demonstrated behavior consistent with abuse. In a Danish study, Frei and colleagues compared transdermal fenatanyl to oral sustained-release morphine in outpatients with CNP. Effectiveness of treatment was measured in days of good pain control and days on initial treatment. Researchers also examined the costs associated with treatment including breakthrough pain, co-medication costs, and control of adverse events. Fentanyl was more effective in terms of total days of good pain control. However, patients remained on initial treatment longer with fentanyl than morphine, and fentanyl cost US $10.26 more per extra day of good pain control (Frei, Anderson, Hole, & Jensen, 2003).

While the studies discussed above examine the effectiveness of opioid therapy in the treatment of CNP, and whether its benefits outweigh the possible risks, to date only two studies examine the role of pre-treatment opioid use in treatment outcomes. In the first study, researchers examined 127 patients with on-the-job injuries who completed a multidisciplinary rehabilitation program for chronic pain. Researchers compared pre-treatment scores and post-treatment outcomes of patients taking opioids to patients not taking opioids, based on self-report at both admission and discharge. Opioid treatment was administered by physicians who were unaffiliated with the program. Both opioid users and nonusers showed improvements in psychological and physical measures, and post-treatment outcomes did not differ between the two groups. Furthermore, at 6-months post-treatment opioid users did not show significantly different return to work rates relative to opioid nonusers (MacLaren, Gross, Sperry, & Boggess, 2006). Patients in the study, however, were not weaned from opioids during the course of treatment and the results included completers only for both pre-treatment and post-treatment findings.

In the second study, researchers at the Mayo Comprehensive Pain Rehabilitation Center (Rome, Townsend, Bruce, Sletten, Luedtke, & Hodgson, 2004) compared patients taking opioids daily (n=135) to patients not taking opioids daily (n=221) at the time of admission. Over the course of the 3-week intensive multidisciplinary outpatient rehabilitation program, opioid use was tapered and discontinued. Based on a cognitive-behavioral model with the goal of

functional restoration, the rehabilitation program included physical reconditioning, relaxation training and biofeedback, stress management, pain management training, chemical health education, and occupational therapy. Patients presented with a variety of pain disorders, but fibromyalgia, low back pain, and chronic headaches were the most frequent, accounting for over half of the study sample. Researchers found that low back pain was more common among the opioid users, while fibromyalgia was more common among the non-opioid users. Program completion rates did not differ significantly between opioid users and non-opioid users. However, program non-completers were taking significantly higher opioid doses at admission than program completers. At post-treatment, opioid users did not differ significantly from non-opioid users on measures of interference due to pain, perceived life control, affective distress, depression, or general activity level. Opioid users, however, did report significantly greater pain severity and catastrophizing than non-opioid users. Researchers concluded that concurrent opioid withdrawal was not a barrier to successful pain rehabilitation, in terms of initial outcomes. The study, however, is limited by its lack of long-term treatment outcomes, such as rates of work return, work retention, and healthcare utilization (Rome, Townsend, Bruce, Sletten, Luedtke, & Hodgson, 2004).

Acknowledging the difficulty of drawing firm conclusions from this body of research, Turk (1996) outlined several factors that contribute to the problem. The first factor is the shortage of double-blind, randomized controls. In some

studies, treatment efficacy was rated retrospectively. In the majority of studies, clinicians providing the treatment also evaluated the treatment outcome, introducing a potential source of bias. Another factor is the definition of treatment success and the subsequent choice of outcome measures. Many studies relied solely on self-reported pain reduction as the criterion for success, however functional outcomes such as return to work must also be considered. Finally, the studies included heterogeneous samples of patients, mixed diagnoses, and a wide variety of medications and dosages.

Risks of Opioid Use

The critical question a physician must face in deciding to use opioid therapies for chronic pain patients is whether or not the benefits of such treatment will outweigh the risks, for any given patient (Bannwarth, 1999; Jamison, et al., 1998; Savage 1996). Thus, physicians must concern themselves with "opioid responsiveness," the term used by researchers to describe the satisfactory relief of pain without intolerable side effects or negative outcomes (Portenoy, Foley & Inturrisi, 1990). With "opioid responsiveness" as the frame of reference, the physician must think beyond the opioid treatment's capacity for analgesia, and evaluate the treatment's impact on other therapeutic outcomes, such as functional abilities, psychological well-being, and avoidance of addiction.

Side effects. A number of potential side effects associated with opioid use can interfere with analgesic effects and functional capacity. The bodily systems in which adverse effects typically manifest include: (a) the gastrointestinal tract, producing nausea, vomiting, and constipation; (b) the central nervous system, producing sedation, dizziness, cognitive impairment, respiratory depression, and myoclonus; (c) the skin, producing pruritus; and (d) the urinary tract, producing urinary retention (Bannwarth, 1999). The underlying mechanisms of these side effects are not well-understood and probably depend on a variety of factors including age, extent of disease, prior opioid exposure, route of opioid administration, and concurrent medications (Inturrisi, 2002). Many side effects can be treated as they appear, and typically abate as tolerance develops (Cherny, 1996). However, some prevalent side effects are more enduring, such as constipation, to which tolerance develops slowly or not at all. In a controlled study of oral morphine, 40% of patients reported constipation (Moulin, et al., 1996). Due to the prevalence and persistence of this side effect, Cherny (1996) recommends that physicians treat constipation prophylactically whenever strong opioids are administered.

The risk of cognitive compromise is a side effect that warrants serious concern (Portenoy, 1996; Savage, 1999), as many opioid receptors are located in areas of the brain involved in learning, memory, and attention (Chapman, Byas-Smith, & Reed, 2002). Sedation and other cognitive changes that sometimes occur with long-term opioid treatment, may adversely impact cognition and

psychomotor performance, thus placing patients at risk for accidents while driving, working, or engaging in any activity (McNairy, Maruta, Ivnik, Swanson, & Hstrup, 1984). For example, this side effect may contribute significantly to the 60% increased risk of hip fracture found in older adults prescribed codeine or propoxyphene (Shorr, Griffin, Daugherty, & Ray, 1992). In contrast, one review of related data suggests that exceptions may occur, but most significant cognitive and psychomotor changes abate once the patient adjusts to a particular opioid dose (Chapman, et al., 2002; Zacny, 1995). Savage (1999) offered anecdotal evidence that suggests that individuals taking high doses of opioids can still function well in a variety of physically- and mentally-demanding occupations including doctor, police officer, engineer, and construction worker. Differences in response to opioid medications underscore the importance of monitoring and educating patients receiving opioid treatment, and the need for careful titration of medication verses level of pain (Savage, 1999).

Opioid-induced hyperalgesia. In addition to the risk of side effects, long-term use of opioid medications might diminish an individual's natural physiological capacities to modulate pain (Compton, 1994; Savage, 1999; Schofferman, 1993). Animal studies have documented changes in the structure, function, and number of opioid receptors as a consequence of long-term opioid administration (Collin & Cesselin, 1991). Additionally, the sustained use of opioids is known to cause changes in the serotonin, norepinephrine, dopamine, and GABA neurotransmitter systems. As a result, the functioning of endogenous

pain modulatory systems and brain reward mechanisms might be altered (Savage, 1993).

As evidence mounts, researchers and clinicians are increasingly concerned about the potential for a phenomenon termed “opioid-induced hyperalgesia” (OIH). In OIH, opioids administered to alleviate pain, instead, may increase a patient’s sensitivity to pain and aggravate pre-existing pain (Angst & Clark, 2006). This phenomenon has been supported for many years by clinical reports that observed improvements in pain after discontinuing long-term opioid treatment, in the absence of additional treatments (Brodner & Taub, 1978; Finlayson, Maruta & Morse, 1986; Terman & Loeser, 1992). For more than thirty years, researchers have known that the systematic administration of opioids to rodents can lead to hyperalgesia during withdrawal. Early animal studies focused on hyperalgesia as a potential quantitative measure of opioid dependence in the field of addiction. Perhaps influenced by the more restricted use of opioids in the treatment of chronic pain at that time, researchers did not highlight the potential impact of these findings for the practice of pain management (Angst & Clark, 2006).

Animal models indicate that hyperalgesia is associated with increased opioid tolerance (Mao, Price, & Mayer, 1994; Basbaum, 1992). Mao, Sung, Ji, and Lim (2002) investigated the role of spinal glutamate transporters in the development of morphine tolerance and hyperalgesia. These researchers found that chronic morphine administration lead to a dose-dependent downregulation of glutamate transporters in the superficial spinal cord dorsal horn of rats. This

downregulation was temporally correlated with the development of morphine tolerance and thermal hyperalgesia. Researchers concluded that spinal glutamate transporters contribute to the neural mechanisms of morphine tolerance and abnormal pain sensitivity by regulating glutamate homeostasis. Researchers have also found that morphine exposure induces an upregulation of neuronal glucocorticoid recepters within the spinal cord dorsal horn. Furthermore, this upregulation was maintained after morphine was discontinued, and significantly enhanced upon a second course of morphine. Thus, prior morphine exposure seems to have a long-term and cumulative influence over the efficacy of morphine and the development of morphine tolerance (Lim, Wang, Zeng, Sung, and Mao, 2005). These findings may have significant implications for the use of opioid therapy (Lim, Wang, Zeng, Sung, and Mao, 2005).

Despite the potential consequences, until recently, no prospective studies existed to document the development of opioid tolerance and OIH in chronic pain patients. In a preliminary study, researchers evaluated the development of opioid tolerance and OIH in a small sample of patients with chronic low back pain. Using the cold pressor test to measure pain sensitivity, patients were assessed prior to and one month after beginning oral morphine therapy. All patients developed tolerance and hyperalgesia after one month of therapy. Again, researchers suggest that these phenomena limit the clinical utility of opioid therapy in chronic pain (Chu, Clark, and Angst, 2006).

Reinforcing properties of opioids. The reinforcing properties of opioids have been recognized for millennia. Savage (1996) classified opioid

reinforcement into two broad categories. The first category is "primary opioid reinforcement," which describes the maintenance or increased frequency of certain behaviors in pursuit of euphoria, tranquility, and well-being. The other category, "secondary opioid reinforcement," describes the maintenance of certain behaviors intended to avoid the aversive stimuli associated with pain and withdrawal symptoms (Savage, 1996).

In a review of animal studies, Zacny and Walker (1998) reported that rats and monkeys, offered opioids in self-administration paradigms, consistently demonstrated drug-seeking behaviors across a range of reinforcement schedules. An analysis of the relationship between opioid dose and response rate showed an inverted U-shaped function, with lower doses producing lower response rates and higher doses yielding higher response rates. However, response rates did not continue to increase indefinitely with dosage increases. Once opioid dosages increased beyond certain levels, their reinforcing potency diminished, which suggests that continued increases in opioid doses might eventually yield diminishing returns in drug-seeking behavior.

Several studies have demonstrated similar reinforcing properties of opioids in humans. As expected, studies have shown the tendency of known opioid abusers to self-administer opioids if given the opportunity (Fraser, Martin, Wolback, & Isabell, 1961; Fraser & Rosenberg, 1964; Mello, Mendelson, & Kuehnle, 1981). In an early study, Schuster, Smith, and Jaffe (1971) inferred opioid reinforcing potency from the number of return visits to a clinic. Subjects in the study had recently undergone heroin detoxification with methadone

tapering. Subjects were assigned to one of four groups that were allowed to self-administer 40 mg methadone, 400 mg codeine, 400 mg pentazocine, or placebo daily, for 10 days, provided that they returned to the clinic to receive the dose. While visits decreased across all groups over time, results indicated that the pentazocine and placebo groups showed the greatest declines. The methadone and codeine groups showed the smallest declines, suggesting these two drugs have relatively stronger reinforcing potencies.

Given that evidence of drug-seeking behavior among known opioid abusers is not necessarily compelling, one study examined the reinforcing effects of opioids in a sample without a history of drug abuse (Zacny, et al., 1996). Subjects in this study were allowed to self-administer 50 mcg of fentanyl via a subject-controlled analgesia pump during 3 separate trials in which they were required to immerse their non-dominant forearm in water varying in temperature across trials, from lukewarm (37˚C) to painfully cold (2˚C). Results indicated that fentanyl was significantly reinforcing in the painfully cold-water condition, but did not exceed chance in the lukewarm water condition. The researchers concluded that among subjects with no drug-abuse history, the presence of a painful stimulus may mediate the reinforcing potency of 50 mcg of fentanyl. The researchers also commented that additional studies are needed to determine whether pain or other stressors are necessary conditions for opioids to be reinforcing to this population.

Addiction, which is strongly related to the phenomenon of reinforcement, is one of the risks involved in opioid therapies that raises serious concern among

physicians treating chronic pain (Covington, 2000; Adams, 2004). However, confusion surrounds the nature and prevalence of addiction among pain patients treated with opioids due to the lack of an accurate definition of addiction for this population. The Diagnostic and Statistical Manual of Mental Disorders-4th Edition (DSM-IV; American Psychiatric Association, 1994) operationally defines addiction by the processes of physical dependence and tolerance. However, physical dependence and tolerance are expected outcomes of long-term opioid therapy (Savage, 2002). While tolerance and physical dependence may coexist with addiction, they can also exist in the absence of opioid misuse (McCaffrey & Pasero, 1999; Wesson, Ling, & Smith, 1993).

Tolerance, Dependence, and Addiction: Clarification of Terms

Clear definitions are necessary for the effective examination of opioid addiction and misuse. The terms tolerance and physical dependence are often misapplied to the phenomenon of opioid misuse, but tolerance, physical dependence, and addiction are discrete phenomena. This confusion of terms could have serious clinical consequences, such as increasing physicians' reluctance to prescribe opioids, thereby increasing the risk of inadequate treatment and suffering, as well as, unjustly stigmatizing patients who experience common outcomes of opioid treatment (Portenoy, 1996; Savage, 1999; Bannwarth, 1999; Brookoff, 2000).

Tolerance. Tolerance for a medication is suspected if increased doses are necessary to sustain the initial effects of the drug (Bannwarth, 1999; McQuay,

1999; Savage, 1999). While tolerance can be present in addiction, is not necessarily indicative of addiction. Multiple factors may contribute to the development of opioid tolerance. Some clinicians argue that the need for dosage increases is based in disease progression (Twycross, 1974; McQuay, 1999). Others suggest that tolerance has a pharmacological basis that involves changes in the metabolism, distribution, or degradation of the drug, as well as, neuroadaptation at the receptor level (Compton, 1994; Savage, 1999). Recent research supports the hypothesis that opioid tolerance is related to changes at the receptor level. In rat models, chronic morphine administration was associated with a dose-dependent down regulation of spinal glutamate transporters (Mao, Sung, Ji, and Lim,2002) and an upregulation of neuronal glucocorticoid receptors (Lim, Wang, Zeng, Sung, and Mao, 2005).

If tolerance to a specific opioid is suspected, Savage (1999) recommends titration of the medication and reassessment of the pain condition. When tolerance persists, rotating opioids may be appropriate. The transfer of tolerance between substances, called "cross-tolerance," is occasionally a problem for patients, but this process is typically not complete which allows for the partial effectiveness of the newly administer opioid. Although tolerance should not be equated with addiction, Bannwarth (1999) cautions that a patient's repeated requests for higher doses of opioid may indicate the development of abuse behaviors, especially when numerous attempts have been made to address tolerance.

Physical dependency. Physical dependency, as explained by Savage (1999), involves a state of neurophysiological adaptation to the presence of a substance, such that the abrupt decrease or discontinuation of the substance, or the introduction of an antagonist, results in withdrawal symptoms. These withdrawal symptoms are characterized by signs of autonomic hyperactivity, including diarrhea, rhinorrhea, piloerection, sweating, increased pulse and blood pressure; and central nervous system arousal including irritability, anxiety, and sleeplessness. Additionally, patients may experience an unusual exacerbation of pain, including strong muscle aches, bone pain, and abdominal cramping (Savage, 2002). For opioids, neurophysiological adaptation is believed to involve second messenger systems, and the result of changes in opioid receptors in both central and peripheral neurons. Physical dependency is a typical consequence of using opioids and is expected to occur in nearly all patients who use them for more than a few days, or with frequent episodic use, depending on dosage intervals and levels (Savage, 1999). Brookoff (2000) emphasized that physical dependency is a medical condition and should not be interpreted as a psychological weakness. Like tolerance, physical dependency may be present in addiction, but it is not sufficient to indicate addiction. Instead, withdrawal symptoms appear related to an increase in norephinephrine availability in the locus ceruleus, which is distinct from the dopaminergic pathways located in the nucleus accumbens and ventral tegmental areas that are involved in mechanisms of reward and addiction. In contrast to patients with an addiction, physically dependent patients typically do

not crave or feel compelled to use when appropriately tapered from opioids (Savage, 2002).

Opioid Addiction

Models of addiction. In order to develop an appropriate definition of addiction in the use of opioid medication, it is helpful to examine the more general models of addiction. McCaffery and Pasero (1999) describe three general models of addiction. The "moral model" considers addiction behaviors to be a reflection of moral weakness or lack of willpower. The "criminal model" views addiction as the product of an evil or bad character. The "disease model" holds that addiction is an acquired brain disease. In the extreme, the moral model views substance abuse as reprehensible behavior in which upstanding citizens would not participate. In this model, resisting addiction behaviors is a matter of will, and individuals who are unable to resist temptation are deemed to have insufficient moral fiber. Under this model, addicts are blamed for their condition, and therefore, are less worthy of care than are patients with other afflictions (McCaffery & Pasero, 1999).

The criminal model focuses on the illegality of many behaviors surrounding addiction (McCaffery & Pasero, 1999). According to this model, using certain drugs for the sole purpose of experiencing their psychoactive effects is illegal in this country, and therefore, individuals who do so are criminals and should be treated like criminals. The pervasiveness of this model in American culture is reflected in the high percentages of individuals incarcerated in federal

prisons (59.5%) and state penitentiaries (22.3%) for drug offenses (Office of National Drug Control Policy, as cited in McCaffery & Pasero, 1999). In fact, the United States of America has an entire government agency, the DEA, devoted to the apprehension and prosecution of individuals who use illicit substances and individuals who support this illicit substance use in some way. Under this model, individuals with addiction are characterized as unethical and deserving of legal and/or social sanctions. Some researchers believe that the pervasiveness of the views described in the moral and criminal models of addiction contribute to patients' fears of taking opioids for the treatment of pain, and to physicians' reluctance in prescribing opioids, even when their use is indicated (Brookoff, 2000; Fins, 1997).

In contrast to the more judgmental moral and criminal models, the medical model conceptualizes addiction as a brain-based disease process with identifiable risk factors and a pathophysiological basis (McCaffery & Pasero, 1999). Under the medical model, addiction, like any other disease, can be diagnosed according to a defined set of symptoms, follows a typical course, and is managed or cured with specific interventions. As with other diseases, addiction is expressed uniquely among individuals and its correlates vary behaviorally, socially, and psychologically. Instead of blaming and stigmatizing the addicted individual, the medical model encourages understanding of the individual and situational conditions under which addiction occurs, and implementation of appropriate interventions. Rather than punishment, this model holds that multi-modal treatment consisting of pharmacotherapy, skills-training, support groups, and

lifestyle change, is the appropriate response to addiction (McCaffery & Pasero, 1999).

Biopsychosocial models of addiction, which incorporate aspects of the models described above, seem to represent the most commonly accepted view of opioid addiction among pain researchers (Portenoy, 1996). Savage (1996), for example, reports: "Addiction has been characterized as a drive state, not unlike hunger, thirst, maternal protective instinct, and sex. When an addict is craving, the pursuit of satiety may lead to aggressive antisocial behavior, including lying, manipulation, stealing, and, sometimes, violence," (p. 280-281). This view emphasizes the physiological core of the addiction process, while highlighting some of the destructive behaviors that can result. Savage advises that these destructive behaviors be viewed as a manifestation of the disease process, as opposed to a character flaw, since treatment of the disease can produce changes in overt behavior. Nestler and Aghajanian (1997) concur that addiction is a biological process that occurs when brain physiology is altered by repeated administrations of a drug. Additionally, these authors acknowledge that psychological and social factors facilitate this biological process.

Further clarifying the distinction between addiction and abuse, Savage (1999) notes that the concept of opioid "abuse" does not necessitate the involvement of physiologically-based reward mechanisms associated with opioid addiction. Therefore, patients may use opioid medications in ways that may be harmful to themselves or others, or for purposes other than prescribed; however, these behaviors may not reach the full extent of the physiological and behavioral

correlates indicative of addiction. Hence, this conceptualization of abuse underscores that clinically significant misuse of opioids can occur without escalating to the level of addiction.

Definition of Addiction. Diagnostic criteria must be established before opioid addiction can be identified in any individual. The most widely accepted criteria for evaluating disordered substance use are based on the traditional medical model and published in the DSM-IV (American Psychiatric Association, 1994). The diagnostic criteria outlined in the DSM-IV distinguish between substance abuse and substance dependence according to certain indices of severity, and describe a clinically maladaptive pattern of substance-related behaviors that include failure to fulfill major role obligations, recurrent involvement in physically hazardous situations (e.g., driving while intoxicated) and illegal activities (e.g., disorderly conduct), and persistent social and interpersonal problems (e.g., physical fights and arguments). To meet criteria for substance abuse, these behaviors must be relatively consistent within a 12-month period. In the definition of substance dependence, the definition of substance abuse is extended by the addition of the presence of tolerance, withdrawal, and compulsive drug taking behaviors.

Although the DSM-IV criteria are generally accepted as the standard for identifying problematic substance use, many researchers debate their appropriateness for assessing opioid misuse in pain patients (Nedeljkovic, et al., 2002; Savage, 1999; Sees & Clark, 1993). Specifically, researchers argue that the inclusion of physical dependence and tolerance as diagnostic criteria for defining

substance dependence is inappropriate because these phenomena are expected consequences of opioid treatment. Nonetheless, some behavioral components of the DSM-IV criteria are still relevant in assessing problematic opioid use in chronic pain patients, especially, the criterion of continued drug use despite negative physical, psychological, or social effects (Robinson, et al., 2001; Savage, 2002).

According to the American Pain Society, misunderstandings among patients, health-care providers, and the general public regarding the use of opioids in the treatment of pain result from inconsistencies in the use of the term addiction. As a result, pain patients may receive inadequate treatment and be unfairly stigmatized (Liaison Committee on Pain and Addiction, 2001). In an effort to resolve inconsistencies in the definition of opioid addiction among chronic pain patients, three national organizations (American Society of Addiction Medicine, American Academy of Pain Medicine, and American Pain Society) have developed a consensus definition of addiction. Addiction, as applied to pain patients taking opioid medications, is defined as:

> a primary, chronic, neurobiological disease, with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized by behaviors that include one of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving (Liaison Committee on Pain and Addiction, 2001, p. 2).

Currently, this definition is the most commonly accepted among researchers of opioid addiction in pain management settings. Some researchers, however,

extend this definition by requiring that compulsive and aberrant use of opioids be extreme enough to cause disruption in the patient's fulfillment of life activities, such as work and social obligations, before it meets the criteria for addiction (Compton, et al., 1998; Miotto, et al., 1996; Portenoy, 1996).

Behavioral signs and correlates. Despite the development of a consensus definition, investigators have continued efforts to develop more specific criteria for identifying opioid misuse and/or addiction. For many diseases, specific physiologically-based laboratory tests have been developed to aid in diagnosis, and the results of these tests operationalize the definition of the disease. Opioid addiction, however, has not yet been operationalized in this way. For example, a patient may test positive for opioids on a urine toxicology screen, but this does not necessarily mean the patient is addicted. This is especially true for pain patients who would be expected to have opioids in their systems. The challenge of operationalizing opioid misuse has prompted investigators to identify and enumerate several specific behaviors that, based on clinical observation, are suggestive of problematic opioid use (Portenoy, 1996; Savage, 1996). Portenoy (1996) compiled a list and divided "aberrant drug-related behaviors" into two categories of risk, "probably more predictive" and "probably less predictive" of opioid misuse. The "probably more predictive" category includes behaviors such as: (a) selling prescription drugs; (b) forging prescriptions; (c) stealing or borrowing drugs from others; (d) repeatedly losing prescriptions; and (e) multiple dose escalations or other non-compliance despite warnings. The "probably less predictive" category includes: (a) aggressive complaining about the need for more

drug; (b) drug hoarding; (c) requesting specific medications; and (d) unsanctioned dose escalation or other non-compliance on one or two occasions. Savage (2002) describes three categories of behavior that warrant further attention by clinicians: (1) continued use of drugs despite adverse consequences or harm; (2) loss of control over drug use; and (3) preoccupation with drug use or drug-seeking behavior. Savage emphasizes that a single incident of these behaviors is not necessarily indicative of a problem. Instead, a pattern of repeated incidents of one behavior or several different behaviors would be more suggestive of a problem.

Pseudoaddiction. The term "pseudoaddiction" is used to describe a pattern of aberrant behaviors reflective of the distress and urgency felt by patients receiving insufficient pain relief, which may be mistaken for opioid medication abuse. Thus, a patient exhibiting pseudoaddictive behaviors might take more opioids than are prescribed and frequently call for early refills, in an effort to obtain adequate pain relief. This highlights the importance of considering the motivation behind the drug-seeking behaviors when developing a definition of addiction. Patients with pain often become preoccupied with their illness and suffering. Hence, whatever brings relief from this suffering takes on special significance. This preoccupation with illness and relief from pain and suffering, however, should not be erroneously attributed to preoccupation with the mood-altering effects of their opioid medications (Wesson, et al., 1993).

As with addiction, developing an operational definition of pseudoaddiction has been a challenge. While some behavioral criteria have been proposed for identifying pseudoaddiction, additional investigation is required for

validation. As a result, physicians and pain management specialists must continue to rely on clinical judgement to identify problematic opioid use. The use of clinical judgement presents its own challenges, including distinguishing between actual opioid addiction and "pseudoaddiction" (Bannwarth, 1999; Brookoff, 2000; Savage, 1996).

Savage (1996) suggests that careful observation of a patient's responsiveness to higher doses of the opioid can be helpful in distinguishing true addictive behavior from pseudoaddictive behavior. Pseudoaddiction might be the appropriate explanation for the original opioid misuse behaviors if the patient reports improved analgesia with a higher dosage, while simultaneously demonstrating improved functioning and lessened drug-seeking. If higher doses do not produce improved analgesia and functioning, and the drug-seeking behaviors do not diminish, this may suggest the patient is more focused on the capacity of the medication to induce sedation and euphoria, and the physician should consider the possibility of addiction (Savage, 1996). So, for some investigators, the patient's degree of preoccupation with the mood-altering properties of opioids, and the motivation behind it, may have utility in distinguishing the pseudoaddicted from the truly addicted patient. Likewise, the distinction between the "primary" and "secondary" reinforcing properties of opioids may be helpful in identifying the motivation behind these behaviors (Savage, 1999). Thus, aberrant behaviors in addicted patients may be motivated by the primary reinforcement of opioid-induced euphoria or tranquility, while

similar behaviors in non-addicted, under-treated patients are motivated by the secondary reinforcement of pain avoidance.

Risk factors for opioid addiction. Risk factors for opioid abuse stem from several domains, including demographic, social, genetic, psychiatric, and personality (Portenoy, 1996; Robinson, et al., 2001; Strain, 2002). Physicians would be well advised to consider the patient's personal history for factors that might raise the probability of opioid abuse, in addition to evaluating aberrant medication-related behaviors.

Although not specific to opioids, the literature points to many risk factors associated with substance abuse among non-pain patients (Robinson, et al., 2001). The 1998 National Institutes of Health Consensus Conference on Opioid Addiction, as summarized by Robinson and colleagues (2001), concluded that there is persuasive evidence that drug abuse may be at least partly related to hereditary factors. Specifically, two genetic pathways are believed to increase the risk for substance abuse: 1) a biological parent with a history of substance abuse; and 2) a biological parent with antisocial personality. Furthermore, the first degree relatives of patients who are opioid dependent have a 6.7 times greater risk of substance abuse, 3.5 times greater risk of alcoholism, and 7.6 times greater risk of antisocial personality, than relatives of patients who are not opioid dependent. Based on such evidence, some investigators speculate that the development of addiction in some individuals may be the result of pre-existing alterations in the limbic system that make those individuals particularly sensitive to the reinforcing effects of addictive substances (Blum, 1989).

Similarly, certain demographic and social variables may influence an individuals risk for substance abuse (Kaplan & Saddock, 1998). Lower socioeconomic status is often associated with elevated risk for substance abuse. Children who come from divorced or single-parent households, or who demonstrate behavioral problems, are also at greater risk (Kaplan & Saddock, 1998). Early family trauma, such as physical or sexual abuse which are more common in families where one or more parents are alcoholic, may be a risk factor for substance abuse also (Savage, 1993).

Specific to opioid abuse, the literature underscores the importance of social, genetic, and psychiatric risk factors. Michna and colleagues (2004) demonstrated that family history of substance abuse, personal history of drug or alcohol abuse, and history of legal problems were useful in predicting opioid abuse. In a study of patients taking short- and long-acting opioids at a hospital-based pain management program, patients whose histories were positive for family substance abuse and legal problems demonstrated more aberrant drug-related behaviors, including higher incidence rates of lost or stolen prescriptions and testing positive for illicit substances, than patients whose histories were negative. Patients with positive histories also had significantly higher rates of motor vehicle crashes and mental health problems. Additionally, patients with positive histories took higher doses of opioids and reported fewer adverse events than patients with negative histories. However, demographic characteristics did not differentiate patients at high risk of opioid abuse from patients at low risk (Michna, et al., 2004).

An individual with a history of substance abuse may be at increased risk for abusing a variety of substances, a phenomenon labeled “cross addiction” (Savage, 1993). Among patients with opioid dependence, several studies have reported the detection of other substances including alcohol, cannabis, cocaine, hallucinogens, inhalants, nicotine, sedatives, and stimulants (Strain, 2002). In a survey of 15,000 households, the Epidemiologic Catchment Area study found that up to 84% of cocaine addicts, 75% of amphetamine addicts, 50% of opioid addicts, and 37% of marijuana addicts had histories of alcoholism (Robins, et al., 1987). Michna and colleagues (2004) also found that a history of substance or alcohol abuse was predictive of aberrant drug-related behaviors in pain patients treated with opioids. However, other researchers found that history of substance or alcohol abuse failed to distinguish between chronic opioid users and opioid abusers among patients with CNP (Chabal, Erjavec, Jacobson, Mariano, & Chaney, 1997).

Opioid abuse also appears to be associated with various forms of psychopathology. Lifetime rates of psychiatric disorders are greater than 40% among opioid abusers, and some studies have reported lifetime rates that exceed 80%. Depression is the major psychiatric disorder diagnosed in opioid abusers, but they show higher than average rates of anxiety disorders, sleep disorders, and antisocial personality disorder, as well (Strain, 2002). Additionally, conduct disorder in children and posttraumatic stress disorder also appear related to higher rates of opioid abuse, according to the DSM-IV (APA, 1994).

Finally, some evidence suggests that certain types of pain patients may be at increased risk for opioid misuse. For example, some researchers propose that patients with "idiopathic" pain (i.e., pain without identifiable organic pathology), and high levels of psychological distress and disability, may be more vulnerable to problematic opioid use (Collet, 1998; Portenoy, 1996). Because patients with these characteristics tend to be referred to interdisciplinary pain management programs, health-care providers in these centers need to be particularly cautious and thorough in assessing risk (Portenoy, 1996). Clinical surveys, on the other hand, indicate that the risk of substance abuse may be lower in patients with organic pain syndromes, and no history of substance abuse or psychiatric illness (Cherny, 1996).

Assessment of Opioid Abuse

Risk assessment for opioid abuse, like the pain phenomenon, is best conceptualized from a biopsychosocial perspective. The hallmark of biopsychosocial assessment is the use of multiple sources and kinds of information in the process of developing a comprehensive understanding of the problem (Robinson, et al., 2001). While a multi-modal assessment strategy is generally preferred, it is particularly important when evaluating substance abuse, due to the complex nature of the problem and its frequently subtle manifestation. In patients who display highly aberrant drug-taking behaviors, such as stealing drugs or injecting oral formulations, the identification of substance abuse may be relatively straightforward. In patients who manifest softer signs, however,

substance abuse may be more difficult to detect (Portenoy, 1996; Adams, 2004). These softer signs of substance abuse may be indirect, and patients displaying them may be able to maintain socially appropriate presentations, at least early in the progression of the problem (Savage, 1993). Miotto and colleagues (1996) offer a continuum of errant behaviors, ranging from mild indiscretion to prescription forgery, that indicate varying degrees of opioid misuse. Consequently, the identification of opioid misuse is not simply a dichotomous decision as to whether or not it is present. Rather, it is a process during which the clinician judges the degree and clinical significance of the problem, and determines the extent to which it necessitates specific intervention (Miotto, et al., 1996; Adams, 2004).

Stepwise approach to assessment. Ideally, risk assessment for opioid misuse is conducted as part of a thorough, integrated medical and psychosocial assessment (Adams, 2004). Gatchel (2001) proposed a "stepwise approach" to biopsychosocial assessment of patients in order to systematize this process. The "stepwise approach" is based on the assumption that no individual instrument or technique can sufficiently assess all the relevant variables in evaluating the treatment needs of a pain patient. Additionally, a patient's status changes over time and therefore, repeated assessments are necessary to monitor treatment progress and modify the treatment plan accordingly. The first step in this approach is a comprehensive initial evaluation that typically includes a history, physical examination, and diagnostic testing. Under circumstances in which a clear diagnosis is reached and an appropriate intervention in determined, the

treatment plan follows in a relatively straightforward fashion. At an appropriate time during treatment, outcome measures are taken. If treatment goals have been reached, the patient then receives routine follow-up care. Frequently, additional steps are required for successful treatment of complex pain syndromes. When the initial evaluation does not result in a clear picture of the problem, additional time and assessment resources are needed to clarify the pain syndrome and the factors influencing it. Then, the clinician must make a decision regarding the appropriateness of the available treatment resources. If the available interventions are unlikely to be successful, the clinician may need to refer the patient to alternative treatment providers. On the other hand, if an available intervention is likely to be successful, that intervention is administered and outcomes are measured at various intervals. If progress is not optimal based on outcome measures, the clinician should re-evaluate the treatment plan for necessary revisions.

The stepwise approach to assessment can be applied to any type of intervention, including opioid treatment (Gatchel, 2001; Adams, 2004). Mayer, Prescott, and Gatchel (2000) note that treatment outcomes should include the patient's self-report of pain, as well as, the patient's functional, psychosocial, financial, and employment status. Thus, analgesia is not the only relevant treatment outcome for pain patients. Accordingly, optimally successful treatment plans result in sufficient analgesia for patients to maximize their ability to carry out important life-functions. Sees and Clark (1993) report that some experts assert that level of functioning is the most important outcome to consider in the

treatment of chronic pain patients. This raises the question of whether or not long-term opioid treatment is appropriate with patients who do not show gains in overall level of functioning. Clearly, long-term opioid treatment is not warranted if it produces diminished functioning; however, some experts question whether pain relief alone is a sufficient clinical outcome to justify the long-term administration of opioids. This question requires clinicians to consider which treatment goals are realistic for an individual patient and to structure the treatment plan accordingly (Sees & Clark, 1993; Adams, 2004).

History and physical examination. A detailed history and physical examination are two standards in a comprehensive assessment. Miotto and colleagues (1996) outline a comprehensive set of interview guidelines and detailed questions for probing analgesic abuse in chronic pain patients. Domains requiring explicit assessment include: (a) evaluation of pain syndrome; (b) patterns of opioid use; (c) history of substance abuse; (d) family history of substance abuse; (e) psychiatric history; and (f) social and family factors. When evaluating the patient's pain syndrome, the clinician needs to assess the nature, duration, and extent of pain, previous treatments and related outcomes. The clinician also needs to identify any factors from the patient's medical, surgical, or legal history, as well as the environment, that may be sustaining his or her pain. Likewise, the clinician should assess the patient's psychological reaction to pain and the patient's pattern of relationships with healthcare providers. During the assessment, the clinician is advised to elicit ways in which the patient obtains and uses opioids, while also listening for evidence of drug-seeking behaviors or loss

of control over opioid use. Assessing the extent of pain relief provided by analgesics and the associated degree of functional restoration is critical. The clinician should also obtain a substance abuse history from both the patient and the patient's family, including any diagnoses of addiction and prior detoxifications. A psychiatric history, including psychological trauma, is valuable in helping to establish the temporal relationship between psychiatric symptoms and chronic pain, and to elicit evidence of a somatoform disorder. Finally, a social history evaluation can yield important information regarding the impact the pain condition has on the patient's family, as well as concerns that family members may have regarding the patient's medication use (Miotto, et al., 1996).

According to Savage (2002), the general medical history and physical examination can often reveal important information regarding risk of opioid abuse. The author recommends that the history include information regarding conditions often associated with alcohol or drug use, such as traumatic injuries, hepatitis, human immunodeficiency virus (HIV), and ulcers. Intoxication, sedation, or cognitive impairment may indicate recent drug use, although they must be distinguished from legitimate medication side effects and concurrent medical conditions. The physical examination should include a review of cutaneous, visceral, neurological, and psychomotor changes that could indicate chronic alcohol or drug use.

Behavioral observations. Beyond the history and physical examination, the clinician can enhance the risk assessment for opioid misuse through behavioral observations during the initial evaluation and follow-up visits (Adams,

2004). Clinicians should extend the assessment of risk for opioid misuse past the initial evaluation in order to capture problems that may arise later in treatment (Robinson, et al., 2001). As discussed above, Portenoy (1996) and Savage (1996) have detailed many potentially risky behaviors. Although, some of these behaviors are not directly observable by the clinician (e.g., borrowing opioid medication from a family member); others might be observable in the clinic environment (e.g., multiple phone calls for early medication refills). However, caution and clinical judgement are necessary for interpreting an instance of "aberrant" behavior (Portenoy, 1996). When a patient displays an ostensibly drug-seeking behavior, the clinician must not automatically assume the patient is misusing or addicted to opioids. Instead, the clinician must explore the nature and implications of the behavior, and monitor it over time.

Assessment Measures. Several researchers have developed assessment measures to screen for and predict risk of opioid misuse. For example, a committee of healthcare providers at the Veterans Affairs Medical Center in Seattle formulated and tested a checklist of behaviors that are suggestive of or consistent with prescription opioid abuse in chronic pain patients (Chabal, et al., 1997). The behaviors were based on the content and intent of the Diagnostic and Statistical Manual of Mental Disorders-3rd Edition Revised (DSM-III-R), and include:

1. The patient displays an overwhelming focus on opiate issues during pain clinic visits that occupy a significant proportion of the pain clinic

visit and impedes progress with other issues regarding the patient's pain. This behavior must persist beyond the third clinic treatment session.

2. The patient has a pattern of early refills (3 or more) or escalating drug use in the absence of an acute change in his or her medical condition.

3. The patient generates multiple telephone calls or visits to the administrative office to request more opioids, early refills, or problems associated with opiate prescription. A patient may qualify with less visits if he or she creates a disturbance with the office staff.

4. There is a pattern of prescription problems for a variety of reasons that may include lost medications, spilled medications, or stolen medications.

5. The patient has supplemental sources of opiates obtained from multiple providers, emergency rooms, or illegal sources (Chabal et al., 1997, p. 51).

The committee determined that a patient may be abusing opioids if three or more of five behaviors on the checklist were observed (Chabal et al., 1997). Similarly, Compton and colleagues (1998) identified 3 items from their 43-item, interview-based opioid abuse screening questionnaire that were particularly useful in identifying opioid misuse: (1) displaying a tendency to increase opioid dose; (2) having a preferred route of administration; and (3) considering oneself addicted.

The Pain Medication Questionnaire (PMQ, Adams, et al., 2004) is a 26-item self-report measure designed to assess risk of opioid misuse in chronic pain patients. Based on suspected behavioral correlates of opioid misuse and input from clinicians, the items address a range of potentially dysfunctional attitudes and aberrant behaviors. Researchers compared patients whose scores fell in the

lower third to patients whose scores fell in the upper third. A significantly greater proportion of high scoring patients were receiving disability payments than low scoring patients. High scoring patients reported greater levels of pain-related disability as measured by the SF-36, and higher levels of depression as measured by the MMPI and BDI. Higher PMQ mean scores were also found among patients who were not working due to pain or injury, relative to patients who were employed or unemployed for non-pain-related reasons (Adams, et al., 2004).

Similar to the PMQ, the Screener and Opioid Assessment for Patients with Pain (SOAPP, Butler, Budman, Fernandez, & Jamison, 2004) was developed to assess potential risk of abuse in chronic pain patients being considered for long-term opioid therapy. Pain and addiction experts suggested the initial pool of 24 items thought to be important characteristics of patients at risk for abusing their medication. These items were administered to a sample of patients taking opioids for chronic pain. Six months later, patients exhibiting aberrant drug-related behavior were identified based on a positive score on the Prescription Drug Use Questionnaire interview, positive urine toxicology screen, and/or staff ratings that the patient had a serious drug problem. Of the original 24 items, 14 were predictive of substance misuse (Butler, Budman, Fernandez, & Jamison, 2004).

The Opioid Risk Tool (ORT), developed by Webster and Webster (2005), showed a high degree of sensitivity and specificity for determining which chronic pain patients treated with opioids are at risk for opioid-related aberrant behaviors. The ORT was based on risk factors identified in the literature including: age, personal and family history of substance abuse, history of preadolescent sexual

abuse, and certain psychological disorders. Among patients scoring in the high risk category, 90.9% displayed aberrant behavior (Webster and Webster, 2005).

Prevalence of Substance Abuse in Chronic Pain Patients

Although there is a fairly extensive body of literature examining rates of substance abuse in patients abusing illicit opioids, few controlled studies have examined substance abuse in chronic pain patients taking prescribed opioids for medical purposes. The interpretation of the existing literature is complicated by several factors including inconsistent definitions of addiction, small sample sizes, selection biases, and variability in measurement techniques (Portenoy, 1996; Fishbain, Rosomoff, & Rosomoff, 1992). Nonetheless, one review of relevant literature found rates of diagnoses for drug abuse, dependence, and addiction ranging from 3.2% to 18.9% among chronic pain patients (Fishbain, et al., 1992). In a sample of patients with chronic disabling occupational spinal disorders, nearly 11% of patients met criteria for substance abuse or dependence (excluding alcohol) in the past month, a rate which is five times higher than the general population estimate (Dersh, Gatchel, Mayer, Polatin, & Temple, 2006). In a study of chronic musculoskeletal pain disability patients, 9.8% met criteria for current drug abuse or dependence, a rate which is four times higher than general population estimate. In the same study, 24.6% of patients had met criteria for drug dependence in their lifetime, a rate which is over three times greater than the general population estimate. More specifically, 8.4% and 15.3% of patients met criteria for current and lifetime opioid dependence, respectively (Dersh, 2000).

Adding support to concerns regarding iatrogenic opioid dependence, Dersh (2000) reported that 1.6% of chronic musculoskeletal pain disability patients met criteria for opioid dependence prior to their injury, while 13.8% met criteria for opioid dependence after their injury. A study examining characteristics of chronic low back pain patients found that 13% of patients being treated with NSAIDs only and 43% of patients being treated with opioids were diagnosed with a substance abuse disorder (Breckenridge & Clark, 2003). According to Savage (2002), reviews of substance use disorders in chronic pain patients have concluded that these patients show higher than expected rates of these disorders compared to the general public.

A number of early survey studies reported low rates of opioid misuse among various pain populations. For example, among 2,369 patients at a large headache center who had access to opioids, only 3 patients were identified with potential opioid addiction (Medina & Diamond, 1977). Likewise, the Boston Collaborative Drug Surveillance Project identified only 4 cases of opioid addiction from among 11,882 hospitalized patients with no history of substance abuse, who received at least one dose of opioid medication (Porter & Jick, 1980). Although, the results of these surveys seem reassuring when compared to the national prevalence of alcoholism (3-16%) and other types of substance abuse (5-6%); the limited time-frames, and highly selective subgroups of pain patients, may not provide an accurate picture of the problem (Portenoy, 1996). Significant subgroup biases may also be reflected in a set of studies involving patients in interdisciplinary pain management programs, given that these studies revealed

relatively higher rates of aberrant drug use among patients participating in these programs (Ready, Sarkis, & Turner, 1982; Turner, Calsyn, Fordyce, & Ready, 1982). However, researchers have subsequently acknowledged that patients referred to these programs frequently have higher levels of psychosocial distress and disability, compared to other chronic pain patients. The extent of conflicting empirical data highlights the need for more systemic, longitudinal studies to clarify rates of substance abuse among chronic pain patients.

Joranson and colleagues (2000) explored opioid use and misuse among a nationally representative sample of hospital emergency room admissions that involved drug abuse. Investigators reviewed medical record data from 1990 to 1996 stored in the databases of the Drug Abuse Warning Network (DAWN), and analyzed trends in medical use and abuse of five opioid analgesics. Results revealed the following trends in medical use of opioids, over the 6 years studied: (a) fentanyl use increased by 1168%; (b) morphine use increased by 59%; (c) oxycodone use increased by 23%; and (d) hydromorphone use increased by 19%. Of the five opioid analgesics under consideration, only meriperidine use decreased, by 35%. The total number of opioid abuse "mentions" (reports) in the medical records increased by 6.6% over the six years. However, the proportion of opioid abuse mentions relative to overall drug abuse mentions declined by 25% (5.1% to 3.8% of total mentions). Investigators reported the following trends from 1990 to 1996 in abuse mentions for specific opioid analgesics: (a) fentanyl abuse mentions declined by 59%; (b) oxycodone abuse mentions declined by 29%; (c) hydromorphone abuse mentions declined by 15%; and (d) morphine

abuse mentions increased by 3%. Therefore, the total number of opioid abuse mentions increased from 1990 to 1996, but failed to keep pace with increases in overall drug abuse mentions. The investigators noted that these results likely underestimate the prevalence of the drug abuse problem, given their data only represents abusers who present to emergency rooms. Joranson and colleagues concluded that pain management specialists should continue their willingness to utilize opioid analgesics, as appropriate, because there does not appear to be an increase in negative health consequences related to increases in opioid use.

In contrast, a review of the DAWN database between 1994 and 2001 revealed that the availability, non-medical use, and abuse of opioids have increased over the eight years studied (Zacny, et al., 2003). A task force of the College on Problems of Drug Dependence focused on the differences between the availability and abuse of hydrocodone and oxycodone, two of the most widely-prescribed and abused opioids. Although the ratio of illicit to licit use of hydrocodone remained relatively stable from 1994 to 2001, the ratio for oxycodone increased significantly from 2000 to 2001. When compared to 1999, the rate of oxycodone abuse relative to its availability for medical use increased by 39% in 2000 and 108% in 2001 (Zacny, et al., 2003).

The September 2004 issue of The DAWN Report (Crane, 2004) focused on drug abuse-related emergency department visits related to opioid analgesics. Opioid dependence was the identified motive behind 47% of the opioid visits. In 2002, 16% of all drug abuse-related emergency department visits involved opioids, for an estimated total of 108,320 visits. Visits involving opioids

increased 20% from 2001 to 2002, and 153% from 1995 to 2002. Between 1995 and 2002, all age groups showed increases in the number of visits involving opioids, except 12- to 17-year-olds. Patients ages 45 to 54 years showed the greatest increase of 298%. Seventy-five percent of visits involving opioids, also involved at least one other substance.

The DAWN Report (2004) focused on the nine opioid analgesics most commonly involved in drug-abuse related emergency department visits: codeine, fentanyl, hydrocodone, methadone, morphine, oxycodone, and propoxyphene. In 2002, hydrocodone and oxycodone were the opioids most frequently mentioned by name, but nearly 40% of visits involved unspecified opioids (Crane, 2004). From 1995 to 2002, drug abuse-related visits increased for oxycodone by 512%, methadone by 176%, hydrocodone by 159%, and morphine by 116% (Crane, 2004).

In response to widespread reports of OxyContin® abuse, Purdue Pharma L.P. funded the development of a proactive abuse surveillance program. The Researched Abuse, Diversion and Addiction-Related Surveillance (RADARS®) system utilized drug abuse experts as a source of data on the prevalence and magnitude of prescription drug abuse. These experts included clinicians, epidemiologists, and treatment counselors (Cicero, Inciardi, and Munoz, 2005). Results indicated that between 2002 and 2004, prescription drug abuse was reported in 60% of the zip codes surveyed. The most commonly abused prescriptions, from highest prevalence to lowest, were: OxyContin, hydrocodone, other oxycodone, methadone, morphine, hydromorphone, fentanyl, and

buprenorphine. Over the 30 month period, prevalence for most of these drugs increased. However, only the trends for OxyContin and hydrocodone were statistically significant. Researchers highlighted that Oxycontin abuse is a pervasive problem in the United States, that effective prevention and intervention strategies are needed, and care must be taken in the implementation of these strategies so the legitimate and appropriate use of these drugs is preserved (Cicero, Inciardi, and Munoz, 2005).

In May 2007, Purdue Pharma plead guilty to one felony charge of fraudulently misbranding a drug, while three current and former executives each plead guilty to one misdemeanor count of misbranding a pharmaceutical. Charges stemmed from sales practices between 1996 and mid-2001, during which the public was misled regarding the addictive potential of OxyContin. Purdue Pharma and the three executive were fined $634.5 million (Zimmerman, 2007).

Theories of Pain

The nature of the pain phenomenon is complex and presents challenges similar to those in conceptualizing addiction. Early theories likely contributed to the negative attitudes held by some physicians toward chronic pain patients, and the subsequent controversy surrounding opioid treatment. As suggested by Weinstein and colleagues (2000), a rigid application of the “medical model” may limit some physicians’ ability to comprehend and effectively treat patients with pain. According to the medical model, the physician’s primary goal is to identify the pathophysiologic cause of the patient’s pain, to diagnosis it, and to choose an

appropriate intervention. When this process leads to a cure, the physician derives a sense of satisfaction and professional mastery from a successful outcome. However, the multi-determined, subjective nature of pain frequently makes it difficult to identify the exact etiology. Likewise, it may be difficult to achieve a satisfactory outcome, even when the etiology is known. When this occurs, a physician who adheres closely to the medical model is more likely to feel frustrated and more reluctant to treat the patient, due to diminished confidence. These feelings, therefore, have significant negative clinical implications for the multitudes of patients suffering from intractable pain (Weinstein, et al., 2000).

Dissimilarly, many researchers in the field of pain have favored the biopsychosocial model for understanding the nature of pain and its management (Gatchel & Turk, 1996; Turk & Rudy, 1987; Weinstein, et al., 2000). The biopsychosocial model acknowledges pain as a complex perceptual phenomenon that involves sensory, affective, interpersonal, and behavioral components that cannot be objectively verified. Consequently, it is important for patients with chronic pain to be treated in a comprehensive manner that incorporates a wide variety of relevant psychological and physiological factors. For some patients, a pathophysiologic process may never be identified or adequately resolved, and then the focus of intervention becomes management of pain (Weinstein, et al., 2000). Currently, the biopsychosocial model of pain is the standard of care, and considered the ideal means for conceptualizing and treating chronic pain patients, but it succeeds many theories. A review of these theories is useful in highlighting

the various factors that led to the development of the biopsychosocial model of pain.

Early models of pain. The traditional biomedical model of pain dates back to ancient Greece. In the 17th century, Descartes was one of the first to describe pain according to the biomedical model, which assumes that pain results from a specific disease state that represents disordered biology. Thus, pain should cease upon removal or remediation of the biological disturbance. According to this model, data from objective tests are used to make a diagnosis of the disease, which is used to select interventions specific to the diagnosis. Interventions are considered successful if they resolve the organic dysfunction or pathology (Turk & Monarch, 2002). In this model, non-biomedical factors are ignored and assumed to have no role in pain. However, this early model is limited by its inability to account for discrepancies between pain report and physical pathology (Turk & Monarch, 2002; Flor & Turk, 1988). To explain these discrepancies, Engel (1959) coined the term "psychogenic pain" and proposed that pain has special meaning to some patients. Engel described the "pain prone patient" as possessing several characteristic features, including guilt, aggressive drives, and specific psychiatric disorders. While the concept of psychogenic pain helped promote the consideration of psychosocial factors in pain, the model also promoted a false dichotomy that organized pain into the overly-simplistic categories of "organic" or "psychogenic," which fail to explain the complex interaction between mind and body found in chronic pain processes (Turk & Monarch, 2002).

Gate control theory. The "gate control theory of pain," introduced by Melzack and Wall in 1965, was one of the first comprehensive pain theories to integrate both physical and psychological factors of the pain phenomenon. Melzack and Wall (1965) hypothesized that central nervous system mechanisms provide a physiological basis for psychological involvement in pain perception. According to the theory, the dorsal horns of the spinal cord act as a "pain gate," regulating the transmission and intensity of nerve impulses from peripheral stimuli to the central nervous system. The gate control theory of pain is recognized as the vanguard in the movement away from traditional dualistic notions of pain and has had extraordinary influence on the research and treatment of pain, despite receiving criticism that its underlying mechanism is "incomplete" (Turk & Monarch, 2002).

Since receiving this criticism, the gate control theory of pain has evolved into a theory of pain perception filtered through a body-self neuromatrix (Turk & Monarch, 2002). As described by Turk and Monarch (2002), Melzack (1999) elaborated on the gate control theory by suggesting that chronic pain is the result of a network of neurons representing the body that can be activated even in the absence of external stimulation. Like the gate control theory of pain, the neuromatrix theory accounts for psychological factors that impact pain perception. The theory proposes that a homeostatic mechanism activated in response to enduring pain, results in neurochemical and neuronal changes that may affect the individual's emotional state via the limbic system (Turk &

Monarch, 2002). Furthermore, prior experiences and genetic predisposition in responsiveness of the neuromatrix explain individual differences in pain report.

Psychosocial issues of the health belief model. In 1966, Mechanic further defined the role of social variables in the onset and maintenance of pain by proposing that people can perceive, evaluate, and respond to physical symptoms in unique ways, comprising individual "illness behaviors." Mechanic suggested that if a patient receives positive reinforcement (e.g., attention and support) for these behaviors, he or she might discover advantages to adopting the "sick role." Adoption of the sick role was considered to reflect both the individual's coping repertoire, and the influence of social and cultural conditioning (Mechanic, 1972). Thus, an individual's report of pain symptoms is the result of the interactions among perceptions of personal vulnerability and illness, social and cultural factors, vocabularies of distress, and the effects of emotional distress (Mechanic, 1972).

The "health belief model" (Becker & Mainman, 1975; Becker, 1979) considered the impact of social variables on treatment compliance. The "health belief model" posits that patients make decisions regarding treatment compliance based on treatment benefits and costs, and comply only when benefits are believed to outweigh costs. Patients' beliefs about their personal susceptibility to illness and its negative effects also influence this decision-making process. This model is helpful in understanding why some individuals with longstanding health problems appear to embrace the "sick role." The sick role affords individuals an excuse from obligations such as work or household chores, and individuals in the

sick role may receive support and care from relatives, friends, or coworkers (Bebbington, 1995). Hence, the sick role may confer the secondary benefits of attention, relaxation, and possibly financial reward, to such an extent that being incapacitated holds greater advantages than being well. Consequently, some individuals may passively resist treatment recommendations, or they may actively hinder their treatment by participating in counterproductive activities (Bebbington, 1995).

Patients who adopt the sick role may also display increased "pain behaviors," which are physical expressions used to convey the severity of their pain to other people. Fordyce (1976) asserted that environmental reinforcers can influence the pattern of pain behaviors, which has potential implications for treatment. For instance, researchers have observed that married male patients experience the impact of their pain differently, depending on the response they receive from significant others. A high correlation has been found between significant other responses and pain impact levels for individuals who report being satisfied with their marriages (Flor, Turk, & Rudy, 1989). Many chronic pain treatment programs are designed to prevent reinforcement of excessive pain behaviors, as a result of the research showing that social reaction and environmental cues can influence the experience of pain (Adams, 2004).

Biopsychosocial model of pain. Since these early theories of pain, an extensive body of evidence has emerged demonstrating a strong relationship between chronic pain and a variety of factors, including psychological, social, occupational, and legal variables (Fordyce, 1976; Flor & Turk, 1984; Katon,

Egan, & Miller, 1985; Fishbain, Goldberg, Meagher, Steele, & Rosomoff, 1985; Kinney, 1991; Polatin, Kinney, Gatchel, Lillo, & Mayer, 1993). In response to these advances, Turk and Rudy (1987) developed the "biopsychosocial" model of pain, which now serves as the standard for conceptualizing chronic pain. The biopsychosocial model proposes a multi-factorial explanation of chronic pain that considers the complex interactions among physiological, biological, cognitive, affective, behavioral, and social dimensions. These multiple dimensions contribute to pain through interaction, reciprocal determinism, and evolution (Turk & Monarch, 2002; Mayer, Gatchel, & Polatin, 1992). From a biopsychosocial perspective, pain is considered to be both a biological manifestation and a subjective experience of pathology. The interactions among biological, psychological, and social factors determine the severity and chronicity of pain. These multiple factors contribute directly to the pain experience, but they also have reciprocal effects on one another that intensify and perpetuate their effects on pain. Finally, the various influences of these multiple factors change over time with the continued chronicity of pain, resulting in an individual's unique and fluctuating course of pain experience (Mayer, Gatchel, & Evans, 2002).

Functional Restoration

The successful rehabilitation of chronic pain patients presents unique obstacles for clinicians because these individuals develop a sedentary lifestyle that often leads to significant decreases in physical functioning and capacity. This

physical deconditioning may then interfere with successful rehabilitation and place patients at increased risk of subsequent re-injury. Furthermore, these patients present with high rates of psychopathology and the combination of these factors frequently entrench an individual into the "sick role" (Gatchel &Turk, 1996).

The number of clinics specializing in the treatment of pain has grown over the last 30 years in response to the recognition that traditional forms of treatment, such as medication and surgery, have rarely resulted in the cessation of pain and its accompanying behaviors, and in some cases have been associated with the prolongation of disability (Faas, Van Eijk, Chavannes, & Gubbels, 1995; Stuckey, Jacobs, & Goldfarb, 1986). Poor surgical outcomes have been well established in the literature among compensation patients with psychosocioeconomic disincentives (e.g., Beals, 1984; Dzioba & Doxey, 1984; Fordyce, 1985). While pain clinics have made important contributions to the progression of comprehensive treatment, by recognizing the role of psychosocial factors in chronic pain, the approach used by these clinics falls short for a variety of reasons. Approaches utilized by these pain clinics are passive and fail to address physical deconditioning and inhibition. Concurrently, they frequently employ subjective report of pain in the development of treatment goals and the measurement of outcome, thus ignoring the more objective concept of functional capacity (Mayer, Polatin, & Gatchel, 1999).

The various shortcomings characteristic of traditional pain clinics prompted the development of more comprehensive treatment programs, such as

functional restoration (Mayer & Gatchel, 1988), that employ an interdisciplinary treatment approach. Functional restoration, specifically, is a form of tertiary rehabilitation developed from the recognition that function is a more useful focus of the treatment process than is subjective experience of pain. A functional restoration approach differs from the traditional methods of pain medication, surgery, and restriction of physical activities; while, it aims to increase muscle strength, endurance, and joint mobility, with the end goal of returning the patient to work and normal activity. Therefore, the primary focus of treatment is function, with the expectation that improvement in functional capacity will be followed by associated improvements in subjective pain and disability.

The assessment of physical condition in chronic pain patients has proven difficult, given that structural imaging techniques, such as CT scanning, magnetic resonance imaging (MRI), and electromyography, fail to identify structural changes consistent with patient self-report of pain or other physical findings (Mayer & Gatchel, 1988). While self-report measures may be useful in understanding the patient's perspective, they are affected by multiple mediating factors and therefore, are of limited utility. Thus, objective measurement of range of motion and strength in combination with aggressive physical reconditioning, lay the foundation of functional restoration (Mayer & Gatchel, 1988).

Functional restoration integrates its strong emphasis on physical conditioning with cognitive-behavioral based pain management. Clinically, patients often tend to avoid difficult issues, such as adjustment, family discord, returning to work, and emotional problems (Capra, Mayer, & Gatchel, 1985). To

counter this, functional restoration not only emphasizes physical capacity, but also psychological functioning, in order to treat comprehensively a variety of clinical factors. Addressing the complex psychological and social aspects of functional disability, allows further physical reconditioning to be achieved with fewer complications.

In summary, a functional restoration approach consists of a quantitatively-directed exercise program, individual counseling, group therapy, and disability management. This comprehensive approach to evaluation and treatment of chronic pain yields higher success rates as measured by work return, additional health utilization, and re-injury rates; and well-coordinated monitoring of these treatment outcomes is a critical element of functional restoration. Early research of functional restoration showed significant improvements in various socioeconomic outcomes in one- and two-year follow-up studies (Mayer, Gatchel, Kishino, Keeley, Capra, Mayer, Barnett, & Mooney, 1985; Mayer, Gatchel, Mayer, Kishino, Keeley, and Mooney, 1987; Hazard, Fenwick, Kalisch, Redmond, Reeves, Reid, & Frymoyer, 1989). Greater than 80% of all patients treated with a functional restoration approach reported returning to work within a year after discharge, while rates for non-treatment comparison groups ranged from 29-41% across studies. Furthermore, comparison groups showed approximately twice the rate of additional spinal surgery and unsettled workers' compensation litigation relative to treatment groups, five times the rate of additional visits to healthcare professionals, and higher rates of re-injury. The fact that these results were obtained by treatment providers in a number of cities,

states, and countries, with a variety of social, economic, and workers' compensation systems, demonstrated the robust clinical efficacy of functional restoration in effecting important clinical outcomes in a "worst-case" cohort of chronic pain disability patients (Bendix & Bendix, 1994).

More recent studies continue to yield support for a functional restoration treatment approach. In a randomized study, French researchers compared functional restoration to active individual therapy for low back pain. While a similar proportion of patients in both treatments returned to work within one week of completing treatment, functional restoration patients reported significantly fewer sick-leave days, more sports and leisure activities, and better physical capacities than active individual therapy patients at a 6-month follow-up (Jousset et al., 2004). In a prospective, randomized study researchers compared functional restoration, no treatment, and two less intensive treatments in several samples of chronic low back pain patients. Researchers examined various socioeconomic outcomes at one-, two- and five-years post-treatment and found the functional restoration group had better outcomes for most variables assessed at the various follow-up periods, including number of contacts with healthcare providers, number of sick days, ability to work, level of physical activity, and subjective pain level (Bendix, Bendix, Haestrup, & Busch, 1998; Bendix, Bendix, Labriola, & Boekgaard, 1998; Bendix, Bendix, Lund, Kirkbak, & Ostenfeld, 1997).

Scope of the Present Investigation

As discussed above, the role of opioids in the treatment of chronic pain is controversial. The efficacy of opioid therapy is debated and little is known about its long-term impact. Unfortunately, there is a paucity of research investigating the utility of pre-treatment level of opioid use in predicting response to chronic pain rehabilitation and identifying which patients are likely to have poorer long-term outcomes. Therefore, the purpose of the present study was to examine pre-treatment level of opioid use as a predictor of chronic pain rehabilitation for a cohort of subjects participating in a functional restoration program. In this study, subjects were divided into groups, based on their pre-treatment level of opioid use, to determine if pre-treatment level of opioid use discriminates subjects' response to treatment, as measured by socioeconomic outcomes, pain report, psychological distress, and physical functioning upon program completion and at one-year follow-up.

Demographic variables assessed included: gender, age, race, and years of education. Socioeconomic and health variables assessed included: net salary at time of injury, disability payments, case settlement status, length of disability, work-related injuries, pre-treatment and post-treatment surgeries, attorney retention, work return, work retention, and healthcare utilization. Psychosocial and physical variables assessed included: Beck Depression Inventory (BDI), Hamilton Rating Scale for Depression (HAM-D), Million Visual Analog Scale (MVAS), Oswestry Disability Questionnaire (OSW), Short-Form 36 (SF-36), Minnesota Multiphasic Personality Inventory-2nd Edition (MMPI), Wechsler

Adult Intelligence Scale-Revised, Cumulative Physical Score, and Quantified Pain Drawing (QPD). Finally, program completion was also assessed.

The following hypotheses were proposed for this study:

1. Pre-treatment level of opioid use will predict differences in response to treatment, such that subjects reporting higher levels of use will have systematically higher rates of program non-completion.
2. Pre-treatment level of opioid use will differentiate patients systematically according to compensation factors, such that subjects reporting higher levels of pre-treatment opioid use will show systematically lower rates of pre-treatment case settlement and greater benefits.
3. Pre-treatment level of opioid use will differentiate patients systematically according to pre-treatment health status, such that subjects reporting higher levels of pre-treatment opioid use will show systematically higher rates of surgery and prior work-related injury.
4. Pre-treatment level of opioid use will differentiate subjects according to level of pre-treatment depressive symptoms, such that those reporting higher levels of pre-treatment opioid use will demonstrate higher pre-treatment scores on the BDI and HAM-D.
5. Pre-treatment level of opioid use will differentiate subjects according to level of pre-treatment disability and health-related quality of life, such that those reporting higher levels of pre-treatment opioid use will demonstrate less desirable pre-treatment scores on the MVAS, OSW, and SF-36.

6. Pre-treatment level of opioid use will differentiate subjects according to level of pre-treatment psychopathology, such that those reporting higher levels of pre-treatment opioid use will demonstrate less desirable MMPI profiles.
7. Pre-treatment level of opioid use will differentiate subjects according to pre-treatment physical functioning and pain, such that patients reporting higher levels of pre-treatment opioid use will display poorer pre-treatment Cumulative Physical Scores and higher pre-treatment QPD scores.
8. Pre-treatment level of opioid use will predict improvements in depressive symptoms, such that subjects reporting higher levels of pre-treatment opioid use will show lesser improvements on the BDI and HAM-D upon program completion.
9. Pre-treatment level of opioid use will predict improvements in disability and health-related quality of life, such that those reporting higher levels of pre-treatment opioid use will demonstrate lesser improvements on the MVAS, OSW, and SF-36 upon program completion.
10. Pre-treatment level of opioid use will predict response to treatment, such that subjects reporting higher levels of pre-treatment opioid use will demonstrate lesser improvements in physical functioning and pain upon completion of functional restoration treatment.
11. Pre-treatment level of opioid use will predict differences in compensation and secondary gain variables, such that subjects reporting higher levels of pre-treatment opioid use will have systematically lower rates of case settlement and higher disability payments at one-year post-treatment.

12. Pre-treatment level of opioid use will predict differences in healthcare use, such that subjects reporting higher levels of pre-treatment opioid use will demonstrate higher rates of post-treatment surgery, post-treatment injury, and healthcare utilization at one-year follow-up.
13. Pre-treatment level of opioid use will predict differences in work-related outcomes, such that subjects reporting higher levels of pre-treatment opioid use will have systematically lower rates of work-return and work-retention at one-year post-treatment.

CHAPTER THREE

Method

Subjects

Subjects in this study included patients who were referred, and consented, to a prescribed course of treatment for chronic pain disability at the Productive Rehabilitation Institute of Dallas for Ergonomics (PRIDE), a facility that utilizes a functional restoration approach. Patients eligible for treatment at PRIDE had received previous treatment for their chronic pain, but had not yet been able to return to work. Inclusion criteria that must be met for acceptance into the PRIDE treatment program include: (1) disability related to injury lasting over four months since the injury was incurred; (2) lack of responsiveness to previous treatments of a primary or secondary nature; (3) unsuccessful surgical interventions aimed at ameliorating pain symptoms and returning the patient to physical function, or the patient does not qualify for surgical intervention; (4) the patient's physical functioning is severely impaired; (5) the ability and willingness to function in groups; (6) English- or Spanish-speaking. Additionally, as a requirement for participation, patients must taper off all opioid medications early in the treatment program. The cohort in this study was comprised of consecutive patients admitted between October 1998 and September 2002, including patients of all musculoskeletal injury types, as well as both those who did complete and did not complete functional restoration treatment. For the purposes of the present study, subjects were divided into five categories based on self-reported pre-treatment

level of opioid use. Conservative a priori estimates of power suggested that approximately 1000 subjects were needed.

Pre-treatment Opioid Use Data

Information regarding average daily dosages of opioid mediation taken at the time of admission was gathered from multiple locations in the patients' medical records. In order to standardize the collection of this information, the following procedures were followed. First, information regarding opioid use was gathered from the initial physician note. This information was compared to the information gathered by staff psychologists during the mental health evaluation (MHE). All opioid medications reported by patients during the initial physician's visit and the MHE were included in the study. If discrepancies in the quantity of medications being taken occurred, the higher dose was used for purposes of the study. Additionally, some patients were referred to the staff psychiatrist. If additional medications were reported in the staff psychiatrist's note, these opioid medications were also considered in the study. Likewise, if discrepancies occurred in the quantity of medications reported, the default used for the study was the highest reported quantity. For example, if Mr. Smith's initial physician's note reported 10 mg of Oxycontin, 5 times per day, but his MHE reported 10 mg of Oxycontin, 7 times per day, his average daily dose of opioid medications was 70 mg of Oxycontin. However, if Mr. Smith was referred to the staff psychiatrist, to whom he reported taking only 5 mg of Hydrocodone, 3 times per day, his average daily dose of opioid medications was 70 mg of Oxycontin and 15 mg of

Hydrocodone. For another example, Mrs. Rogers' initial physician's note stated that she took 5 mg of Hydrocodone, 3 times per day, and 65 mg of Darvocet, 3 times per day. Mrs. Roger's MHE reported that she took 65 mg of Darvocet, twice daily. Mrs. Rogers did not have a note from the staff psychiatrist. Thus, Mrs. Roger's average daily dose of opioid medications was 15 mg of Hydrocodone, and 195 mg of Darvocet.

Often, the opioid medication information listed in the medical record was vague or incomplete. In such cases, the following procedures were used to clarify the data. If a medication was listed without a dosage, the dosage was gathered from the initial physician's note, the MHE, or the Oral Medication Record located in the medical chart. If a number of tablets was reported, but not a dose, the lowest available dose was used by default. For example, 3 Vicodin per day defaulted to 3-5mg tablets per day, which yielded an average daily dose of 15 mg per day. If a range of tablets was listed, the midpoint of this range was considered for purposes of the study. For example, 65 mg of Darvocet, 0-8 times per day, became 4 times per day and yielded an average daily dose of 260 mg. For another example, if a patient reported taking 3-7.5 mg tablets of Vicodin per week, this yielded an average daily dose of 3.2 mg.

Once the average daily dose of opioid medications was calculated, this information was converted into equianalgesic dosages of morphine (Polatin & Gajraj, 2002; Beers & Berkow, 1999; Global RPh, 2005), and used to classify subjects into one of five categories of pre-treatment opioid use. Table 1 presents

equianalgesic doses of various opioid analgesics used in the conversion process, along with the lowest available dose.

Cases were labeled “ambiguous” if patients reported taking an opioid medication, but no specific information regarding dose or number of tablets; for example, “prn” or “occasional” use. Ambiguous cases were included in comparisons between patients taking opioids and patients not taking opioids, but were excluded from comparisons of various levels of opioid use. In some cases, patients reported taking “unknown” pain medications. These cases were excluded from analyses because whether or not these patients were taking opioids specifically, could not be determined.

Information on non-opioid analgesics (including aspirin, acetaminophen, and NSAIDS), as well as psychotropic medications (including antidepressants, anxiolytics, mood stabilizers, and neuroleptics) was also gathered using the same procedures outlined for opioids. Finally, information on whether or not patients were taking opioids, non-opioid analgesics, or psychotropic medications at the time of discharge was gathered from the “header sheet” located in the front of the medical chart.

Explanation of the Samples

Between October 1998 and September 2002, 1,369 consecutive patients were admitted to the PRIDE Functional Restoration Program. Of these 1,369 patients, 28 were classified as “Quality of Life.” “Quality of Life” patients were those who entered the PRIDE program for purposes of improving their quality of

life, but did not expect nor plan to re-enter the workforce (e.g., patients who had retired). They were excluded from the analyses of program outcomes and were, therefore, excluded from the study sample.

Of the remaining 1,341 patients, 115 reported taking unknown pain medications. Because whether or not these patients were taking pre-treatment opioids could not be determined based on the information in the medical chart, these 115 patients were excluded from the study sample.

Whether or not pre-treatment opioids were taken could be, and was, determined for the remaining 1,226 patients. Results for these 1,226 patients were labeled as "Study Sample" in the text and tables. For purposes of analyses, the 1,226 patients in the Study Sample were divided into two groups: those not taking pre-treatment opioids (the NO group, n=630), and those taking pre-treatment opioids (the YES group, n=596).

To further examine the role of pre-treatment opioid use, the Study Sample was classified into subgroups based on daily milligrams of oral morphine. An unambiguous dosage could be determined for 1,146 of the 1,226 patients in the Study Sample. These patients were divided into the following subgroups based on level of pretreatment opioid use: the NO subgroup (0 mg, n=630); the LOW subgroup (>0-30 mg, n=267); the MEDIUM subgroup (>30-60 mg, n=112); the HIGH subgroup (>60-120 mg, n=78); and the VERY HIGH subgroup (>120 mg, n=59). The remaining 80 patients had ambiguous dosages, meaning the dosage could not be determined based on the information recorded in the medical chart. For example, these patients reported "prn" or "occasional" use. Many patients

reported dosages for some, but not all of their opioid medications. Because the total dosage level could not be determined, these patients were excluded from the statistical analyses performed for opioid subgroups.

Procedure

All of the chronic pain patients received an initial evaluation before beginning treatment, which included a physical examination, a medical history, a medical case management disability assessment interview, a quantitative functional capacity evaluation, and a psychological intake interview. The patients' tertiary rehabilitative treatment at PRIDE consisted of a quantitatively-directed exercise program supervised by both physical and occupational therapists, in conjunction with a multimodal disability management component which included individual counseling, group therapy, stress management training, vocational reintegration, and future fitness management (e.g., Mayer et al., 1987).

Patient demographic data were collected as part of the intake interview noted above, at the end of which the patient was asked to complete a series of physical and functional capacity measures normalized to age, gender, and body weight. On the first day of the intensive treatment phase of the PRIDE rehabilitation program, patients were asked to complete the following instruments: the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961); the Million Visual Analog Scale (MVAS; Million, Hall, Naavik-Nilsen, Jayson & Baker, 1981), a visual analog questionnaire used to assess disability; the Oswestry Disability Questionnaire (OSW); the Short-Form

36 Health Survey (SF-36); the Minnesota Multiphasic Personality Inventory – 2nd Edition (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen & Kaemmer, 1989); and the Quantified Pain Drawing (QPD; Mooney, 1984), an analog self-report measure of perceived pain intensity. Also, patients were given the following clinician-administered instruments: the Hamilton Rating Scale for Depression (HAM-D; Hedlung & Vieweg, 1979) and the Wechsler Adult Intelligence Scale – Revised (WAIS-R; Wechsler, 1981). The BDI, HAM-D, MVAS, OSW, SF-36, and the QPD were repeated at program completion in order to evaluate response to treatment. Additionally, one year after program completion, a structured telephone interview was conducted with subjects to evaluate socioeconomic outcomes including: work status, healthcare utilization, recurrent surgery to the same body part, recurrent injury to the same body part, and case settlement status (Mayer, Prescott, & Gatchel, 2000).

Instruments and Outcome Measures

Beck Depression Inventory (BDI). The Beck Depression Inventory is a 21-item multiple-choice test designed to measure physical and emotional symptoms of depression, and is currently one of the most widely used measure of depression in both medical and psychological research. It was originally developed by Beck, Ward, Mendelson, Mock and Erbaugh (1961) with the purpose of offering a reliable and valid measure of the presence and/or severity of depression. Suggested cutoff scores are: <10 for absence of depression; 10-18 for mild to moderate depression; 19-29 for moderate to severe depression; and >29

for severe depression (Beck, Steer, & Garbin, 1988). Reliability of the BDI is good, with internal consistency coefficients exceeding .73 in nonpsychiatric samples (Beck et al., 1988). Validity is adequate, with the BDI demonstrating a correlation of .60 with the MMPI Depression Scale in a nonpsychiatric sample, and .73 with the Hamilton Rating Scale for Depression (Beck et al., 1988). Many researchers have demonstrated the validity of the measure with chronic pain patients (Geisser, Roth, & Robinson, 1997; Novy, Nelson, Berry, & Averill, 1995; Romano & Turner, 1985; Turner & Romano, 1984), although some researchers have recommended the removal of several items (Wesley, Gatchel, Garofalo, & Polatin, 1999) and/or modification of depression cutoff scores (Geisser et al., 1997; Wesley et al., 1999) because somatic items were confounded with pain symptomatology (Wesley, Gatchel, Polatin, Kinney, & Mayer, 1991).

Hamilton Depression Rating Scale (HAM-D). The Hamilton Depression Rating Scale is another frequently used measure, based on a 17-question structured interview designed to evaluate various aspects of depression rated on Likert scales ranging from 0 to 2 and 0 to 4. Points from each question in the interview are summed to yield a total score. Based on total score, the following cut-off scores are used to interpret severity of depressive symptoms: <12 (none or minimal); 12-20 (mild to moderate); 21-29 (moderate to severe); and 30+ (severe). A study by Rush, Beck, Kovacs, and Holton (1977), found an inter-rater reliability correlation coefficient of .9, which is considered good. The HAM-D demonstrated acceptable concurrent validity with the BDI (.73; Beck et al, 1988)

and the Inventory of Depressive Symptomatology (.78; Rush, Gullion, Basco, Jarrett, & Trivedi, 1996).

Million Visual Analog Scale (MVAS). The MVAS is a self-report measure comprised of 15 items evaluating pain intensity, etiological factors of pain, activities that exacerbate pain, and 10 measures of spinal movement (Helliwell, Moll, & Wright, 1992). Scores from the 15 items are summed to a total score, which can range from 0 to 150 points. Established cut-off points for interpretation are as follows: 1-40 for mildly disabling; 41-70 for moderately disabling; 71-100 for severely disabling; 101-130 for very severely disabling; and 131-150 for extremely disabling (Anagnostis, Mayer, Gatchel, & Proctor, 2003).

Oswestry Disability Questionnaire (OSW). The OSW is a self-report scale that evaluations the degree of functional impairment in activities of daily living caused by pain (Fairbanks, Couper, Davies & O'Brien, 1980). The Oswestry has demonstrated adequate reliability, with 24-hour test-retest reliability of .99; it has also shown adequate validity (Kaplan, Wurtele, & Gillis, 1996; Leclaire, Blier, Fortin, & Proulx, 1997).

Short-Form 36 Health Survey (SF-36). The SF-36 is a multipurpose, short-form health survey with 36 questions. The measure yields scores for two multi-item scales, physical health and mental health, and covers eight general health concepts: physical functioning, role limitations due to health problems, bodily pain, social functioning, general mental health, role limitations resulting from emotional problems, vitality, and general health perceptions (Ware & Sherbourne, 1992). The SF-36 has excellent psychometric properties with test-

retest reliability coefficients of approximately .80 (Ware, Snow, & Kosinski, 1993). Additionally, the SF-36 has supported validity with strong correlations with other generic health surveys, as well as, measures of health distress, cognitive functioning, self-esteem, and family functioning (Kravitz, Greenfield, & Rogers, 1992; McHorney, Ware & Raczek, 1993; Ware, Kosinski & Bayliss, 1995; Ware et al., 1994; Ware et al., 1993). For the purposes of this study, the SF-36 was used as a measure of health-related quality of life (Arnold, Witzeman, Swank, McElroy & Keck, 2000; Meijer, Schene & Koeter, 2002).

Minnesota Multiphasic Personality Inventory - 2nd Edition (MMPI-2). The MMPI-2 is a 567-item true-false, self-report questionnaire that provides information on psychiatric symptoms, personality organization, and coping. The original inventory was developed by Hathaway and McKinley (MMPI; 1942), but was revised in 1989 by Butcher, Dahlstrom, Graham, Tellegen, and Kaemer. The MMPI has shown adequate reliability with test-retest correlations exceeding .70 (Greene, 1991), and average internal consistency correlations of .87 across a number of samples (Parker, Hanson, & Hunsley, 1988). Although the use of the MMPI with chronic pain patients has been debated (e.g., Prokop, 1986), based on a large body of research over the past fifty years, the instrument is generally considered valid for assessing psychological status in this population of patients (Weisberg & Keefe, 1999).

Wechsler Adult Intelligence Scale – Revised (WAIS-R). The WAIS-R is a widely used, individually administered intelligence test for adults developed by Wechsler (1981), with excellent psychometric properties (Anastasi, 1988).

Quantified Pain Drawing (QPD). This instrument is a non-verbal measure that consists of two sections: pain location and pain severity. In the first section, the subject is asked to mark the location of her/his pain symptoms on outlines of a human figure, front and back. This section is scored by superimposing a grid over the marked figures to determine the surface area affected, based on number of squares. In the second section, the subject is asked to rate the severity of her/his pain by placing a mark along a 10 centimeter line. Established cut-off points for interpretation are as follows: <4 (mild), 4-6 (moderate), >7 (severe) (Mooney, 1984; Mayer, Gatchel, & Polatin, 1992).

PRIDE Medical Case Management Disability Assessment and Initial Evaluation. At the beginning of the treatment program, the medical case management staff at PRIDE conducted a standardized interview of all patients. Information gathered during the interview was recorded on a standardized worksheet and included case settlement venue and status, financial situation, employment history, work status, and other information relevant to treatment considerations and resource planning.

PRIDE Quantitative Evaluation of Physical Functioning. Physical functioning was assessed before and after completion of the intensive rehabilitation program, and a Cumulative Physical Score was calculated. The Cumulative Physical Score is a weighted average score that represents the equivalent of a grade-point average for overall physical performance on a variety of functional capacity tests that include measures of range of motion and functional task performance.

PRIDE One-Year Interview. One-year following participation in the PRIDE functional restoration program, subjects were contacted for a structured phone interview consisting of seven general questions relevant to long-term program outcome measures. These outcome measures included: work return, defined as returning to work for any period of time after completing the PRIDE program; work retention, defined as working at the time of the follow-up interview; post-discharge healthcare utilization; post-discharge surgeries; recurrent injuries; subjective pain level; and case settlement.

Design and Statistical Analyses

The current study was designed to determine the predictive value of pre-treatment level of opioid use for short-term and long-term treatment outcomes of chronic pain rehabilitation. This study examined differences in demographic variables and treatment response outcomes as a function of reported level of opioid use. For purposes of the study, subjects were divided into two groups and five subgroups based on self-reported pre-treatment level of opioid use, as gathered from subjects' medical records.

Opioid Use and Demographic Variables. Subjects in both groups and each subgroup were compared on the following, using analyses of variance: age, years of education, length of disability (months), net salary at time of injury, and disability payments (pre- and 1-year follow-up). Subjects in both groups and each subgroup were compared on the following using Chi-square analyses: gender, race, pre-treatment surgery rate, presence of a prior work-related injury, whether

or not the subject has retained an attorney, whether or not the subject is receiving Social Security Disability Insurance (SSDI) or Supplemental Security Income (SSI), pre-treatment case settlement status, and program completion status.

Opioid Use and Socioeconomic/Health Variables. Subjects were compared by pre-treatment level of opioid use group and subgroup using Chi-square analyses on the following one-year treatment outcome variables: work return, work retention, presence of a post-treatment injury to the treated body part, post-treatment surgery to the treated body part, healthcare utilization, and case settlement status. Logistic regression analyses were performed to determine if pre-treatment level of opioid use was predictive of one-year socioeconomic and health outcomes.

Opioid Use and Psychosocial/Physical Variables. Univariate analyses of variance were used to identify differences between pre-treatment level of opioid use groups and among subgroups for the following pre-treatment variables: BDI, HAM-D, MVAS, OSW, SF-36, Cumulative Physical Score, Quantified Pain Drawing, and WAIS-R Full Scale IQ. Likewise, univariate analyses of variance (ANOVA) were used to identify differences between and among pre-treatment level of opioid use groups and subgroups, respectively, for the following post-treatment variables: BDI, HAM-D, MVAS, OSW, SF-36, Cumulative Physical Score, and Quantified Pain Drawing. To determine if differences in MMPI scores were present, a multivariate analysis of variance was performed. ANOVAs were then used to explore mean clinical scale score differences, and Chi-square analyses were used to examine differences in rates of clinical scale elevations. A

k-means cluster analysis was performed, in an attempt to identify homogenous clusters of cases, and rates of four common MMPI profiles were examined by Chi-square analyses. Linear regression analyses were performed to determine if pre-treatment level of opioid use was predictive of post-treatment physical and psychological scores.

CHAPTER FOUR

Results:

Basic Demographic Variables

As noted earlier, subjects in the study were classified in two different ways, based on their pre-treatment level of opioid use. The first classification was dichotomous, based on whether or not they were taking pre-treatment opioids. These two groups were labeled the YES group and the NO group, respectively. The second classification was based on dosage of pre-treatment opioids and yielded five subgroups, labeled the NO subgroup, the LOW subgroup, the MEDIUM subgroup, the HIGH subgroup, and the VERY HIGH subgroup. Results based on these two classifications are presented in the text and tables. Samples were evaluated on a variety of demographic variables, including: gender, age, race, years of education, and program completion. Statistical significance was set at the .05 level for all analyses performed, unless otherwise specified.

Basic Demographic Variables of the NO and YES Groups

Pre-treatment demographic characteristics of the NO and YES groups are presented in Tables 2 and 3, respectively. Analyses conducted to determine demographic differences between the NO and YES groups revealed no significant differences in gender, age, and years of education. Significant differences were found for racial representation and rate of program completion. Racial representation differed significantly, $\chi^2(3) = 20.20$, $\underline{p} < .001$, with Caucasians

being proportionally over-represented, and Hispanics being proportionally under-represented in the YES group. Program completion rates also differed significantly between the two groups, with 81.4% of the NO group and 74.0% of the YES group completing the Functional Restoration Program, $\chi^2(1) = 9.80$, $\underline{p} = .002$. The YES group was at higher risk of program non-completion, with the YES group being more than 1.5 times as likely as the NO group to discontinue treatment prematurely, OR = 1.54 (CI = 1.18, 2.02).

Basic Demographic Variables of the Opioid Subgroups

Pre-treatment demographic characteristics of the five opioid subgroups under consideration are presented in Tables 4 and 5. The subgroups did not show significant differences in gender, age, and years of education. However, the subgroups demonstrated significant differences in racial representation and rate of program completion. Racial representation varied significantly among the subgroups, $\chi^2(12) = 34.74$, $\underline{p} = .001$. Group representation by Caucasian individuals increased linearly as dosage level increased, from 48.6% in the LOW subgroup to 80.0% in the VERY HIGH subgroup. Conversely, the proportion of Hispanic individuals decreased as dosage level increased, from 24.6% in the LOW subgroup to 8.0% in the VERY HIGH subgroup. Chi-square analysis conducted for the five opioid subgroups revealed significant differences in the proportions of patients successfully completing the PRIDE Functional Restoration Program, $\chi^2(4) = 15.74$, $\underline{p} = .003$. Specifically, 81.5% of the NO subgroup, 76.0%

of the LOW subgroup, 68.7% of the MEDIUM subgroup, 68.1% of the HIGH subgroup, and 70.6% of the VERY HIGH subgroup completed the program.

CHAPTER FIVE

Results:

Pre-treatment Socioeconomic and Health Variables

The samples were also evaluated on pre-treatment socioeconomic and health variables associated with pre-injury and pre-treatment. These socioeconomic variables included: weekly net salary at time of injury; workers' compensation rate; pre-treatment case settlement; attorney retention; and SSDI/SSI. Health variables included: length of disability; presence of a prior work-related injury; number of prior work-related injuries; and pre-treatment surgery.

Pre-treatment Socioeconomic and Health Variables of the NO and YES Groups

Pre-treatment socioeconomic and health variables of the NO and YES groups are presented in Tables 6 and 7, respectively. Analyses conducted to determine socioeconomic differences between the NO and YES groups yielded no significant differences in mean weekly net salary at time of injury, pre-treatment case settlement, or the proportion of patients who retained an attorney prior to treatment. Among those receiving workers' compensation, mean weekly compensation rates differed significantly, with the NO group reporting higher mean weekly compensation rates ($\underline{M}$ = 354.12, $\underline{SD}$ = 135.24) than the YES group ($\underline{M}$ = 328.60, $\underline{SD}$ = 131.71), $\underline{F}(1, 787) = 7.21$, $\underline{p}$ = .007. Proportions of patients

receiving SSDI or SSI also differed significantly. The YES group (2.6%) was four times more likely than the NO group (0.6%) to be receiving SSDI or SSI $\chi^2(1) = 7.20$, $p = .007$; OR = 4.07 (CI = 1.34, 12.34). Analyses revealed significant differences in health variables, including length of disability, prior work-related injury, and pre-treatment surgery. Total length of disability differed, with the NO group showing significantly fewer months ($M = 11.05$, $SD = 10.76$) than the YES group ($M = 15.60$, $SD = 17.25$), $F(1, 1070) = 27.14$, $p < .001$. The YES group (43.5%) was nearly 1.5 times as likely as the NO group (35.8%) to report a prior work-related injury, $\chi^2(1) = 6.26$, $p = .012$; OR = 1.38 (CI = 1.07, 1.78). The YES group (49.5%) was also more than 1.5 times as likely as the NO group (44.8%) to have a pre-treatment surgery, $\chi^2(1) = 15.47$, $p < .001$; OR = 1.63 (CI = 1.28, 2.08).

Pre-treatment Socioeconomic and Health Variables of the Opioid Subgroups

Pre-treatment socioeconomic and health variables for the five opioid subgroups are presented in Tables 8 and 9. The subgroups did not show significant differences in weekly net salary at time of injury, pre-treatment case settlement, attorney retention, or SSDI/SSI. Total length of disability (months), however, differed significantly among the subgroups, $F(4, 961) = 8.27$, $p < .001$. Post-hoc analyses revealed that the NO subgroup ($M = 11.08$), the LOW subgroup ($M = 15.18$), and the MEDIUM subgroup ($M = 18.65$) all differed significantly from each another. The MEDIUM subgroup ($M = 18.65$) also differed from the HIGH subgroup ($M = 13.02$). The VERY HIGH subgroup ($M =$

15.66) did not differ significantly from any of the other subgroups. Among those receiving workers' compensation, mean weekly compensation rates varied significantly among the subgroups, $\underline{F}(4, 707) = 2.417$, $\underline{p} = .047$. Chi-square analysis revealed significant differences in the proportions of patients with pre-treatment surgeries, $\chi^2(4) = 25.07$, $\underline{p} < .001$. Results revealed lower pre-treatment surgery rates for the NO (37.7%) and LOW (43.1%) subgroups, and higher rates for the MEDIUM (58.1%), HIGH (52.3%), and VERY HIGH (64.4%) subgroups. However, Chi-square analysis failed to reveal significant differences in the proportion of patients reporting prior work-related injury, $\chi^2(4) = 8.48$, $\underline{p} = .076$. The NO subgroup showed the lowest proportion of prior work related injuries at 35.80%, while the HIGH subgroup showed the highest proportion at 48.30%.

CHAPTER SIX

Results:

Psychosocial Variables-Depression and Disability

The current chapter presents analyses of various pre-treatment and post-treatment psychosocial variables, including the BDI, HAM-D, MVAS, OSW, and SF-36. The depression and disability measures were administered to all patients prior to, and upon completion of, the functional restoration program. Because the post-treatment measures were only gathered from those who completed the program, the post-treatment sample is smaller than the pre-treatment sample (which is reflected in the tables). Once again, comparisons between the NO and YES groups, and among the five opioid subgroups, will be presented. Linear regression analyses, used to evaluate pre-treatment level of opioid use as a predictor of depression and disability outcomes, are presented at the end of the chapter.

NO AND YES GROUP COMPARISONS

Pre-treatment Depression and Disability Measures

Pre-treatment depression and disability variables, except the SF-36, were analyzed in two ways. First, these measures were analyzed as continuous variables by ANOVA to explore group differences based on level of opioid use. Second, in order to further explore their clinical significance, these measures were analyzed as categorical variables based on level of symptom severity using Chi-

square analyses. BDI scores were analyzed as categorical variables based on the following cutoffs: <10 for absence of depression; 10-18 for mild to moderate depression; 19-29 for moderate to severe depression; and >29 for severe depression (Beck, Steer, & Garbin, 1988). HAM-D scores were analyzed as categorical variables based on the following cutoffs: <12 for none to minimal symptoms; 12-20 for mild to moderate symptoms; 21-29 for moderate to severe; and 30+ for severe symptoms (Hedlung & Vieweg, 1979). MVAS scores were analyzed as categorical variables based on the following cutoffs: 1-40 for mildly disabling; 41-70 for moderately disabling; 71-100 for severely disabling; 101-130 for very severely disabling; and 131-150 for extremely disabling (Anagnostis, Mayer, Gatchel, & Proctor, 2003). OSW scores were analyzed as categorical variables based on the following cutoffs: 0-20 minimal disability, 21-40 moderate disability, 41-60 severe disability, 61-80 crippled, 81-100 either bedbound or exaggerating symptoms (Fairbanks et al., 1980). Results of these analyses are presented in Tables 10 through 16.

Pre-treatment BDI. The NO group ($\underline{M}$ = 13.71, SD = 9.33) averaged significantly lower pre-treatment BDI scores than the YES group ($\underline{M}$ = 16.43, $\underline{SD}$ = 10.07), $\underline{F}(1, 1008) = 19.88$, $\underline{p} < .001$. Chi-square analyses revealed that the proportions of patients reporting various categories of depressive symptoms differed between the NO and YES groups, $\chi^2(3) = 20.67$, $p < .001$. Within the NO group, 39.6% of patients reported an absence of depressive symptoms, 34.8% reported mild to moderate depressive symptoms, 19.1% reported moderate to severe depressive symptoms, and only 6.4% reported severe depressive

symptoms. Within the YES group, 27.2% of patients reported an absence of depressive symptoms, 38.4% reported mild to moderate depressive symptoms, 23.4% reported moderate to severe depressive symptoms, and 11.0% reported severe depressive symptoms. Results for pre-treatment BDI are presented in Tables 10 through 12.

Pre-treatment HAM-D. The NO group ($\underline{M}$ = 13.41, $\underline{SD}$ = 6.32) averaged significantly lower pre-treatment HAM-D scores than the YES group ($\underline{M}$ = 15.41, $\underline{SD}$ = 5.70), $\underline{F}(1, 1000 = 27.56)$, $\underline{p} < .001$. Chi-square analyses revealed that the proportions of patients rated with various symptom levels differed significantly between the NO and YES groups, $\chi^2(3) = 22.55$, $\underline{p} < .001$. Within the NO group, 38.9% rated none to minimal symptoms, 49.6% rated mild to moderate symptoms, and 10.7% rated moderate to severe symptoms. Within the YES group, 26.2% rated none to minimal symptoms, 55.4% rated mild to moderate symptoms, and 17.6% rated moderate to severe symptoms. Thus, the YES group showed higher proportions of patients with mild to moderate, and moderate to severe, symptoms. In both the NO and YES groups, 0.8% of patients rated severe symptoms. Results for pre-treatment HAM-D are presented in Tables 10, 11, and 13.

Pre-treatment MVAS. The NO group ($\underline{M}$ = 85.77, $\underline{SD}$ = 26.08) reported significantly lower MVAS scores than the YES group ($\underline{M}$ = 94.92, $\underline{SD}$ = 20.51), $\underline{F}(1, 1006) = 37.85$, $\underline{p} < .001$. Chi-square analyses revealed that the proportions of patients reporting various levels of disability differed significantly between the NO and YES groups, $\chi^2(4) = 51.13$, $\underline{p} < .001$. Within the NO group, 3.0%

reported extremely, 26.5% reported very severely, 43.0% reported severely, 22.2% reported moderately, and 5.3% reported mildly disabling symptoms. Within the YES group, 2.1% reported extremely, 39.2% reported very severely, 46.9% reported severely, 11.5% reported moderately, and 0.4% reported mildly disabling symptoms. To further explore MVAS, scores were divided into two ranges: not extreme (0-70) and extreme (71-150). Proportions of patients scoring in these ranges were then analyzed by Chi-square. The NO and YES groups differed significantly, with 72.5% of the NO group and 88.1% of the YES group reporting MVAS scores in the extreme range, $\chi^2(1) = 38.12$, $\underline{p} < .001$. Thus, the NO group was nearly three times more likely than the YES group to report MVAS scores in the not extreme range, OR = 2.81 (CI = 2.01, 3.93). Results for pre-treatment MVAS are presented in Tables 10, 11, 14 and 15.

Pre-treatment OSW. The NO group averaged significantly lower pre-treatment OSW scores than the YES group, $\underline{F}(1, 933) = 39.22$, $\underline{p} < .001$. The average score of the NO group fell in the moderate disability range ($\underline{M} = 35.32$, $\underline{SD} = 14.57$), while the average score of the YES group fell in the severe disability range ($\underline{M} = 41.35$, $\underline{SD} = 14.86$). Chi-square analyses revealed that the proportions of patients reporting various levels of disability differed significantly between the NO and YES groups, $\chi^2(4) = 42.55$, $\underline{p} < .001$. Within the NO group, 15.4% of patients reported minimal disability, while 54.3% and 25.5% of patients reported moderate disability and severe disability, respectively. Four percent of patients in the NO group reported crippling disability, and less than 1% reported scores that would suggest being bedbound or the exaggeration of disability. In the YES

group, 6.5% of patients reported minimal disability, 47.6% reported moderate disability, and 33.7% reported severe disability. Another 11.4% reported crippling disability, and less than 1% reported scores that would suggest being bedbound or the exaggeration of disability. Results for pre-treatment OSW are presented in Tables 10, 11, and 16.

Pre-treatment SF-36. The SF-36 yields two summary scores, one for the mental health scale (SF-36MHS) and one for the physical health scale (SF-36PHS). The NO group ($\underline{M}$ = 40.12, $\underline{SD}$ = 9.61) averaged significantly higher (more favorable) pre-treatment SF-36MHS scores than the YES group ($\underline{M}$ = 38.09, $\underline{SD}$ = 9.60), $\underline{F}(1, 971) = 10.88$, $\underline{p}$ = .001. Likewise, the NO group ($\underline{M}$ = 30.83, $\underline{SD}$ = 5.84) averaged significantly higher (more favorable) pre-treatment SF-36PHS scores than the YES group ($\underline{M}$ = 29.61, $\underline{SD}$ = 5.92), $\underline{F}(1, 971) = 10.44$, $\underline{p}$ = .001. Pre-treatment SF-36 scores and analyses are presented in Tables 10 and 11.

Post-treatment Depression and Disability Measures

Post-treatment depression and disability variables were analyzed by ANOVA and, when appropriate, ANCOVA. To further explore the clinical significance of depression and disability variables, categorical analyses were conducted using Chi-square analyses. Additionally, pre- to post-treatment delta scores were calculated and analyzed using ANOVA. Results are shown in Tables 17 through 23.

Post-treatment BDI. The NO group (M = 8.06, SD = 6.93) reported significantly lower post-treatment BDI scores than the YES group (M = 9.79, SD = 7.87; F(1, 881) = 12.10, p < .001). When controlling for differences in pre-treatment BDI scores, however, post-treatment BDI scores were not significantly different between the groups, F(1, 818) = 3.29, p =.070.

Chi-square analyses revealed that the proportions of patients reporting various symptom levels did not differ significantly at post-treatment between the NO and YES groups, $\chi^2(3) = 7.73$, p = .05. Within the NO group, 67.5% reported an absence of depression, 23.1% reported mild to moderate symptoms, 8.0% reported moderate to severe symptoms, and 1.5% reported severe symptoms. Within the YES group, 59.6% of patients reported an absence of depression, 25.9% reported mild to moderate symptoms, 12.1% reported moderate to severe symptoms, and 2.5% reported severe symptoms.

Patients who took opioids prior to starting function restoration did not show significantly different improvements in levels of depressive symptoms, as measured by the BDI, than did patients who did not take pre-treatment opioids, F(1, 815) = 0.537, p = .464. Results for post-treatment BDI are presented in Tables 17 through 19.

Post-treatment HAM-D. The YES group did not show significantly different improvements in depressive symptoms than the NO group as measured by the HAM-D, F(1, 807) = .043, p = .836.

Chi-square analyses revealed that the proportions of patients receiving various symptom ratings differed significantly at post-treatment between the NO

and YES groups, $\chi^2(2) = 22.84$, $p < .001$. Within the NO group, 79.2% rated none to minimal symptoms of depression, 20.0% rated mild to moderate symptoms, and 0.8% rated moderate to severe symptoms. Within the YES group, 65.4% of patients received none to minimal symptom ratings, 31.6% received mild to moderate symptom ratings, and 3.0% received moderate to severe symptom ratings. No patients in either the NO or YES groups received symptom ratings in the severe range at post-treatment. Results for post-treatment HAM-D are displayed in Tables 17, 18, and 20.

Post-treatment MVAS. The NO group demonstrated significantly lower post-treatment MVAS scores ($M = 59.41$, $SD = 28.20$) than the YES group ($M = 70.24$, $SD = 28.15$), $F(1, 817) = 12.81$, $p < .001$, when controlling for pre-treatment MVAS scores.

Chi-square analyses showed that the proportions of patients reporting various levels of disability differed significantly at post-treatment between the NO and YES groups, $\chi^2(4) = 32.13$, $p < .001$. Within the NO group, 0.6% reported extremely, 7.1% reported very severely, 26.2% reported severely, 39.1% reported moderately, and 27.0% reported mildly disabling symptoms. Within the YES group, 0.7% reported extremely, 14.0% reported very severely, 36.7% reported severely, 32.0% reported moderately, and 16.5% reported mildly disabling symptoms. To further explore MVAS, scores were divided into two ranges: not extreme (0-70) and extreme (71-150). Proportions of patients scoring in these ranges were then analyzed by Chi-square. The NO and YES groups differed significantly, with 33.9% of the NO group and 51.5% of the YES group reporting

MVAS scores in the extreme range, $\chi^2(1) = 27.88$, $p < .001$. Thus, the YES group was twice as likely as the NO group to report extreme MVAS scores, OR = 2.07 (CI = 1.58, 2.72).

Patients who took pre-treatment opioids did not show significantly different changes in pre-treatment to post-treatment MVAS scores compared with patients who did not take pre-treatment opioids, $F(1, 814) = 3.287$, $p = .070$. Results for post-treatment MVAS are presented in Tables 17, 18, 21 and 22.

Post-treatment OSW. When differences in pre-treatment OSW were controlled, average post-treatment OSW scores did not differ significantly between the NO and YES groups, $F(1, 168) = 1.15$, $p = .268$. The average post-treatment score of the NO group fell on the cusp between the minimal and moderate disability range ($M = 20.66$, $SD = 7.06$), while the average score of the YES group fell in the low end of the moderate disability range ($M = 23.56$, $SD = 7.58$). Pre- to post-treatment change scores also did not differ between the NO and YES groups, $F(1, 167) = .009$, $p = .923$. Chi-square analyses revealed that the proportions of patients reporting various levels of disability at post-treatment differed significantly between the NO and YES groups, $\chi^2(2) = 8.29$, $p = .016$. Within the NO group, 54.3% of patients reported minimal disability, while 45.7% of patients reported moderate disability. In the YES group, 35.2% of patients reported minimal disability, 63.6% reported moderate disability, and 1.1% reported severe disability. Results for post-treatment OSW are presented in Tables 17, 18, and 23.

Post-treatment SF-36. The NO group (M = 46.67, SD = 8.76) did not report significantly different post-treatment SF-36MHS scores than the YES group (M = 46.29, SD = 9.20; F(1, 241) = 0.11, p = .741). Likewise, mean SF-36MHS scores did not differ significantly when controlling for pre-treatment SF-36MHS scores, F(1, 222) = 0.04, p = .842. However, the NO group (M = 36.68, SD = 6.97) reported significantly higher post-treatment SF-36PHS scores than the YES group (M = 34.84, SD = 6.24; F(1, 241) = 4.50, p = .035).

Comparisons between the NO and YES groups did not reveal significant differences in pre-treatment to post-treatment changes in SF-36MHS [F(1, 223) = 1.416, p = .235)], or SF-36PHS [F(1, 223) = .157, p = .692] scores. Results for post-treatment SF-36 are presented in Tables 17 and 18.

OPIOID SUBGROUPS COMPARISONS

Pre-treatment Depression and Disability Measures

Pre-treatment BDI. Pre-treatment BDI scores differed significantly among the five opioid subgroups, F(4, 903) = 11.03, p < .001. Post-hoc analyses showed significant differences between the NO subgroup (M = 13.74) and all other subgroups except the MEDIUM subgroup (M = 15.10). The LOW subgroup (M = 15.88) differed significantly from the NO (M = 13.74) and VERY HIGH subgroups (M = 23.02). The MEDIUM subgroup (M = 15.10) differed only from the VERY HIGH subgroup (M = 23.02), and the HIGH subgroup (M = 17.73) differed only from the NO subgroup (M = 13.74). The VERY HIGH

subgroup (M = 23.02) differed from all other subgroups except the HIGH subgroup (M = 17.73). Results are displayed in Tables 24 and 25.

Among the five opioid subgroups, Mantel-Haenszel Chi-square analysis revealed a significant linear trend in the proportion of patients reporting various categories of depressive symptoms, $\chi^2(1) = 28.61$, $p < .001$. Within the NO subgroup, the greatest proportion of patients reported an absence of depressive symptoms (39.5%). Approximately 35% of the NO subgroup reported mild to moderate depressive symptoms, while 19.2% reported moderate to severe, and 6.5% reported severe, depressive symptoms. The MEDIUM subgroup demonstrated similar proportions with 37.2% reporting an absence of depressive symptoms, 31.4% reporting mild to moderate, 20.9% reporting moderate to severe, and 10.5% reporting severe depressive symptoms. Approximately 42.1% of the LOW subgroup reported mild to moderate depressive symptoms, with 25.1% and 24.6% reporting an absence and moderate to severe depressive symptoms, respectively. In the LOW subgroup, 8.2% of patients reported severe depressive symptoms. Approximately 32.2% of the HIGH subgroup reported mild to moderate depressive symptoms, with 27.1% and 27.1% reporting an absence and moderate to severe depressive symptoms, respectively. In the HIGH subgroup, 13.6% of patients reported severe depressive symptoms. Within the VERY HIGH subgroup, a greater proportion of patients reported more severe depressive symptoms, with 29.3% and 29.3% reporting moderate to severe, and severe, depressive symptoms, respectively. Approximately one-quarter (26.8%)

of the VERY HIGH subgroup reported mild to moderate depressive symptoms, and 14.6% reported an absence of depressive symptoms (Table 26).

Pre-treatment HAM-D. Pre-treatment HAM-D scores increased among the five opioid subgroups as pre-treatment level of opioid increased, resulting in significant differences among the subgroups, $\underline{F}(4, 895) = 8.952$, $\underline{p} < .001$. Post-hoc analyses revealed significant differences between the NO subgroup ($\underline{M}$ = 13.43, $\underline{SD}$ = 6.30) and all other subgroups except the MEDIUM subgroup ($\underline{M}$ = 15.26, $\underline{SD}$ = 5.68). Results are presented in Tables 24 and 25.

Among the five opioid subgroups, Mantel-Haenszel Chi-square analysis revealed a significant linear trend in the proportions of patients rated with various levels of pre-treatment depressive symptoms, $\chi^2(1) = 22.86$, $\underline{p} < .001$. In general, approximately half of the patients in all of the opioid subgroups received mild to moderate symptom ratings, with increasing proportions of patients receiving moderate to severe symptom ratings in higher opioid subgroups. Within the NO subgroup, the greatest proportion of patients received mild to moderate symptom ratings (49.7%). Nearly forty percent (38.8%) rated none to minimal symptoms, while 10.7% and <1% rated moderate to severe, and severe, symptoms, respectively. Within the LOW subgroup, 27.1% of patients rated none to minimal symptoms, 55.2% rated mild to moderate symptoms, while, 17.2% and <1% rated moderate to severe, and severe symptoms, respectively. The MEDIUM subgroup yielded similar proportions, with 30.2% of patients rated none to minimal symptoms, 53.2% rated mild to moderate symptoms, 15.0% rated moderate to severe symptoms, and 1.2% rated severe symptoms. In the HIGH subgroup, a

greater proportion of patients rated moderate to severe symptoms (25.4%), while no patients rated severe symptoms. Relative to the lower opioid subgroups, fewer patients in the HIGH subgroup rated none to minimal symptoms (23.7%). Approximately half of the patients in the HIGH subgroup (50.8%) rated mild to moderate symptoms. Within the VERY HIGH subgroup, a smaller proportion of patients rated none to minimal symptoms (17.5%) and a greater proportion of patients rated moderate to severe (27.5%) and severe (2.5%) symptoms, relative to the other opioid subgroups. Slightly over half (52.5%) of the VERY HIGH subgroup received mild to moderate symptom ratings (Table 27).

Pre-treatment MVAS. Pre-treatment MVAS scores differed significantly among the five opioid subgroups, $\underline{F}(4, 901) = 10.84$, $\underline{p} < .001$. Post-hoc analyses revealed that the significance was accounted for by differences between the NO subgroup and all other subgroups. No other subgroups differed from each other. These results are presented in Tables 24 and 25.

Among the five opioid subgroups, Mantel-Haenszel Chi-square analysis revealed a significant linear trend in the proportion of patients reporting various levels of disability, $\chi^2(1) = 31.34$, $\underline{p} < .001$. To further explore MVAS, scores were divided into two ranges: not extreme (0-70) and extreme (71-150). Proportions of patients scoring in these ranges were then analyzed by Chi-square. The proportion of patients reporting disability symptoms in the extreme range differed significantly among the five opioid subgroups, $\chi^2(4) = 41.70$, $\underline{p} < .001$. The NO subgroup showed the lowest proportion of extreme scores at 72.5%,

while the VERY HIGH subgroup showed the highest proportion of extreme scores at 95.1% (Tables 28 and 29).

Pre-treatment OSW. Among the five opioid subgroups, pre-treatment OSW scores differed significantly, $\underline{F}(4, 838) = 13.40$, $\underline{p} < .000$. Post-hoc analyses indicated that this difference is accounted for by differences between the NO subgroup ($\underline{M} = 35.32$, $\underline{SD} = 14.57$) and all other subgroups, and differences between the LOW subgroup ($\underline{M} = 39.86$, $\underline{SD} = 14.68$) and the MEDIUM subgroup ($\underline{M} = 45.58$, $\underline{SD} = 15.88$). The NO and LOW subgroups averaged pre-treatment OSW scores in the moderate disability range, while all other subgroups averaged scores in the severe disability range. Results are displayed in Tables 24 and 25.

Analysis of pre-treatment OSW scores coded as a categorical variable showed significant differences in the proportion of patients reporting various levels of disability among the five opioid subgroups, Mantel-Haenszel $\chi^2(1) = 38.04$, $\underline{p} < .001$. The proportion of patients reporting minimal disability ranged from 15.4% of the NO subgroup to 1.8% of the HIGH subgroup, while the proportion of patients reporting severe disability ranged from 25.5% in the NO subgroup to 42.1% in the MEDIUM subgroup. In the NO subgroup, 4.0% of patients reported crippling disability, while 19.3% of patients in the HIGH subgroup reported disability in this range (Table 30).

Pre-treatment SF-36. Among the five opioid subgroups, pre-treatment SF-36MHS scores differed significantly, $\underline{F}(4, 873) = 4.33$, $\underline{p} = .002$ (Tables 24 and 25). Post-hoc analyses showed that this difference is accounted for by

significant differences between the NO subgroup (M = 40.12, SD = 9.61) and the LOW subgroup (M = 37.86, SD = 9.24), and the NO subgroup and the VERY HIGH subgroup (M = 34.91, SD = 8.50). Pre-treatment SF-36PHS scores also differed significantly, F(4, 873) = 3.471, p = .008. Post-hoc analyses revealed that the difference was accounted for by differences between the NO subgroup (M = 30.83, SD = 5.84) and the HIGH subgroup (M = 28.62, SD = 5.19).

Post-treatment Depression and Disability Measures

Post-treatment BDI. For the five opioid subgroups, results for post-treatment BDI are presented in Tables 31 and 32. ANCOVA yielded no significant differences among the five opioid subgroups in post-treatment BDI scores, when controlling for pre-treatment BDI scores, F(4, 732) = 1.21, p =.304.

Mantel-Haenszel Chi-square analysis revealed a significant linear trend in the proportion of patients reporting various levels of depressive symptoms at post-treatment, $\chi^2(1) = 12.41$, p < .001. The majority of patients in all opioid subgroups reported an absence of depression. However, the proportion of patients reporting moderate to severe, and severe, symptoms increased with pre-treatment opioid level (Table 33).

The opioid subgroups, however, did show significant differences in pre- to post-treatment improvement, F(4, 731) = 2.72, p = .029. Post-hoc analyses revealed that the significant difference was attributable to differences between the HIGH and VERY HIGH subgroups, versus the other subgroups. The HIGH and VERY HIGH subgroups did not differ significantly from each other. The HIGH

and VERY HIGH subgroups showed greater improvements in self-reported depressive symptoms than patients in lower pre-treatment opioid subgroups (Table 31 and 32).

Post-treatment HAM-D. Results for post-treatment HAM-D are displayed in Tables 31 and 32. Among the five opioid subgroups, an ANOVA yielded significant differences in post-treatment HAM-D scores, $\underline{F}(4, 785) = 8.27$, $\underline{p} < .001$. Post-hoc analyses revealed these differences were accounted for by differences between the NO subgroup ($\underline{M} = 7.88$, $\underline{SD} = 4.41$) and the LOW subgroup ($\underline{M} = 9.76$, $\underline{SD} = 4.74$), and between the NO subgroup and the VERY HIGH subgroup ($\underline{M} = 10.81$, $\underline{SD} = 5.95$) only. Changes in pre-treatment to post-treatment HAM-D scores did not differ significantly among the five opioid subgroups, $\underline{F}(4, 723) = .698$, $\underline{p} = .594$.

Mantel-Haenszel Chi-square analysis revealed a significant linear trend in the proportion of patients rated with various levels of depressive symptoms at post-treatment, $\chi^2(1) = 23.10$, $\underline{p} < .001$. While the majority of patients in all opioid subgroups received none to minimal symptom ratings, greater proportions of patients received none to minimal symptom ratings in lower opioid subgroups. Likewise greater proportions of patients received mild to moderate and moderate to severe symptom ratings in higher opioid subgroups (Table 34).

Post-treatment MVAS. Significant group differences were also noted by ANCOVA among the five opioid subgroups for post-treatment MVAS scores, $\underline{F}(4, 731) = 3.28$, $\underline{p} = .011$. The NO subgroup showed the lowest post-treatment MVAS scores ($\underline{M} = 58.73$, $\underline{SD} = 28.49$). While the MEDIUM ($\underline{M} = 67.43$, $\underline{SD} =$

26.97), HIGH (M = 67.75, SD = 29.72), and VERY HIGH (M = 68.80, SD = 30.39) subgroups showed higher post-treatment MVAS scores, the LOW subgroup (M = 70.87, SD = 28.21) showed the highest scores. However, pre-treatment to post-treatment changes in MVAS scores did not differ significantly among the five opioid subgroups, $F(4, 730) = 1.779$, $p = .131$. Results for post-treatment MVAS are presented in Tables 31 and 32.

Mantel-Haenszel Chi-square analysis revealed a significant linear association in the proportion of patients reporting various levels of disability at post-treatment, $\chi^2(1) = 14.94$, $p < .001$. To further explore MVAS, scores were divided into two ranges: not extreme (0-70) and extreme (71-150). Proportions of patients scoring in these ranges were then analyzed by Chi-square. The proportion of patients reporting disability symptoms in the extreme range differed significantly among the five opioid subgroups, $\chi^2(4) = 25.50$, $p < .001$. The NO subgroup showed the lowest proportion of extreme scores at 33.9%, while the LOW subgroup showed the highest proportion of extreme scores at 53.7% (Tables 35 and 36).

Post-treatment OSW. Patients who completed the functional restoration program did not show significant differences in post-treatment OSW mean scores based on opioid subgroup membership, $F(4, 171) = .690$, $p = .600$. Similarly, mean pre-treatment to post-treatment change scores did not differ significantly, $F(4, 139) = 1.471$, $p = .214$ (Tables 31 and 32). When post-treatment OSW was analyzed as a categorical variable all patients fell in the minimal or moderate disability range, and the proportion of patients falling into these two ranges did

not differ significantly among the five opioid subgroups, Mantel-Haenszel $\chi^2(1) = 1.197$, $p = .274$ (Table 37).

Post-treatment SF-36. Among the five opioid subgroups, patients who completed the functional restoration program did not show significant differences in post-treatment SF-36MHS or SF-36PHS scores, even when controlling for pre-treatment scores.

Likewise, examination of change scores among the five opioid subgroups, did not reveal significant differences for SF-36MHS [$F(4, 191) = 1.018$, $p = .399$] or SF-36PHS [$F(4, 191) = 1.319$, $p = .264$]. Results are displayed in Tables 31 and 32.

LINEAR REGRESSION ANALYSES-DEPRESSION AND DISABILITY

Linear regression analyses were used to evaluate pre-treatment level of opioid use as a predictor of depression and disability outcomes, as measured by post-treatment BDI, HAM-D, MVAS, OSW, and SF-36.

Prediction of Post-treatment BDI Scores

A linear regression analysis was conducted to evaluate the prediction of post-treatment BDI scores from pre-treatment opioid use (average daily milligrams of morphine). Results showed that pre-treatment opioid dose significantly predicted post-treatment BDI scores, $F(1, 807) = 12.74$, $p < .001$, accounting for approximately 2% of the variance.

Prediction of Post-treatment HAM-D Scores

A linear regression analysis was conducted to evaluate the prediction of post-treatment HAM-D scores from pre-treatment opioid use (average daily milligrams of morphine). Results indicated that pre-treatment opioid dose significantly predicted post-treatment HAM-D scores, $F(1, 805) = 17.86$, $p < .001$, accounting for approximately 2% of the variance.

Prediction of Post-treatment MVAS Scores

A linear regression analysis was conducted to evaluate the prediction of post-treatment MVAS scores from pre-treatment opioid use (average daily milligrams of morphine). Results demonstrated that pre-treatment opioid dose significantly predicted post-treatment MVAS scores, $F(1, 808) = 6.38$ $p = .012$, accounting for approximately 1% of the variance.

Prediction of Post-treatment OSW Scores

A linear regression analysis was conducted to evaluate the prediction of post-treatment OSW scores from pre-treatment opioid use (average daily milligrams of morphine). Results showed that pre-treatment opioid dose did not predict post-treatment OSW scores, $F(1, 181) = .008$, $p = .929$.

Prediction of Post-treatment SF-36 Scores

Linear regression analyses conducted to evaluate the prediction of post-treatment SF-36 scores from pre-treatment opioid use (average daily milligrams

of morphine) did not yield significant results for either SF-36MHS [$F(1, 220 = 1.16$ $p = .282$] or SF-36PHS [$F(1, 220) = 1.85$ $p = .176$].

CHAPTER SEVEN

Results:

Psychosocial Variables-

Personality and Intelligence

The current chapter presents analyses of personality and intelligence variables, specifically the MMPI Validity and Clinical Scales, and the WAIS-R Verbal, Performance, and Full Scale IQs. Several approaches were utilized to examine the relationship between pre-treatment level of opioid use and MMPI scores, including an analysis of mean clinical scale scores, an analysis of clinical scale elevations, a *k*-means cluster analysis, and finally, an examination of four profile types commonly discussed in the psychiatric literature. A description of each of these approaches is given here, and results from these analyses are presented in the same order in the chapter. The results of these analyses are displayed in Tables 38 through 47.

First, multivariate analysis of variance (MANOVA) was utilized to determine the effect of pre-treatment opioid use on MMPI validity and clinical scales, followed by ANOVAs on each clinical scale using a Bonferroni correction. Second, Chi-square analyses were performed on the proportion of patients showing elevations on each clinical scale.

To further explore the relationship between pre-treatment opioid use and MMPI profiles, the third approach utilized a *k*-means cluster analysis. *K*-means cluster analysis uses Euclidean distance to find homogenous clusters of cases.

Initial cluster centers are chosen in a first pass of the data. Each subsequent iteration groups cases based on nearest Euclidean distance to the mean of the cluster. The researcher must specify the number of clusters desired, K, in advance. Thus, the cluster center changes with each iteration. The process continues until the cluster means do not shift more than a given cut-off value or the iteration limit is reached.

In the present study, *k*-means cluster analysis was performed to attempt to replicate four homogenous clusters of MMPI profiles identified by Jordan (1996). These four clusters were later re-identified in heterogeneous samples of chronic musculoskeletal pain patients by Robinson (1998), Evans (1999), Dersh (2000), and Proctor (2001). The four profile clusters have been termed defended, distressed, somatoform, and depressed. While these one-word labels are convenient, they do not adequately describe the complexity of the four profile clusters.

Previous studies (e.g., Jordan, 1996; Evans, 1999) have offered the following descriptions of personality characteristics and behavioral patterns associated with each of the four MMPI profile clusters. The defended cluster is marked by a moderate elevation on Scale L and a lack of elevations on the clinical scales, except for a mild elevation on Scale 1. This profile indicates that individuals in the defended cluster may be responding in a moderately guarded and defensive manner. These individuals may be overly concerned and rigid about matters of self-control, morality, and conventionality. They may also be unwilling or unable to acknowledge, either to themselves or others, that they are

experiencing emotional difficulties (Graham, 1993; Friedman, Lewak, Nichols, & Webb, 2001).

MMPI profiles in the distressed cluster indicate a "plea for help." Individuals in this cluster are desperately attempting to elicit attention from others. They are easily frustrated and tend to act out their emotional discomfort. The profile associated with the distressed cluster suggests dissatisfaction with self and others, as well as limited insight into one's difficulties. This profile is often associated with a severe decline in psychological functioning. Patients in the present study, who are being treated in an outpatient, non-psychiatric facility, however, are unlikely to show such a severe level of decompensation (Graham, 1993; Friedman, Lewak, Nichols, & Webb, 2001).

As the name suggests, the somatoform cluster is associated with difficulties in expressing emotional distress directly and the tendency to manifest emotional distress indirectly as physical symptoms. Thus, stress is often converted into physical symptoms, and psychological interpretations are resisted. Individuals with somatoform profiles are similar to those in the defended cluster in their inability and/or unwillingness to express emotional distress (Graham, 1993; Friedman, Lewak, Nichols, & Webb, 2001).

Patients in the depressed cluster do not necessarily meet full diagnostic criteria for a depressive episode or report depressive symptoms as their chief complaint. However, MMPI profiles in this cluster suggest unexaggerated psychological distress, dissatisfaction with self, and the experience of cognitive and physical correlates of depression. Like those in the somatoform cluster,

individuals in the depressed cluster have limited psychological insight and numerous somatic complaints. Individuals in the depressed cluster, however, are psychologically more uncomfortable than individuals in the somatoform cluster. Therefore, they tend to be more open to psychological interpretations and treatment (Graham, 1993; Friedman, Lewak, Nichols, & Webb, 2001).

The fourth approach to analyzing MMPI scores examined common profiles, or code types, believed to be useful in predicting treatment outcomes. The first two profiles have been found in the psychiatric and chronic pain literature for decades. These include the Conversion V and the Neurotic Triad, which represent elevations on clinical scales 1 and 3; and 1, 2, and 3, respectively. The third profile included in the analysis is common in the psychiatric literature, but has only recently been explored for its utility with patients who have chronic disabling work-related spinal injuries (Kidner, Mayer, & Gatchel, 2004; Gatchel, Mayer, & Eddington, 2006). This third profile consists of four or more clinical elevations, and is frequently referred to as a "Floating Profile" (Friedman, Lewak, Nichols, & Webb, 2001).

More recently, Gatchel, Mayer, and Eddington (2006) have coined these multiple elevations, the Disability Profile, based on its high prevalence rates among patients with chronic occupational spinal disorders. Prevalence rates of these three profiles, along with Normal profiles, were examined across the pre-treatment opioid groups and subgroups using Chi-square and Mantel-Haenszel Chi-square analyses.

MMPI Validity Scales

Statistical analyses involving MMPI scores excluded cases with invalid MMPI profiles. "Invalid" profiles were those in which the pattern and overall level of scores on the validity scales were suggestive of purposeful over-exaggeration of psychological difficulties, or failure to understand items due to confusion or psychosis. Operationally, cases were defined as invalid if they met the following criteria: Variable Response Inconsistency (VRIN) Scale T-scores > 85; Infrequency (F) Scale T-scores >120; F-Back (FB) Scale T-scores >120; or Lie (L) Scale T-scores >82 (Friedman, Lewak, Nichols, & Webb, 2001). Based on this definition, 7.2% of the sample produced invalid MMPI profiles and were excluded from analyses. The proportion of invalid MMPI profiles did not differ between patients who took pre-treatment opioids and those who did not, $\chi^2(1) = 2.163$, $\underline{p} = .141$.

NO AND YES GROUP COMPARISONS

MMPI Clinical Scales

Analysis of Mean Clinical Scale Scores. A MANOVA was conducted to determine the effect of pre-treatment opioid use on MMPI clinical scales. Significant differences were found between the NO and YES groups, Wilks' $\Lambda = .96$, $\underline{F}(10, 757) = 3.25$, $\underline{p} < .001$. ANOVAs on each clinical scale were then conducted as follow-up tests to the MANOVA. Using the Bonferroni method, each ANOVA was tested at the .005 level. Results are presented in Table 38.

The YES group showed significantly higher mean scores than the NO group on several MMPI clinical scales. Patients taking pre-treatment opioids showed significantly higher mean scores on Scale 1 (Hypochondriasis), Scale 2 (Depression), Scale 3 (Hysteria), Scale 7 (Psychasthenia), and Scale 8 (Schizophrenia).

Analysis of Clinical Scale Elevations. In order to examine the clinical significance of differences in MMPI clinical scales, Chi-square analyses were conducted on the proportion of patients showing elevations (T-scores $\geq$ 65). Odds ratios are reported for opioid group comparisons in the text and tables. Odds ratios can be used as an estimate of relative risk when the event is rare. However, odds ratios must be interpreted with caution for more common events because they will overestimate the risk of events occurring (Bland & Altman, 2000). For this reason, the interpretations of odds ratios are worded in the direction of the less commonly occurring event. Results are displayed in Table 39.

The NO group was twice as likely as the YES group to have Scale 1 (Hypochondriasis) scores in the normal range (T-score $\leq$ 65), $\chi^2(1) = 12.29$, $p < .001$; OR = 2.01 (CI = 1.36, 2.99). The NO group was 1.5 times more likely than the YES group to have Scale 3 (Hysteria) scores in the normal range, $\chi^2(1) = 7.41$, $p = .006$; OR = 1.57 (CI = 1.13, 2.16). The YES group was more than 1.5 times as likely as the NO group to produce a Scale 7 (Psychasthenia) elevation, $\chi^2(1) = 10.68$, $p = .001$; OR = 1.62 (CI = 1.21, 2.16). No significant differences in the proportions of patients showing clinical elevations were found for Scale 2 (Depression), Scale 4 (Psychopathic Deviate), Scale 5 (Masculine-Feminine),

Scale 6 (Paranoia), Scale 8 (Schizophrenia), Scale 9 (Hypomania), and Scale 0 (Social Isolation).

MMPI Clinical Scales Cluster Analysis. In the present study, *k*-means cluster analysis also produced these four clusters of MMPI profiles. For the Study Sample, 32.2% of patients fell in the defended cluster, 11.3% in the distressed cluster, 29.7% in the somatoform cluster, and 26.8% in the depressed cluster. Chi-square analysis revealed significant differences in the proportions of MMPI profile clusters found between patient not taking pre-treatment opioid medications and those patients taking pre-treatment opioid medications, $\chi^2(3) = 13.26$, $p = .004$. Specifically, defended was over-represented in the NO group, while distressed was over-represented in the YES group (Table 40).

Analysis of MMPI Profiles. Of the 768 patients in the NO and YES groups who produced a valid MMPI profile, 577 (75.1%) were classified into one of the four MMPI profiles examined. Of the 577 classified patients, 41 (7.1%) produced a profile that was within normal limits (Normal), 88 (15.3%) produced a Conversion V profile, 50 (8.7%) produced a Neurotic Triad profile, and 398 (69.0%) produced a Disability profile. The proportions of each of these profiles differed significantly between the NO group and YES group, $\chi^2(3) = 8.195$, $p = .042$. In general, the NO group showed higher proportions of Normal and Conversion V profiles, while the YES group showed a higher proportion of Disability profiles. Closer examination revealed that the YES group was more than 1.5 times as likely as the NO group to produce the Disability profile, $\chi^2(1) =$

7.70, p = .006; OR = 1.66 (CI = 1.16, 2.37). Table 41 displays the proportions of each profile produced in the NO and YES groups.

WAIS-R

For the NO and YES groups combined, the mean WAIS-R Verbal and Performance Indices were 86.37 (SD = 13.18) and 86.68 (SD = 13.54), respectively. The mean WAIS-R Full Scale IQ score was 85.84 (SD = 12.96). The NO and YES groups did not differ significantly on the Verbal, Performance, or Full Scale IQs. The means for both groups fell in the Low Average range. WAIS VIQ, PIQ, and FSIQ results are presented in Table 42.

OPIOID SUBGROUPS COMPARISONS

MMPI Clinical Scales

Analysis of Mean Clinical Scale Scores. A MANOVA was also conducted to determine the effect of pre-treatment opioid use on MMPI clinical scales. Significant differences were found among the five opioid subgroups, Wilks' Λ = .89, $F(40, 2542) = 1.91$, $p = .001$. Among the five opioid subgroups, an ANOVA revealed significant differences for Scale 1 (Hypochondriasis), Scale 2 (Depression), Scale 3 (Hysteria), Scale 4 (Psychopathic Deviate), Scale 6 (Paranoia), Scale 7 (Psychasthenia), Scale 8 (Schizophrenia), and Scale 0 (Social Isolation). In general, mean scores of these scales increased with increased pre-treatment opioid use. Post-hoc analyses revealed that significant results were attributable to differences between the NO subgroup and the VERY HIGH

subgroup. The NO subgroup and the HIGH subgroup also differed significantly on Scale 1 (Hypochondriasis). The LOW and VERY HIGH subgroups differed on Scale 4 (Psychopathic Deviate), Scale 8 (Schizophrenia), and Scale 0 (Social Isolation). Also, the MEDIUM subgroup differed from the VERY HIGH subgroup on Scale 0 (Social Isolation). Means, standard deviations, and results from ANOVA are displayed in Table 43.

Analysis of Clinical Scale Elevations. Chi-square analyses were performed for the five opioid subgroups on the proportions of patients showing elevations on each clinical scale (Table 44). Results indicated significant differences in the proportions of patients producing elevations for all MMPI clinical scales, except Scale 5 (Masculine-Feminine) and Scale 9 (Hypomania). In general, the proportions of patients showing elevations increased as pre-treatment opioid dose increased. Specifically, proportions of patient demonstrating elevations on Scale 1 (Hypochondriasis) ranged from 78.8% in the NO subgroup, to 91.5% in the HIGH subgroup, $\chi^2(4) = 11.78$, $\underline{p} = .019$. Proportions of patients demonstrating elevations on Scale 2 (Depression) ranged from 55.9% in the NO subgroup to 80.1% in the VERY HIGH subgroup, $\chi^2(4) = 9.75$, $\underline{p} = .045$. Scale 3 (Hysteria) elevations ranged from 69.0% in the NO subgroup to 88.6% in the VERY HIGH subgroup, $\chi^2(4) = 11.60$, $\underline{p} = .021$. Meanwhile, Scale 4 (Psychopathic Deviate) elevations ranged from 26.4% in the LOW subgroup to 60.0% in the VERY HIGH subgroup, $\chi^2(4) = 20.11$, $\underline{p} < .001$. Proportions of patients demonstrating elevations on Scale 6 (Paranoia) ranged from 23.7% in the NO subgroup to 45.7% in the VERY HIGH subgroup, $\chi^2(4) =$

10.63, p = .031. Proportions of patients demonstrating elevations on Scale 7 (Psychasthenia) ranged from 35.8% in the NO subgroup to 59.6% in the HIGH subgroup, $\chi^2(4) = 15.79$, p = .003. For Scale 8 (Schizophrenia), approximately 40.0% patients in the NO subgroup, the LOW subgroup, and the MEDIUM subgroup produced elevations, while 57.4% and 68.6% of the HIGH subgroup and the VERY HIGH subgroup, respectively showed elevations, $\chi^2(4) = 14.60$, p = .006. Finally, for Scale 0 (Social Isolation), proportions of patients showing elevations ranged from 18.9% in the LOW subgroup to 48.6% of patients in the VERY HIGH subgroup, $\chi^2(4) = 16.22$, p = .003

MMPI Clinical Scales Cluster Analysis. Among the five opioid subgroups, Chi-square analysis revealed significant differences in the proportions of MMPI profile clusters, $\chi^2(12) = 41.33$, p < .001. Higher proportions of patients fell in the defended cluster among lower opioid subgroups, while higher proportions of patients fell in the distressed cluster among higher opioid subgroups (Table 45).

Analysis of MMPI Profiles. Among the 685 patients in the five opioid subgroups who produced a valid MMPI profile, 519 (75.8%) were classified into one of the four MMPI profiles examined. Of the 519 classified patients, 40 (7.7%) produced a profile that was within normal limits (Normal), 80 (15.4%) produced a Conversion V profile, 46 (8.9%) produced a Neurotic Triad profile, and 353 (68.0%) produced a Disability profile. The proportions of each of these profiles differed significantly among the five opioid subgroups, Mantel-Haenszel $\chi^2(1) = 4.278$, p = .039. The VERY HIGH subgroup showed the highest

proportion of Disability profiles (83.3%) and the lowest proportion among the five subgroups of Conversion V (3.3%) and Neurotic Triad (6.7%) profiles. Closer inspection showed that the VERY HIGH subgroup was significantly more likely than the NO subgroup to produce the Disability profile, $\chi^2(1) = 4.64$, $\underline{p} = .031$; OR = 2.85 (CI = 1.06, 7.66). The NO subgroup was more than six times as likely as VERY HIGH subgroup to produce a Conversion V profile, $\chi^2(1) = 4.145$, $\underline{p} = .042$, OR = 6.28 (CI = 0.84, 47.61). Table 46 displays the proportions of each profile produced in each of the opioid subgroups.

WAIS-R

The five subgroups did not differ significantly on the Verbal, Performance, or Full Scale IQs. The means for both groups fell in the Low Average range. WAIS VIQ, PIQ, and FSIQ results are presented in Table 47.

CHAPTER EIGHT

Results:

Physical Variables

Physical data analyzed for the purposes of the study included the composite index of the physical performance referred to as the "cumulative physical score" and the Quantified Pain Drawing.

Physical data were gathered from all patients prior to and upon completion of treatment. Because the post-treatment scores were only gathered from those who completed the program, the post-treatment sample is smaller than the pre-treatment sample (which is reflected in the tables). Pre- and post-treatment comparisons between the NO and YES groups, and among the five opioid subgroups, were conducted by ANOVA. Proportions of patients rating "extreme" pain intensity (scores $\geq$8 points) were analyzed by Chi-square. Pre-treatment to post-treatment change scores were also calculated and examined to further explore the clinical significance of treatment gains associated with level of pre-treatment opioid use. Linear regression analyses, employed to evaluate pre-treatment level of opioid use as a predictor of physical performance and pain outcomes, are presented at the end of the chapter. The physical data analyses presented below are summarized in Tables 48 through 59.

NO AND YES GROUP COMPARISONS

Pre-treatment Physical Variables

Pre-treatment Cumulative Physical Score. An ANOVA revealed a significant difference in mean pre-treatment cumulative physical scores between the NO and YES groups, $F(1, 1202) = 68.02$, $p < .001$. Patients taking pre-treatment opioids showed significantly lower (less desirable) pre-treatment cumulative physical scores ($M = 37.53$, $SD = 17.44$) than patients not taking pre-treatment opioids ($M = 45.88$, $SD = 17.69$). Results are displayed in Tables 48 and 49.

Pre-treatment Quantified Pain Drawing. An ANOVA also revealed a significant difference in mean pre-treatment QPD trunk scores between the NO and YES groups, $F(1, 1004) = 29.93$, $p < .001$. The YES group reported significantly higher QPD trunk scores ($M = 11.29$, $SD = 9.31$) than the NO group ($M = 8.33$, $SD = 7.83$). Likewise, the YES group reported significantly higher QPD extremity scores ($M = 15.14$, $SD = 17.86$) than the NO group ($M = 11.20$, $SD = 14.10$). The YES group also reported significantly higher pain intensity scores ($M = 6.61$, $SD = 1.69$) than the NO group [$M = 6.27$, $SD = 1.69$; $F(1, 1008) = 8.745$, $p = .003$]. The proportions of patients who rated their pain intensity in the "extreme" range did not differ significantly between the YES (35.4%) and NO (30.4%) groups, $\chi^2(1) = 2.90$, $p = .088$. Results of pre-treatment QPD are presented in Tables 48 through 50.

Post-treatment Physical Variables

Post-treatment Cumulative Physical Score. Results for post-treatment cumulative physical score are presented in Tables 51 and 52. An ANOVA showed a significant difference in mean post-treatment cumulative physical scores between the NO and YES groups, $\underline{F}(1, 890) = 5.09$, $\underline{p} = .024$. Patients taking pre-treatment opioids showed significantly lower (less desirable) post-treatment cumulative physical scores ($\underline{M} = 76.82$, $\underline{SD} = 15.59$) than patients not taking pre-treatment opioids ($\underline{M} = 79.09$, $\underline{SD} = 14.49$). However, when differences in pre-treatment cumulative physical scores were controlled, the groups did not differ significantly, $\underline{F}(1, 878) = .244$, $\underline{p} = .622$.

Post-treatment Quantified Pain Drawing. Between the NO and YES groups, an ANOVA showed a significant difference in mean post-treatment QPD trunk scores, $\underline{F}(1, 877) = 13.84$, $\underline{p} < .001$. The YES group showed significantly higher post-treatment QPD trunk scores ($\underline{M} = 9.04$, $\underline{SD} = 8.46$) than the NO group ($\underline{M} = 7.02$, $\underline{SD} = 7.68$). Similarly, the YES group showed significantly higher post-treatment QPD extremity scores ($\underline{M} = 10.72$ $\underline{SD} = 13.49$) than the NO group ($\underline{M} = 8.98$, $\underline{SD} = 11.25$; $\underline{F}(1, 876) = 4.33$, $\underline{p} = .038$). The YES group also showed significantly higher post-treatment pain intensity scores ($\underline{M} = 4.91$, $\underline{SD} = 2.14$) than the NO group ($\underline{M} = 4.38$, $\underline{SD} = 2.13$; $\underline{F}(1, 882) = 13.26$, $\underline{p} < .001$), even when controlling for pre-treatment differences in pain intensity. The proportions of patients who rated their pain intensity in the "extreme" range did not differ significantly between the YES (12.8%) and NO (9.0%) groups, $\chi^2(1) = 3.33$, $\underline{p} = .068$. Results of post-treatment QPD are presented in Tables 51 through 53.

OPIOID SUBGROUPS COMPARISONS

Pre-treatment Physical Variables

Pre-treatment Cumulative Physical Score. ANOVA revealed significant differences in mean pre-treatment cumulative physical scores among the five opioid subgroups, $F(4, 1079) = 22.02$, $p < .001$. The NO subgroup showed the most favorable mean cumulative physical score of 45.86 ($SD = 17.71$), and the LOW subgroup showed the second most favorable score of 38.31 ($SD = 16.845$). The next most favorable mean score of 36.96 ($SD = 17.08$) was demonstrated by the VERY HIGH subgroup. The HIGH and MEDIUM subgroups showed less favorable mean cumulative physical scores, with 34.28 ($SD = 15.71$) and 34.31 ($SD = 18.00$), respectively. Post-hoc analyses revealed that the significance was accounted for by differences between the NO subgroup and the other four subgroups. Significant differences were not found among the LOW, MEDIUM, HIGH, and VERY HIGH subgroups (Tables 54 and 55).

Pre-treatment Quantified Pain Drawing. An ANOVA showed significant differences in pre-treatment QPD trunk, $F(4, 900) = 7.31$, $p < .001$, QPD extremity scores, $F(4, 900) = 5.19$, $p < .001$, and pain intensity, $F(4, 903) = 3.45$, $p = .008$, among the five opioid subgroups (Tables 54 and 55). Post-hoc analyses revealed that differences in QPD trunk scores were accounted for by differences between the NO and LOW subgroups, the NO and MEDIUM subgroups, and the LOW and VERY HIGH subgroups. Differences in QPD extremity scores were accounted for by differences between the NO subgroup and the VERY HIGH

subgroup. Differences in pain intensity were accounted for by differences between the NO subgroup and all other subgroups, except the LOW subgroup. Proportions of patients reporting pain intensity in the "extreme" range did not differ significantly among the five opioid subgroups, $\chi^2(4) = 4.52$, $\underline{p} = .341$. Proportions ranged from 30.4% in the NO subgroup to 40.7% in the HIGH subgroup (Table 56).

Post-treatment Physical Variables

Post-treatment Cumulative Physical Score. An ANOVA also revealed significant differences in mean post-treatment cumulative physical scores among the five opioid subgroups, $\underline{F}(4, 795) = 2.84$, $\underline{p} = .024$. The NO subgroup produced the most favorable mean cumulative physical score of 79.07 ($\underline{SD} = 14.49$), and the LOW subgroup showed the second most favorable score of 77.25 ($\underline{SD} = 16.94$). The next most favorable mean score of 77.40 ($\underline{SD} = 12.94$) belonged to the HIGH subgroup. The VERY HIGH and MEDIUM subgroups showed less favorable mean cumulative physical scores, with 72.82 ($\underline{SD} = 12.86$) and 74.00 ($\underline{SD} = 15.35$), respectively. Post-hoc analyses indicated that the significance was accounted for by differences between the NO and MEDIUM subgroups, the NO and VERY HIGH subgroups, and the LOW and MEDIUM subgroups. Again, ANCOVA failed to find these differences when pre-treatment scores were controlled, $\underline{F}(4, 786) = 1.01$, $\underline{p} = .404$.

Examination of change scores among the five opioid subgroups revealed significant differences, $\underline{F}(4, 787) = 5.03$, $\underline{p} = .001$) which, based on post-hoc

analyses, were attributable to a difference between the NO subgroup and the LOW subgroup. The NO subgroup (M = -31.83, SD = 19.87) showed the smallest gains in physical cumulative score, while the HIGH subgroup (M = -40.00, SD = 16.41), followed by the LOW subgroup (M = -38.24, SD = 21.04), showed the greatest gains. Tables 57 and 58 present results for post-treatment cumulative physical score.

Post-treatment Quantified Pain Drawing. Among the five opioid subgroups, post-treatment QPD trunk scores did not differ significantly when pre-treatment differences in QPD trunk scores were controlled, $F(4, 731) = 0.567$, $p = .687$. An ANOVA showed no significant differences in post-treatment QPD extremity scores, $F(4, 785) = 1.569$, $p = .181$. Post-treatment pain intensity scores, however, did differ significantly among the five opioid subgroups when pre-treatment pain intensity was controlled, $F(4, 731) = 2.934$, $p = .020$. Patients in the NO subgroup reported the lowest post-treatment pain intensity scores (M = 4.39, SD = 2.13). Patients in the LOW subgroup reported the highest post-treatment pain intensity scores (M = 5.07, SD = 2.14), followed by patients in the VERY HIGH subgroup (M = 4.94, SD = 2.15). The proportion of patients reporting pain intensity in the "extreme" range at post-treatment, did not differ significantly among the five opioid subgroups, $\chi^2(4) = 4.040$, $p = .401$ (Tables 57 through 59).

LINEAR REGRESSION ANALYSES-PHYSICAL VARIABLES

Post-treatment Cumulative Physical Score

A linear regression analysis was conducted to evaluate the prediction of post-treatment cumulative physical scores from pre-treatment opioid use (average daily milligrams of morphine). Results showed that pre-treatment opioid dose significantly predicted post-treatment cumulative physical scores, $F(1, 801) = 7.85$ $p = .005$, accounting for approximately 1% of the variance.

Post-treatment Quantified Pain Drawing

Linear regression analyses conducted to evaluate the prediction of post-treatment QPD scores from pre-treatment opioid use (average daily milligrams of morphine) did not yield significant results for QPD trunk [$F(1, 806 = 0.91$, $p = .340$], extremity [$F(1, 805 = 3.30$, $p = .070$], or pain intensity [$F(1, 808) = 2.93$, $p = .088$].

CHAPTER NINE

Results:

One-year Socioeconomic and Health Variables for Program Completers

This chapter presents the results of analyses of socioeconomic and health outcome data collected at one-year post-treatment. These long-term outcome data are associated with program completion. Thus, group sizes are smaller than those presented for pre-treatment. Chi-square analyses were conducted to detect differences in socioeconomic and health outcomes as a function of pre-treatment level of opioid use. Results of logistic regression analyses are presented at the end of the chapter. One-year post-treatment outcomes are summarized in Tables 60 through 63.

One-year Socioeconomic and Health Outcomes of the NO and YES Groups

Of those patients who completed the PRIDE Functional Restoration Program, 93.7% of the NO group and 88.1% of the YES group reported returning to work at some point during the year after rehabilitation. This difference was statistically significant, with the YES group being twice as likely as the NO group to have not worked in the year following treatment completion, $\chi^2(1) = 8.05$, $\underline{p} = .005$; OR = 2.00 (CI = 1.23, 3.25). Work retention for the NO group was 85.3%, while work retention for the YES group was 68.8%. This difference was also statistically significant, with the YES group being more than 2.5 times as likely as the NO group to not be working at the time of the one-year follow-up, $\chi^2(1) =$

32.09, $\underline{p} < .001$; OR = 2.62 (CI = 1.87, 3.68). The YES group was also more than 2.5 times as likely to seek healthcare from a new provider as the NO group, $\chi^2(1) = 27.42$, $\underline{p} < .001$; OR = 2.63 (CI = 1.82, 3.81). Proportions of patients receiving SSDI or SSI also differed significantly. The YES group (5.2%) was nearly three times more likely than the NO group (1.9%) to be receiving SSDI or SSI, $\chi^2(1) = 6.31$, $\underline{p} = .012$; OR = 2.81 (CI = 1.21, 6.49). Significant differences were not found between the two groups for proportions of patients who reported working 40+ hours per week, new injury to the same body part, new surgery to the same body part, and case settlement. Tables 60 and 61 provide detailed information regarding one-year socioeconomic and health outcomes for the opioid groups.

One-year Socioeconomic and Health Outcomes of the Opioid Subgroups

Chi-square analyses of the five opioid subgroups yielded significant differences in proportions of patients who reported work return, work retention, seeking treatment from a new provider, and receiving SSDI or SSI. Patients reporting work return ranged from 93.7% in the NO subgroup to 75.9% in the VERY HIGH subgroup, $\chi^2(4) = 13.82$, $\underline{p} = .008$. Patients reporting work retention ranged from 85.2% in the NO subgroup to 55.2% in the VERY HIGH subgroup, $\chi^2(4) = 37.75$, $\underline{p} < .001$. The proportion of patients seeking treatment from a new provider was 14.0% in the NO subgroup, and ranged from 28.2 to 29.6 in the LOW, HIGH, and VERY HIGH subgroups, $\chi^2(4) = 26.83$, $\underline{p} < .001$. The MEDIUM subgroup showed the highest rate at nearly 37%, making them more than three and a half times as likely as the NO subgroup to have sought treatment

from a new provider, OR = 3.58 (CI = 1.87, 6.85). The proportions of patients reporting SSDI/SSI ranged from 1.9% in the NO subgroup to 18.5% in the VERY HIGH subgroup, $\chi^2(4) = 22.06$, $\underline{p} < .001$. Thus, the VERY HIGH subgroup was 11.6 times as likely as the NO subgroup to be receiving SSDI or SSI at the one-year follow-up, OR = 11.62 (CI = 3.51, 38.46).

Significant differences among the five opioid subgroups were not found for proportions of patients who reported working 40+ hours per week, new surgery to the same body part, new injury to the same body part, and case settlement at one-year post-treatment. Tables 62 and 63 provide detailed information regarding one-year socioeconomic and health outcomes for the five opioid subgroups.

Logistic Regression Analyses

Binary logistic regression analyses were conducted to evaluate the prediction of one-year socioeconomic outcomes from pre-treatment level of opioid use. Results demonstrated that pre-treatment level of opioid use significantly predicted work return and work retention. Opioid level accounted for 2.5% of the variance in work return (Wald[1] = 9.18, $\underline{p} = .002$, $\underline{R}^2 = .025$). The overall classification rate for the binary logistic regression model was 91.4%, with 100% sensitivity and 0% specificity. Opioid level accounted for 5.7% of the variance in work retention (Wald[1] = 28.90, $\underline{p} < .001$, $\underline{R}^2 = .057$). The overall classification rate for the model was 77.7%, with 97.2% sensitivity and 8.0% specificity. Furthermore, opioid level significantly predicted new surgery to the

same body part at one-year follow-up (Wald[1] = 4.48, $\underline{p}$ = .034, $\underline{R}^2$ = .024). The overall classification rate was 96.6%, with 100% and 0%, sensitivity and specificity, respectively. Finally, opioid level significantly predicted whether patients had sought treatment from a new provider during the year following program completion (Wald[1] = 16.25, $\underline{p}$ < .001, $\underline{R}^2$ = .037. The overall classification rate for the model was 79.5%, with 100% sensitivity and 0% specificity.

CHAPTER TEN

Results:

One-year Socioeconomic and Health Variables for Program Non-completers

This chapter presents the results of analyses of socioeconomic and health outcome data collected during the one-year follow-up from patients who did not complete the functional restoration program. Group sizes are smaller than those presented for pre-treatment because they represent non-completers only. Chi-square analyses were conducted to detect differences in socioeconomic and health outcomes as a function of pre-treatment level of opioid use. One-year post-treatment outcomes for non-completers are summarized in Tables 64 through 67.

One-year Socioeconomic and Health Outcomes of the NO and YES Groups

Of those patients who did not complete the PRIDE Functional Restoration Program, 71.2% of the NO group and 53.5% of the YES group reported returning to work at some point during the year after rehabilitation. This difference was statistically significant, with the YES group being more than twice as likely as the NO group to have not worked in the year following treatment completion, $\chi^2(1) = 5.26$, $\underline{p} = .022$; OR = 2.16 (CI = 1.11, 4.17). Work retention for the NO group was 58.9%, while work retention for the YES group was 44.2%. This difference, however, was not statistically significant, $\chi^2(1) = 3.42$, $\underline{p} = .064$. The YES group (49.5%) was almost twice as likely to seek healthcare from a new provider as the NO group (34.6%), $\chi^2(1) = 3.90$, $\underline{p} = .048$; OR = 1.85 (CI = 1.00, 3.42).

Proportions of patients receiving SSDI or SSI also differed significantly. The YES group (14.5%) was nearly five times more likely than the NO group (3.5%) to be receiving SSDI or SSI, $\chi^2(1) = 4.29$, $\underline{p} = .038$; OR = 4.67 (CI = 0.96, 22.63). Significant differences were not found between the two groups for proportions of patients who reported working 40+ hours per week, new injury to the same body part, new surgery to the same body part, and case settlement. Tables 64 and 65 provide detailed information regarding one-year socioeconomic and health outcomes for non-completers in the opioid groups.

One-year Socioeconomic and Health Outcomes of the Opioid Subgroups

Chi-square analyses of the five opioid subgroups failed to yield significant differences in proportions of patients who reported work return, work retention, 40+ hour work week, healthcare utilization, new injury to the same body part, new surgery to the same body part, case settlement and SSDI/SSI. Tables 66 and 67 provide detailed information regarding one-year socioeconomic and health outcomes for non-completers in the five opioid subgroups.

CHAPTER ELEVEN

Results:

Extreme Group Comparisons

To further examine the clinical relevance of differences among the five opioid subgroups, direct comparisons by odds ratios were conducted. Odds ratios can be used as an estimate of relative risk when the event is rare. However, again, it should be noted that odds ratios must be interpreted with caution for more common events because they will overestimate the risk of the event occurring (Bland & Altman, 2000). For this reason, the interpretations of odds ratios are worded in the direction of the less commonly occurring event. First, odds ratios were conducted between the NO subgroup and the HIGH subgroup, and the NO subgroup and the VERY HIGH subgroup. Then, odds ratios were conducted between the NO group and the combination of the HIGH and VERY HIGH subgroups (HIGH/VERY HIGH). Significant findings are presented below.

Rates of program completion for the NO subgroup and the VERY HIGH subgroup did not differ significantly, $\chi^2(1) = 3.55$, $\underline{p} = .060$. However, the risk of program non-completion for the HIGH subgroup was twice that of the NO subgroup, OR = 2.06 (CI = 1.21, 3.51). The HIGH/VERY HIGH subgroup was almost twice as likely as the NO subgroup to terminate treatment prematurely, OR = 1.96 (CI = 1.27, 3.02).

Comparisons of pre-treatment socioeconomic and health variables showed that the VERY HIGH subgroup was more than six times as likely as the NO

subgroup to be receiving SSDI or SSI. The HIGH/VERY HIGH subgroup was one and a half times more likely than the NO subgroup to report a prior work-related injury, OR = 1.56 (CI = 1.01, 2.40). The HIGH subgroup was nearly twice as likely to report a pre-treatment surgery as the NO group, OR = 1.82 (CI = 1.09, 3.06). Similarly, the HIGH/VERY HIGH subgroup was more than twice as likely as the NO subgroup to report a pre-treatment surgery, OR = 2.23 (CI = 1.47, 3.37). The VERY HIGH subgroup was more than three times as likely to report a pre-treatment surgery as the NO subgroup, OR = 3.01 (CI = 1.60, 5.68).

Extreme group comparisons of MMPI clinical scale score elevations yielded numerous significant findings, with odds ratios as high as 4.03. Results of these comparisons are presented in Table 68.

Closer examination of one-year health outcomes revealed that the VERY HIGH subgroup was more than two and a half times as likely as the NO subgroup to have sought treatment from a new provider, OR = 2.60 (CI = 1.08, 6.23). Similarly, the HIGH subgroup was nearly two and a half times as likely as the NO subgroup to have sought treatment from a new provider, OR = 2.42 (CI = 1.14, 5.15). The HIGH subgroup was nearly four times as likely as the NO subgroup to have reported a new surgery to the same body part at one-year follow-up, OR = 3.92 (CI = 1.00, 15.42); as was the HIGH/VERY HIGH subgroup, OR = 3.85 (CI = 1.22, 12.16).

Odds ratios were also conducted to examine differences between the extremes of pre-treatment opioid subgroups on dichotomous one-year socioeconomic outcomes. The VERY HIGH subgroup was nearly five times

more likely than the NO subgroup to have not returned to work during the year following treatment completion, OR = 4.74 (CI = 1.87, 12.05). The HIGH/VERY HIGH subgroup was still more than two and a half times more likely than the NO subgroup to have not returned to work, OR = 2.68 (CI = 1.27, 5.65). Results were similar for work retention. The HIGH subgroup was 2.6 times more likely [OR = 2.60 (1.28, 5.24)], and the VERY HIGH subgroup was 4.7 times more likely than the NO subgroup to not be working at the one-year follow-up [OR = 4.70 (CI = 2.16, 10.20)]. Furthermore, the VERY HIGH subgroup was 11.6 times as likely as the NO subgroup to be receiving SSDI or SSI.

CHAPTER TWELVE

Results:

Opioid and Non-opioid Analgesic Medications

Patients identified as taking pre-treatment opioids were classified into opioid subgroups based on average daily equivalent of milligrams of oral morphine: LOW (>0-30), MEDIUM (>30-60), HIGH (>60-120), VERY HIGH (>120). Tables 69 and 70 present descriptive statistics for the LOW subgroup, the MEDIUM subgroup, the HIGH subgroup, and the VERY HIGH subgroup, as well as, for the four opioid subgroups combined. Information on pre-treatment non-opioid analgesics and muscle relaxants are presented in Tables 71 and 72, respectively.

CHAPTER THIRTEEN

Discussion

Based on self-report, subjects were classified into groups (NO and YES), as well as subgroups (NO, LOW, MEDIUM, HIGH, and VERY HIGH), to determine if pre-treatment level of opioid use discriminates subjects' response to treatment, as measured by program completion, physical functioning, depressive symptoms, pain report, disability, health-related quality of life, socioeconomic, and health outcomes. This chapter presents the findings of this examination, limitations of the study, and makes suggestions for future research.

Demographics

Demographic analyses of the pre-treatment opioid groups and subgroups revealed significant differences in racial representation. Caucasians were over-represented among patients reporting opioid use, and Hispanics were under-represented. Possible explanations for this finding are beyond the scope of this investigation, but might include racial differences in attitudes regarding medication use and access to pharmaceutical interventions prior to entering functional restoration.

Program Completion

As predicted in Hypothesis 1, pre-treatment level of opioid use was associated with rate of functional restoration program completion, such that

patients reporting higher levels of pre-treatment opioid use were at greater risk of program non-completion. The YES group was more than 1.5 times as likely as the NO group to discontinue functional restoration prematurely. Interestingly, the VERY HIGH subgroup was not the least likely to complete functional restoration. Rather, the HIGH and MEDIUM subgroups showed the lowest completion rates. Extreme group comparisons revealed that the HIGH subgroup was twice as likely as the NO subgroup to not complete functional restoration. Patients who participate in the PRIDE Functional Restoration Program are weaned from opioid medications at the onset of treatment. A possible interpretation of findings related to program completion rates among the opioid levels is that patients taking low doses of opioid medications are easily weaned and thus, continue with functional restoration. Patients taking MEDIUM and HIGH doses might be more reluctant to or have more difficulty tapering opioid use, thus leading to increased program drop-out. Results for the VERY HIGH subgroup might be explained in part by small sample size. However, the willingness to discontinue very high doses might be indicative of high motivation for treatment among these individuals.

Rome and colleagues (2004) failed to find a difference in completion rates between patients taking and not taking pre-treatment opioids. However, among patients taking pre-treatment opioids, those who did not complete the program were taking significantly higher doses of morphine equivalent analgesics than patients who did complete the program. MacLaren and colleagues (2006) examined pre-treatment opioid use in program completers only, and did not report proportions of non-completers who were taking opioids. Thus, the impact of

opioid use on program completion could not be evaluated. Differences in program completion rates may be attributable to differences in length of disability, pre-treatment surgery rates, pre-treatment pain intensity, and comorbid psychiatric and substance use disorders. Likewise, differences in program completion rates may be, in part, attributable to pre-treatment differences in levels of depression and self-reported disability.

Pre-treatment Socioeconomic and Health Variables

Findings related to compensation factors (Hypothesis 2) were mixed. Pre-treatment opioid use was not associated with higher rates of pre-treatment case settlement, nor attorney retention. However, the YES group was four times more likely than the NO group to be receiving SSDI or SSI. Contrary to Hypothesis 2, workers' compensation benefits were significantly higher for the NO group compared to the YES group. Highest levels of compensation were found among the NO, followed by the LOW and VERY HIGH subgroups; while the MEDIUM and HIGH subgroups showed the lowest levels of compensation. While these differences are statistically significant, they might have little meaning practically. Workers' compensation benefits varied by only $45 per week across the entire sample and standard deviations were greater than $130 for all subgroups except the VERY HIGH subgroup, which had a smaller sample size.

Hypothesis 3 regarding pre-treatment health status was supported by the findings. The YES group was nearly 1.5 times more likely to report a prior work-related injury, and over 1.5 times as likely to report a pre-treatment surgery as the

NO group. Further, the VERY HIGH subgroup was more than three times as likely to report a pre-treatment surgery. Possibly, individuals taking higher levels of opioids are suffering from more severe injuries that are less amenable to treatment.

Pre-treatment Depression

Hypothesis 4 proposed that pre-treatment level of opioid use would be associated with pre-treatment level of depressive symptoms, with higher levels of depressive symptoms found in patients reporting higher levels of pre-treatment opioid use. Findings supported this hypothesis, with the YES group showing significantly higher scores than the NO group on both self-reported and clinician-reported measures of pre-treatment depressive symptoms. Closer examination of the scores based on level of opioid use showed interesting differences between self-reported and clinician-rated measures. On both the BDI (self-report) and HAM-D (clinician-rated), average scores for the NO, LOW, MEDIUM, and HIGH subgroups fell in the "mild to moderate" range. On the BDI, the VERY HIGH subgroup reported the highest levels of depressive symptoms, with the average scores in the "moderate to severe" range (Beck, Steer, & Garbin, 1988). However, HAM-D ratings of the VERY HIGH subgroup fell in the "mild to moderate" range (Hedlung & Vieweg, 1979). Thus, patients taking the highest levels of opioids rated their depressive symptoms as being more severe than their treating clinicians. Possibly, individuals taking the highest opioid levels have a tendency to over-report or exaggerate their distress in order to get the attention of

others or for medication seeking. However, clinicians might have biases toward these patients, believing that they tend to exaggerate, and therefore, under-rate depressive symptoms.

Other researchers have failed to find differences in pre-treatment depression based on pre-treatment opioid use. Rome, et al. (2004) found that over 70% of patients met the 16 point cut-off score for depression as measured by the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), however, mean CES-D scores did not differ between the opioid and non-opioid groups. One possible explanation for this discordant finding is that the CES-D is intended to be a screening tool for depression, not a measure of symptom severity, as are the BDI and HAM-D. Thus, average scores on the CES-D are less meaningful than the proportion of patients who met the cut-off score. MacLaren, et al. (2006) used the BDI to measure pre-treatment depressive symptoms and also failed to find differences based on pre-treatment opioid use. In addition to the study's small sample size (n=127), both pre-treatment and post-treatment analyses included completers only. The authors failed to report whether or not pre-treatment depression differed among non-completers based on opioid use. Furthermore, they did not report whether or not completers and non-completers differed in the proportion of patients taking opioids. While completers and non-completers did not differ in pre-treatment depressive symptoms, if opioid users had higher rates of program non-completion than non-opioid users, as demonstrated in the present study, this could explain why differences in pre-treatment depression were not found.

Pre-treatment Disability and Health-related Quality of Life

As predicted in Hypothesis 5, pre-treatment opioid use was linearly associated with levels of self-reported disability, as measured by MVAS. The NO subgroup reported significantly lower levels of disability than all other opioid subgroups individually, and combined. However, all opioid subgroups, including the NO subgroup, reported mean pre-treatment MVAS scores in the "severely disabling" range (Anagnostis, Mayer, Gatchel, & Proctor, 2003). Similarly, the NO subgroup reported significantly lower levels of pain-related disability compared to all other subgroups, as measured by the OSW. Although, the proportions of patients falling in higher disability ranges increased as opioid dose increased.

Hypothesis 5 also predicted that patients reporting higher levels of pre-treatment opioid use would report lower health-related quality of life at pre-treatment than patients taking lower levels of opioids, as measured by SF-36. This hypothesis was partially supported. The YES group averaged significantly lower (less desirable) scores on quality of life related to both mental and physical health, relative to the NO group. However, these differences were largely attributable to differences between the NO and LOW subgroups, and the NO and VERY HIGH subgroups for mental health; and differences between the NO and HIGH subgroups for physical health. Although statistically significant, these differences have less meaning clinically. For mental health, average scores ranged from 34.91 to 40.12, with standard deviations ranging from 8.50 to 10.52.

For physical health, average scores ranged from 28.62 to 30.83, although standard deviations were tighter, ranging from 5.19 to 6.72.

MMPI Profiles

Hypothesis 6 proposed that increasing levels of pre-treatment opioid use would be associated with less desirable MMPI profiles. Several approaches were utilized to test this hypothesis, including an analysis of mean clinical scale scores, an analysis of clinical scale elevations, a *k*-means cluster analysis, and finally, an examination of four profile types commonly discussed in the psychiatric literature. Findings from each analysis supported the hypothesis. The YES group displayed significantly higher mean scores for Scale 1 (Hypochondriasis), Scale 2 (Depression), Scale 3 (Hysteria), Scale 7 (Psychasthenia), and Scale 8 (Schizophrenia) than the NO group. In terms of clinical significance, however, these differences are less meaningful. Both the YES and NO groups displayed mean scores in a clinically significant range for Scale 1 (Hypochondriasis), Scale 2 (Depression), and Scale 3 (Hysteria). Mean scores on Scale 7 (Psychasthenia) for both groups were not clinically significant. For Scale 8 (Schizophrenia), mean scores for the NO group were not clinically significant, while mean scores for the YES group were at the threshold for clinical significance.

Further examination of the opioid subgroups revealed that, in general, mean scores on Scale 1 (Hypochondriasis), Scale 2 (Depression), Scale 3 (Hysteria), Scale 4 (Psychopathic Deviate), Scale 6 (Paranoia), Scale 7 (Psychasthenia), Scale 8 (Schizophrenia), and Scale 0 (Social Isolation) differed

significantly and increased with pre-treatment level of opioid use. As implied above, mean scores for all opioid subgroups on Scale 1 (Hypochondriasis), Scale 2 (Depression), and Scale 3 (Hysteria) fell in the clinically significant range. On Scale 4 (Psychopathic Deviate) and Scale 6 (Paranoia), clinically significant mean scores were obtained only by the VERY HIGH subgroup. On Scale 7 (Psychasthenia) and Scale 8 (Schizophrenia), clinically significant mean scores were obtained only by the HIGH and VERY HIGH subgroups.

As a corollary to Hypothesis 6, increased pre-treatment level of opioid use would be associated with greater proportions of patients displaying clinical elevations on MMPI clinical scales. This was supported by the data for Scale 1 (Hypochondriasis), Scale 2 (Depression), Scale 3 (Hysteria), Scale 4 (Psychopathic Deviate), Scale 6 (Paranoia), Scale 7 (Psychasthenia), Scale 8 (Schizophrenia), and Scale 0 (Social Isolation). Not surprisingly, a high proportion of all patients showed clinical elevations on Scale 1 (Hypochondriasis), indicating a denial of good physical health and a preoccupation with bodily functioning. However, the NO group was twice as likely as the YES group to yield Scale 1 (Hypochondriasis) scores in the normal range. Furthermore, the NO subgroup was nearly three times as likely as the HIGH and HIGH/VERY HIGH subgroups to produce Scale 1 (Hypochondriasis) scores in the normal range. Proportions of patients showing clinical elevations on Scale 1 (Hypochondriasis) increased linearly with pre-treatment opioid level from nearly 79% in the NO subgroup to over 91% in the VERY HIGH subgroup.

Clinical elevations on Scale 2 (Depression) signify depressive symptoms and pessimism. Proportions of patients showing clinical elevations on Scale 2 (Depression) ranged from approximately 56% of the NO subgroup to over 80% of the VERY HIGH subgroup. Thus, the NO subgroup was more than three times as likely as the VERY HIGH subgroup to produce scores in the normal range. A high proportion of all patients also demonstrated clinical elevations on Scale 3 (Hysteria), indicating a potential to manifest emotional distress as physical symptomatology. Proportions of patients displaying elevations increased as pre-treatment opioid level increased from nearly 69% of the NO subgroup to over 88% of the VERY HIGH subgroup. The NO group was one and a half times as likely as the YES group to have a Scale 3 (Hysteria) score in the normal range. The NO subgroup was twice as likely as the HIGH subgroup, and more than three times as likely as the VERY HIGH subgroup to produce Scale 3 (Hysteria) scores in the normal range.

Individuals with elevations on Scale 4 (Psychopathic Deviate) tend to be asocial and/or antisocial, hostile, aggressive, rebellious, and immature. They may be impulsive, untrustworthy, and are often manipulative. They have the potential for acting out, desire immediate gratification, and fail to learn from experience. As a result, psychological treatment prognosis is poor for these individuals. (Friedman, Lewak, Nichols, and Webb, 2001). Proportions of patients showing clinical elevations on Scale 4 (Psychopathic Deviate) ranged from approximately 27% of the NO subgroup to 60% of the VERY HIGH subgroup. Thus, the VERY HIGH subgroup was more than four times as likely as the NO subgroup to show

an elevation. Hence, the disproportionate number of patients displaying elevations among higher opioid subgroups may indicate high levels of anger, perhaps regarding injury or subsequent treatment. Higher proportions of Scale 4 elevations may also suggest an increase potential for substance (opioid) abuse, and the need for close observation for aberrant behaviors and assessment of opioid misuse.

Individuals with Scale 6 (Paranoia) elevations may appear hostile, suspicious, guarded, or aloof. They tend to be overly sensitive, lack guilt, and externalize blame (Friedman, Lewak, Nichols, and Webb, 2001). On Scale 6, proportions of patients displaying clinical elevations ranged from nearly 24% of the NO subgroup to nearly 46% of the VERY HIGH subgroup, making the VERY HIGH subgroup more than two and a half times as likely as the NO subgroup to show an elevation.

Scale 7 (Psychasthenia) elevations are indicative of anxiety, discomfort, dissatisfaction, indecisiveness, emotional turmoil, poor concentration, and rumination. Individuals with Scale 7 (Psychasthenia) elevations tend to be perfectionistic, and utilize rationalization and intellectualization, which slows treatment (Friedman, Lewak, Nichols, and Webb, 2001). In the sample, proportions of patients showing clinical elevations on Scale 7 (Psychasthenia) ranged from 35.8% of the NO subgroup to 59.6% of the HIGH subgroup. The YES group was more than one and a half times as likely to show an elevation as the NO group, while the HIGH subgroup was more than two and a half times as likely.

Scale 8 (Schizophrenia) elevations suggest a sense of disconnection or alienation from others, withdrawal, and thought and communication difficulties. This elevation is associated with poor treatment prognosis (Friedman, Lewak, Nichols, and Webb, 2001). For Scale 8 (Schizophrenia), approximately 40% of the NO, LOW, and MEDIUM subgroups showed clinical elevations. However, 57% of the HIGH subgroup and nearly 69% of the VERY HIGH subgroup displayed clinical elevations. Thus, the HIGH subgroup was nearly twice as likely as the NO subgroup, and the VERY HIGH subgroup was more than three times as likely as the NO subgroup to have a Scale 8 (Schizophrenia) elevation. Increased proportions of elevations among higher opioid use subgroups might be suggestive of side effects associated with opioid use, such as confusion or cognitive impairment.

Finally, approximately 20% of the NO, LOW, and MEDIUM subgroups had clinical elevations on Scale 0 (Social Isolation), indicating introversion, avoidance, and lack of social support. A slightly higher proportion (25.5%) of the HIGH subgroup, showed an elevation. However, over 48% of the VERY HIGH subgroup showed Scale 0 (Social Isolation) elevations, making the VERY HIGH subgroup more than three times as likely as the NO subgroup to show an elevation. While this finding might be an artifact of small sample size, the VERY HIGH subgroup might be associated with higher levels of opioid abuse or dependence, which interferes with social relationships. Impaired social functioning might, in turn, impact one's ability to successfully return to and retain work.

The relationship between pre-treatment opioid use and MMPI profiles was explored first through the replication and analysis of MMPI profile clusters established by Jordan (1996). Both pre-treatment opioid groups showed significant differences in the proportion of patients with MMPI profiles classified as defended, distressed, somatoform, and depressed. Proportions of patients classified as somatoform and depressed were similar across levels of opioid use. As expected, lower opioid levels were associated with greater proportions of patients classified as defended, while higher opioid levels were associated with greater proportions of patients classified as distressed. This might suggest that individuals taking no or lower levels of opioids tend to defend against distress and fight-back psychologically against pain, which could be associated with a reluctance to take opioid medications altogether. Meanwhile, individuals taking higher levels of opioids express significant distress and dissatisfaction with self and others, as well as, display limited insight into their own difficulties. Individuals in the distressed cluster are desperately attempting to elicit attention from others. They are easily frustrated and tend to act out their emotional discomfort. MMPI profiles that show multiple elevations, such as those in the distressed cluster, are often indicative of a personality disorder (Friedman, Lewak, Nichols, and Webb, 2001). Several researchers have suggested that severe personality disorders are associated with increased risk for addiction in long-term opioid therapy for chronic pain (Nedeljkovic, Wasan & Jamison, 2002; Portenoy & Foley, 1986; Strain, 2002). However, further research is needed to clarify the

relationships between this profile type, opioid use, opioid addiction, and personality disorders.

Finally, the relationship between pre-treatment opioid use and MMPI profiles was explored through the examination of three commonly studied MMPI profiles and a fourth profile, consisting of four or more clinical scale elevations, newly coined the Disability profile (Gatchel, Mayer, & Eddington, 2006). In prior studies, researchers found that approximately 80% of patients with chronic disabling work-related spinal disorders could be classified into one of these four MMPI profiles, and patients who produced the Disability profile displayed the most severe psychopathology. Patients with this profile were 14 times more likely to have an Axis I disorder, 12 times more likely to have a major depressive disorder, and 15 times more likely to have an anxiety disorder than patients with normal profiles. Furthermore, patients who produced the Disability profile were 5 time more likely to be diagnosed with an Axis II personality disorder (Kidner, Mayer, & Gatchel, 2004 ; Gatchel, Mayer, & Eddington, 2006).

In the present study, 75% of the patients who produced valid MMPI profiles could be classified into one of the four profiles. Of those patients who could be classified, approximately 7% showed a Normal profile, 15% showed a Conversion V, 9% showed a Neurotic Triad, and 69% showed the Disability profile. These proportions are very similar to those found in previous studies (Kidner, Mayer, & Gatchel, 2004 ; Gatchel, Mayer, & Eddington, 2006). While the Disability profile accounted for the majority of patients in all opioid subgroups, the proportions did increase with pre-treatment opioid dose, as

expected, indicating a relationship between degree of psychopathology and level of pre-treatment opioid use. Patients who did not take pre-treatment opioids showed the highest proportions of Conversion V and Normal profiles, which indicate a lesser degree or absence of psychopathology, respectively. Patients who took pre-treatment opioids were more than one and a half times as likely as patients who did not take pre-treatment opioids to produce the Disability profile, while patients taking very high doses of pre-treatment opioids were nearly three times as likely to produce this profile as patients who took no pre-treatment opioids. Again, the results of the present study further support the hypothesis that increasing levels of pre-treatment opioid use is associated with less desirable MMPI profiles, and thus, greater levels of pre-treatment psychopathology.

Pre-treatment Physical Functioning and Pain

Hypothesis 7 stated that higher pre-treatment levels of opioid use would be associated with poor pre-treatment physical functioning and higher pain ratings. As predicted, patients who reported pretreatment opioid use demonstrated significantly lower (less desirable) pre-treatment cumulative physical scores than patients who did not report pre-treatment opioid use.

With regard to pain, Hypothesis 7 was only partially supported. Pre-treatment opioid use was associated with higher QPD trunk, QPD extremity, and pain intensity scores. The YES group reported significantly higher trunk, extremity, and pain intensity scores than the NO group. Further analyses showed that trunk scores differed between the NO and LOW subgroups, the NO and

MEDIUM subgroups, and the LOW and VERY HIGH subgroups, but did not differ significantly between the NO and HIGH subgroups. Differences in extremity scores between the NO and YES groups were accounted for by differences between the NO and VERY HIGH subgroups. Differences in pain intensity were accounted for by differences between the NO subgroup and all other levels, except the LOW subgroup. While statistically significant, these findings must be interpreted with caution. For trunk and extremity scores, standard deviations were quite large indicating considerable overlap among the levels. For pain intensity, the results were statistically significant, but the average scores across all opioid levels ranged from 6.26 for the NO subgroup to 7.0 for the VERY HIGH subgroup, thus the differences have no relevance clinically. Furthermore, the proportions of patients scoring in the "extreme" pain intensity range did not differ based on pre-treatment opioid use (McGeary, Mayer, Gatchel, 2006).

Post-treatment Depression

Findings contradicted Hypothesis 8 that higher levels of pre-treatment opioid use would be associated with lesser improvements in depressive symptoms. Relative to their pre-treatment scores, both the YES and NO groups showed similar improvements in self-reported and clinician-rated depressive symptoms. Furthermore, the HIGH and VERY HIGH subgroups showed relatively greater improvements in self-reported depressive symptoms than the NO, LOW, and MEDIUM subgroups. Average BDI scores for all groups, except

the VERY HIGH subgroup, fell in the "absence of depression" range. Average BDI score for the VERY HIGH subgroup fell at the low end of the "mild to moderate" range (Beck, Steer, & Garbin, 1988). Average HAM-D scores for all opioid levels fell in the "none to minimal" range (Hedlung & Vieweg, 1979). Rome and colleagues (2004) also found that CNP patients taking pre-treatment opioids reported similar levels of depressive symptoms compared with patients who were not taking opioids daily, following a 3-week rehabilitation program. This is a significant finding clinically as it indicates that patients taking pre-treatment opioids show similar benefits, in terms of depressive symptoms, from participation in functional restoration as patients reporting no pre-treatment opioid use.

Post-treatment Disability and Health-related Quality of Life Outcomes

Hypothesis 9 stated that pre-treatment level of opioid use would predict improvements in disability and health-related quality of life, such that higher levels of opioid use would be associated with lesser improvements upon program completion. Results for this hypothesis were mixed. For improvements in disability, Hypothesis 9 was partially supported. Pre-treatment level of opioid use was identified as a significant predictor of post-treatment disability as measured by MVAS, however, opioid dose only accounted for 1% of the total variance in scores. The YES group reported significantly higher levels of disability than the NO group, even when pre-treatment differences were controlled. Surprisingly, the highest levels of post-treatment disability were reported by the LOW

subgroup rather than by patients who took higher doses. These differences, however, may be less significant clinically. The average disability score for the LOW subgroup fell at the lower cut-off of the "severely disabling" range; while, average disability scores for all other subgroups fell at the upper end of the "moderately disabling" range (Anagnostis, Mayer, Gatchel, & Proctor, 2003). Furthermore, the opioid levels did not differ significantly in average pre-treatment to post-treatment change scores, indicating that patients reporting pre-treatment opioid use showed similar gains in self-reported disability ratings as patients reporting no opioid use. Results for post-treatment pain-related disability, as measured by OSW, were more favorable. Mean post-treatment scores did not differ significantly based on opioid subgroup, nor did the proportions of patients reporting various levels of disability.

Hypothesis 9 further stated that higher levels of pre-treatment opioid use would be associated with lesser improvements in health-related quality of life upon program completion, but this was not supported by the data. Linear regression analyses found no significant relationship between pre-treatment opioid dose and post-treatment health-related quality of life, as measured by SF-36MHS and SF-36PHS. The YES and NO groups did not show significant differences in mean post-treatment SF-36MHS scores. The YES group did show mean post-treatment SF-36PHS scores that were statistically higher than the NO group. However, the difference in mean scores was less than two points, and therefore, not considered clinically significant. Among the five opioid levels, patients who completed the functional restoration program did not show

significant differences in post-treatment SF-36MHS or SF-36PHS scores. Similarly, pre-treatment to post-treatment improvements in quality of life ratings were not associated with pre-treatment level of opioid use. Thus, differences in health-related quality of life that were found at pre-treatment did not persist after completion of functional restoration, indicating positive treatment efficacy for all opioid levels.

Post-treatment Physical Functioning and Pain Outcomes

Hypothesis 10 stated that pre-treatment level of opioid use would predict response to treatment such that patients taking higher dosages would demonstrate lesser improvements in physical functioning at program completion. This hypothesis was not fully supported. Although the YES group, and more specifically the MEDIUM and VERY HIGH subgroups, showed lower (less desirable) cumulative physical scores than the NO group, the subgroups were not significantly different when pre-treatment differences in physical functioning were controlled. Furthermore, the YES group actually showed significantly greater gains in pre-treatment to post-treatment physical functioning scores than the NO group demonstrated. The greatest average gains were found among the LOW and HIGH subgroups. This is a significant finding clinically as it indicates that relative to patients taking none, patients taking pre-treatment opioids show similar physical benefits from participation in functional restoration.

Hypothesis 10 further stated that higher pre-treatment opioid use would be associated with poorer pain outcomes at post-treatment. As with physical

functioning, this hypothesis was only partially supported. The YES group reported significantly higher post-treatment trunk and extremity pain scores than the NO group. The YES group also reported higher pain intensity scores than the NO group, even when pre-treatment differences were controlled. As expected, the NO subgroup had the lowest average pain intensity scores. However, the LOW subgroup and the VERY HIGH subgroup showed the highest and second highest pain intensity scores, respectively.

As with pre-treatment pain, differences in post-treatment pain scores must be interpreted with caution. Although the differences are statistically significant, standard deviations were large and the average scores for trunk and extremity pain across the five subgroups differed by fewer than three points. For pain intensity, the range of scores was less than one point. As such, these differences have little utility or meaning clinically. Furthermore, when pre- to post-treatment change scores were examined, treatment gains were not associated with level of opioid use. Thus, patients experienced similar palliative benefits from functional restoration regardless of their pre-treatment opioid status.

Findings for post-treatment pain were similar in a study of CNP patients participating in an outpatient multidisciplinary rehabilitation program. Opioid users reported greater post-treatment pain severity than non-opioid users, but reported similar reductions in pain at program completion. Again, indicating that patients achieve similar improvements in pain regardless of pre-treatment opioid status (Rome et al., 2004).

One-year Socioeconomic and Health Outcomes

Hypothesis 11 predicted differences in compensation and secondary gain variables, such that program completers reporting higher levels of pre-treatment opioid use will have systematically lower rates of case settlement and higher workers' compensation benefits at the one-year follow-up. Hypothesis 11 was largely unsupported by the findings. Differences were not found for case settlement, attorney retention, or workers compensation benefits. However, the YES group (5.2%) was three times more likely than the NO group (1.9%) to be receiving SSDI or SSI at the one-year follow-up, although rates were low for both groups. One explanation for these findings is that patients taking pre-treatment opioids who discontinue opioid use and complete functional restoration might be more motivated for treatment and less driven by secondary gain than patients taking pre-treatment opioid who do not complete functional restoration.

Hypothesis 12 predicted an association between higher levels of pre-treatment opioid use and higher rates of healthcare use, including post-treatment surgery, post-treatment injury, and healthcare utilization at one-year follow-up. Results of the study only partially supported this finding. No association was found between pre-treatment level of opioid use and rates of new injury to the same body part among program completers. Overall, the YES group was not more likely than the NO group to report a post-treatment surgery to the same body part. However, the HIGH and VERY HIGH subgroups were nearly four times as likely as the NO subgroup to report a post-treatment surgery to the same body part

at one-year follow-up. Binary logistic regression analysis indicated that pre-treatment opioid level accounted for 2.4% of the variance.

Investigation of healthcare utilization yielded results that supported Hypothesis 12. Fourteen percent of the NO group had sought treatment from a new provider during the year after completing functional restoration. Compared to the NO group, the YES group was more the 2.5 times as likely to have sought healthcare from a new provider, with nearly 30% having sought treatment across all dosage levels. The MEDIUM subgroup showed the highest rate at nearly 37%, making them nearly four times as likely as the NO subgroup to have sought treatment from a new provider. Binary logistic regression analysis indicated that pre-treatment opioid level accounted for 3.7% of the variance. In a previous study, patients who had sought treatment from a new provider in the year following completion of functional restoration were more than 1.5 times as likely to carry a current substance disorder diagnosis (17.4%) as patients who had not sought treatment (11.2%; Dersh, 2000). Thus, a possible explanation for the present study's finding is that patients who discontinued opioid medications at admission, returned to using opioids after completing the functional restoration program. Unfortunately, opioid status at the one-year follow-up was not available.

Hypothesis 13 predicted that program completers reporting higher levels of pre-treatment opioid use would have systematically lower rates of work return and work retention at one-year post-treatment, and this was supported by the data. Overall, for the NO and YES groups combined , 91.2% of patients completing the

PRIDE Functional Restoration Program reported working during the year after rehabilitation, while 77.7% reported employment at the 1-year follow-up. Work return rates ranged from nearly 94% in the NO subgroup to approximately 76% in the VERY HIGH subgroup. Work retention rates ranged from slightly over 85% in the NO subgroup to approximately 55% in the VERY HIGH subgroup. Thus, among program completers, the YES group was twice as likely as the NO group to have not returned to work during the year after treatment, and more than 2.6 times as likely as the NO group to not be working at the time of the one-year follow-up. Furthermore, the VERY HIGH subgroup was more than 4.5 times more likely than the NO subgroup to have not returned or retained work. Binary logistic regression analyses indicated that opioid level accounted for 2.5% of the variance in work return and 5.7% if the variance in work retention, further supporting the hypothesis.

Neither of the two similar studies found in the literature reported work-return and work-retention rates based on pre-treatment opioid status, so comparisons with the findings of the present study could not be made. One study did not report any long-term treatment outcomes (Rome, et al., 2004). The other reported work-return rates at a 6-month follow-up, based on opioid use status at discharge only. However, in a study of chronic musculoskeletal pain disability, patients who did not return to work during the year following completion of functional restoration were twice as likely to have a current substance disorder diagnosis as patients who did return to work. Similarly, patients who did not retain work were also twice as likely to have a current substance disorder

diagnosis as patients who were working at the one-year follow-up (Dersh, 2000). Thus, once again, a possible explanation for the present study's finding related to post-treatment work status is that patients who discontinued opioid medications at admission, returned to using opioids after completing the functional restoration program.

Limitations of the Present Study

The present study represents a good starting point in the exploration of the role of pre-treatment level of opioid use in chronic pain rehabilitation outcomes for patients participating in functional restoration. Like all studies, it is not without limitations. One limitation of the study is that pre-treatment level of opioid use was based, in part, on self-report. As with other substances, patients might under-report their level of use. This concern was addressed through the selective verification of medical records. Pre-treatment level of opioid use was determined through a review of multiple clinician reports, including, in some cases, notes from the prescribing physician. This concern was also addressed statistically, by a two-fold strategy of data analysis. Patients were first identified as having reported pre-treatment opioid use or not, and the subsequent groups were compared. Then, patients were classified into subgroups based on the dosage they reported, and data analyses were repeated.

A second limitation related to opioid use is the lack of objective confirmation (i.e., urine toxicology) that patients participating in functional restoration have discontinued opioid use. If continued opioid use was suspected

by clinicians or suggested by fellow patients, these concerns were discussed with the patient. Urine toxicology screening was used in some cases. If opioid use continued following these interventions, patients were offered inpatient detoxification. Patients who refused detoxification were discharged as non-completers due to non-compliance.

Another limitation of the study was the difficulty in determining dosage-based data recorded in medical charts. In the vast majority of cases, dose was specified. However, some cases were less specific; for example, listing a number of tablets taken, instead of a dose. A conservative approach was taken in these cases and the tablets were assumed to be of the lowest dose available of a given medication. When no dose could be determined, (for example, "prn" or "occasional" use), cases were included in the two group comparisons, but excluded from the dosage level comparisons.

The dosage levels used to classify patients in the present study were developed a priori. The sample size of the VERY HIGH level was small relative to the other levels. As such, power might have been insufficient to yield significant results among these patients. Likewise, the cut-offs for the levels themselves might be improved upon in preparation for future studies.

Logistic regression, which was utilized for the dichotomous 1-year outcome variables, does not have an equivalent to the R-squared produced by linear regression. R-squared terms presented for binary logistic regression analyses in the present study are actually pseudo-R-squared statistics. Thus, interpretations of the amount of variance accounted for by opioid dose in rates of

work return, work retention, new surgery, and healthcare utilization must be interpreted with caution.

Another possible limitation of the study is the sample size for post-treatment pain-related disability and health-related quality of life scores. Due to limitations of the dataset utilized in this study, the post-treatment sample for the OSW and SF-36 is much smaller than pre-treatment sample. While the results for these measures appeared consistent with other study findings, they must be interpreted with caution.

Another important consideration for the present study is that pre-treatment findings were based on both completers and non-completers of functional restoration, while post-treatment and one-year treatment outcomes were based on completers only. Therefore, conclusions regarding the role of pre-treatment level of opioid use may only be generalizable to patients who complete functional restoration.

Finally, numerous statistical analyses were conducted, which may increase the rate of Type I error. For the present study, this was judged preferable to risking analyses that were too conservative and might fail to capture important effects. Of course, future replication of the findings is recommended.

Directions for Future Research

The present study raises numerous questions to be addressed by future research. Examination of the relationship between pre-treatment level of opioid use and chronic pain rehabilitation outcomes sought to identify linear

relationships. However, these relationships might be better described by higher-order polynomial functions. For that reason, future research might seek to identify higher-order trends that describe more accurately the associations between pre-treatment opioid use and response to functional restoration. Results of the present study indicate that patients who reported pre-treatment opioid use showed similar benefits from functional restoration compared to patients who reported no pre-treatment opioid use, in terms of physical functioning, depressive symptoms, pain report, disability, and health-related quality of life. Nevertheless, patients who reported pre-treatment opioid use showed poorer socioeconomic and health outcomes at one-year follow-up, including work return, work retention, and healthcare utilization rates.

Explanations for these findings are likely multifaceted and are beyond the scope of the present study. However, one avenue of exploration might be to examine the interaction of pre-treatment level of opioid use with prior work-related injury, pre-treatment surgery, and total length of disability by controlling for differences in these variables.

Other possible avenues for exploring differences in socioeconomic and health outcomes include rates of opioid dependence and Axis II diagnoses among these patients. This line of research could begin by gathering information regarding opioid use, abuse, and dependence at the 1-year follow-up. Additional research is needed to account for these differences in treatment outcomes and develop interventions that more effectively address socioeconomic and health issues among patients taking higher doses of pre-treatment opioids.

As mentioned above, pre-treatment findings were based on both completers and non-completers of functional restoration, while post-treatment and one-year treatment outcomes were based on completers only. Thus, conclusions regarding the role of pre-treatment level of opioid use may not be generalizable to patients who do not complete functional restoration. More information must be gathered to determine the differences between patients who report pre-treatment opioid use that complete functional restoration and those who do not complete. Since higher levels of pre-treatment opioid use are associated with increase risk of program non-completion, future research might help identify risk factors for treatment drop-out and subsequent interventions. Lastly, replication of the present study is recommended.

Summary and Conclusions

In summary, the present study found that pre-treatment opioid dose was not associated with clinically significant differences pre-treatment socioeconomic variables, pain report, self-reported disability, or health-related quality of life. At pre-treatment, only patients reporting the highest pre-treatment levels of opioid use showed greater self-reported depressive symptoms. All other pre-treatment opioid levels averaged similar self-reported depressive symptoms. Likewise, clinician-ratings of depressive symptoms were not associated with pre-treatment level of opioid use. However, pre-treatment opioid use was associated with differences in pre-treatment health variables, with patients reporting pre-treatment

opioid use being one and a half times more likely to report a prior work-related injury and a pre-treatment surgery.

Moreover, level of pre-treatment opioid use did not play a significant role in post-treatment outcomes related to gains in physical functioning, pain report, self-reported disability, or health-related quality of life. In general, patients who reported pre-treatment opioid use who completed functional restoration showed similar treatment gains to patients who did not report pre-treatment opioid use. Like pre-treatment findings, only patients reporting the highest pre-treatment levels of opioid use showed greater self-reported depressive symptoms at post-treatment. But, symptoms fell in the low range of "mild to moderate" and represented a significant improvement over pre-treatment levels. Furthermore, clinician-ratings of post-treatment depressive symptoms were not associated with pre-treatment level opioid use.

As expected, higher levels of pre-treatment opioid use were associated with less desirable MMPI profiles. Opioid use was associated with increased elevation rates for all clinical scales, except Scale 9 (Hypomania). Analysis of MMPI profile clusters showed that lower opioid levels were associated with a greater proportion of patients demonstrating defended profiles, while higher opioid levels of were associated with a greater proportion of patients demonstrating distressed profiles. This finding indicates that differences in psychological functioning are associated with level of pre-treatment opioid use.

Further exploration of MMPI profiles, determined that 75% of patients with valid MMPI profiles could be classified into one of four profiles: Normal,

Conversion V, Neurotic Triad, and Disability. For all opioid levels, the majority of patients were classified as producing the Disability profile. However, the proportions of this profile increased with pre-treatment opioid dose, indicating higher levels of psychopathology in patients taking higher doses of opioids.

Although, pre-treatment opioid use was not associated with differences in one-year case settlement rates, rates of new injury to the same body part, or rates of new surgery to the same body part (except among those taking the highest levels of opioids), it was associated with poorer outcomes on other important socioeconomic and health variables. Contrary to the many positive treatment outcomes reviewed above, patients who reported pre-treatment opioid use showed significantly higher rates of healthcare utilization, and lower rates of work return and work retention. Additionally, level of pre-treatment opioid use was inversely related to rate of program completion. While these differences were significant statistically and clinically, it should be noted that the majority of patients, even at the highest pre-treatment opioid levels, returned and retained work at the 1-year follow-up.

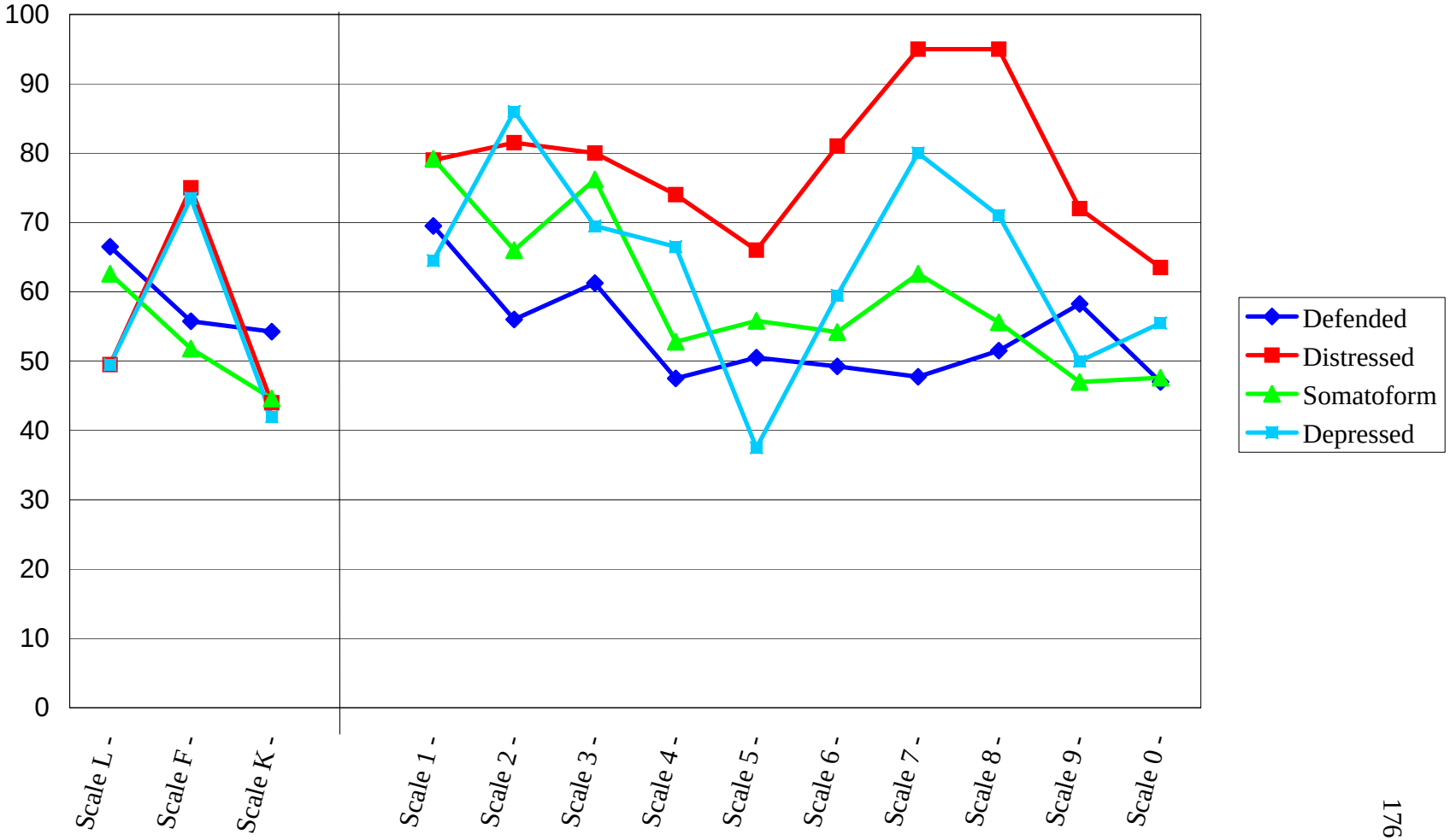
In conclusion, findings of the present study lend continued support for the efficacy of functional restoration in the treatment of chronic pain. They also suggest that patients who discontinue opioid medications show similar benefits in depressive symptoms, pain report, disability, and quality of life from completing functional restoration treatment as patients who did not report pre-treatment opioid use. Perhaps, this finding will help improve clinicians' attitudes toward chronic pain patients taking opioid medications and help ameliorate "opiophobia."

Despite positive treatment outcomes in other areas, however, patients taking pre-treatment opioids show significantly poorer outcomes on some important socioeconomic and health variables. Given this finding, treating clinicians could modify interventions to more closely address the difficulties these patients have with returning to and retaining work. Thus, pre-treatment level of opioid use, as described in this study, could be useful guide for identifying patients who are at increased risk for poorer socioeconomic outcomes, and targeting treatment interventions to improve the likelihood of program completion and positive long-term treatment outcomes for these patients.

APPENDIX A
Figures

Figure 1

MMPI Profiles Produced by K-mean Cluster Analysis of the Total Opioid Cohort.



APPENDIX B
Tables

Table 1

Opioid Dosage Conversions

Opioid Medication	Equianalgesic Doses (mg)		Lowest Available Dose (mg)	
Morphine (oral)	30		15	
Codeine	200		15	
Darvocet	65		50	
Darvon-N	65		100	
Duragesic Patch	25	mcg/h	25	mcg/h
Fioricet with Codeine	20		30	
Fiorinal with Codeine	20		30	
Hydrocodone	20		5	
Lorcet	20		5	
Lortab	20		2.5	
Methadone	20		5	
Norco	20		5	
Oxycodone	30		10	
Pentazocine	180		50	
Percocet	20		2.5	
Percodan	20		5	
Soma with Codeine	200		16	
Talacen	180		25	
Tylenol with Codeine-#2	200		15	
Tylenol with Codeine-#3	200		30	
Tylenol with Codeine-#4	200		60	
Vicoden	20		5	
Vicoden ES	20		7.5	
Vicoprofen	20		7.5	
Zydone	20		5	

(Polatin & Gajraj, 2002; Beers & Berkow, 1999; Global RPh, 2005)

Table 2

Basic Demographic Variables: Opioid Groups

Variable	NO	YES
N=1226 n (% of Total)	630 (51.39)	596 (48.61)
Gender (% male)	50.00	48.20
Mean Age in Years (SD)	43.41 (1.00)	44.11 (9.32)
Race (%)		
Caucasian	49.40	58.00
African-American	23.90	25.40
Hispanic	24.50	15.90
Other	2.30	0.70
Mean Years of Education (SD)	11.37 (3.34)	11.61 (2.68)
Program Completion (%)	81.40	74.00

Table 3

Statistical Analyses of Basic Demographic Variables: Opioid Groups

Gender					
Group		% Male	χ^2	df	p
NO		50.00	.417	1	.518
YES		51.80			
Age					
Group		Mean (SD)	F	df	p
NO		43.41 (10.00)	1.594	1, 1224	.207
YES		44.11 (9.32)			
Race					
Group		Percent	χ^2	df	p
NO					
	Caucasian	49.40	20.196	3	<.001
	African American	23.90			
	Hispanic	24.50			
	Other	2.30			
YES					
	Caucasian	58.00			
	African American	25.40			
	Hispanic	15.90			
	Other	0.70			

Table 3 (cont.)

Education (years)				
Group	Mean (SD)	F	df	p
NO	11.37 (3.34)	1.546	1, 074	.214
YES	11.61 (2.68)			
Program Completion				
Group	Percent	χ^2	df	p
NO	81.40	9.807	1	.002
YES	74.00			

Table 4

Basic Demographic Variables: Opioid Subgroups

Variable	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=1146					
n	630	267	112	78	59
(% of Total)	(54.97)	(23.30)	(9.77)	(6.81)	(5.15)
Gender-% Male	50.00	50.00	39.40	47.20	52.90
Mean Age in Years (SD)	43.32 (9.90)	44.66 (9.45)	43.37 (9.25)	42.92 (9.55)	44.75 (8.89)
Race (%)					
Caucasian	49.20	48.60	59.20	63.20	80.00
African-American	23.90	30.00	27.60	22.10	12.00
Hispanic	24.60	20.20	12.20	14.70	8.00
Other	2.30	1.20	1.00	0.00	0.00
Mean Years of Education (SD)	11.37 (3.34)	11.44 (2.97)	11.53 (2.48)	11.81 (1.75)	11.43 (2.37)
Program Completion (%)	81.50	76.00	68.70	68.10	70.60

Table 5

Statistical Analyses of Basic Demographic Variables: Opioid Subgroups

Age				
Subgroup	Mean (SD)	F	df	p
NO	43.32 (9.90)	1.139	4, 1095	.336
LOW	44.66 (9.45)			
MEDIUM	43.37 (9.25)			
HIGH	42.92 (9.55)			
VERY HIGH	44.75 (8.89)			

Gender				
Subgroup	% Male	χ^2	df	p
NO	50.00	4.415	4	.353
LOW	50.00			
MEDIUM	60.60			
HIGH	52.80			
VERY HIGH	47.1			

Table 5 (cont.)

Race Subgroup	Percent	χ^2	df	p
NO				
Caucasian	49.20	34.736	12	.001
African American	23.90			
Hispanic	24.60			
Other	2.30			
LOW				
Caucasian	48.60			
African American	30.00			
Hispanic	20.20			
Other	1.20			
MEDIUM				
Caucasian	59.20			
African American	27.60			
Hispanic	12.20			
Other	1.00			
HIGH				
Caucasian	63.20			
African American	22.10			
Hispanic	14.70			
Other	0.00			
VERY HIGH				
Caucasian	80.00			
African American	12.00			
Hispanic	8.00			
Other	0.00			

Table 5 (cont.)

Education (years)				
Subgroup	Mean (SD)	F	df	p
NO	11.37 (3.34)	.304	4, 960	.875
LOW	11.44 (2.97)			
MEDIUM	11.53 (2.48)			
HIGH	11.81 (1.75)			
VERY HIGH	11.43 (2.37)			
Program Completion				
Subgroup	Percent	χ^2	df	p
NO	81.50	15.738	4	.003
LOW	76.00			
MEDIUM	68.70			
HIGH	68.10			
VERY HIGH	70.60			

Table 6

Pre-treatment Socioeconomic and Health Variables: Opioid Groups

Variable	NO	YES
N=1226 n (% of Total)	630 (51.39)	596 (48.61)
Mean Salary at Time of Injury in Dollars per Week (SD)	508.60 (288.86)	493.02 (247.85)
Mean Workers Compensation Benefit in Dollars per Week (SD)	354.12 (135.24)	328.61 (131.71)
Pre-treatment Case Settlement (%)	96.60	96.90
Mean Total Length of Disability in Months (SD)	11.05 (10.76)	15.60 (17.25)
Prior Work-related Injury (%)	35.80	43.50
Pre-treatment Surgery (%)	37.60	49.50
Attorney Retention (%)	18.10	21.00
SSDI/SSI (%)	0.60	2.60

Table 7

Statistical Analyses of Pre-treatment Socioeconomic and Health Variables: Opioid Groups

Salary at Time of Injury (Dollars per Week)				
Group	Mean (SD)	F	df	p
NO	508.60 (288.86)	.877	1, 1052	.349
YES	493.02 (247.85)			
Workers Compensation Benefit (Dollars per Week)				
Group	Mean (SD)	F	df	p
NO	354.12 (135.24)	7.208	1, 787	.007
YES	328.60 (131.71)			
Pre-treatment Case Settlement				
Group	Percent	χ^2	df	p
NO	96.60	.087	1	.768
YES	96.90			

Table 7 (cont.)

Total Length of Disability (months)				
Group	Mean (SD)	F	df	p
NO	11.05 (10.76)	27.137	1, 1070	<.001
YES	15.60 (17.25)			
Prior Work-related Injury				
Group	Percent	χ^2	df	p
NO	35.80	6.262	1	.012
YES	43.50			
Pre-treatment Surgery				
Group	Percent	χ^2	df	p
NO	44.80	15.471	1	<.001
YES	49.50			
Attorney Retention				
Group	Percent	χ^2	df	p
NO	18.10	1.279	1	.258
YES	21.00			
SSDI/SSI				
Group	Percent	χ^2	df	p
NO	0.60	7.195	1	.007
YES	2.60			

Table 8

Pre-treatment Socioeconomic and Health Variables: Opioid Subgroups

Variable	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=1146					
n	630	267	112	78	59
(% of Total)	(54.97)	(23.30)	(9.77)	(6.81)	(5.15)
Mean Salary at Time of Injury in Dollars per Week (SD)	508.60 (288.86)	510.10 (252.21)	480.93 (274.22)	468.79 (251.89)	435.96 (178.14)
Mean Workers Compensation Benefit in Dollars per Week (SD)	354.12 (135.24)	340.57 (134.95)	308.61 (131.41)	318.31 (141.93)	325.17 (96.76)
Pre-treatment Case Settlement (%)	96.60	96.80	98.80	96.90	97.70
Mean Total Length of Disability in Months (SD)	11.08 (10.76)	15.18 (17.94)	18.65 (19.66)	13.02 (9.13)	15.66 (11.31)
Prior Work-related Injury (%)	35.80	45.50	41.80	48.30	43.90
Pre-treatment Surgery (%)	37.70	43.10	58.10	52.30	64.40
Attorney Retention (%)	18.20	22.60	23.10	20.80	17.50
SSDI/SSI (%)	0.60	2.80	1.00	1.40	3.90

Table 9

Statistical Analyses of Pre-treatment Socioeconomic and Health Variables: Opioid Subgroups

Salary at Time of Injury (Dollars per Week)				
Subgroup	Mean (SD)	F	df	p
NO	508.60 (288.86)	1.133	4, 948	.339
LOW	510.10 (252.21)			
MEDIUM	480.93 (274.22)			
HIGH	468.79 (251.89)			
VERY HIGH	435.96 (178.14)			
Workers Compensation Benefit (Dollars per Week)				
Subgroup	Mean (SD)	F	df	p
NO	354.12 (135.24)	2.417	4, 707	.047
LOW	340.57 (134.95)			
MEDIUM	308.61 (131.41)			
HIGH	318.31 (141.93)			
VERY HIGH	325.17 (96.76)			

Table 9 (cont.)

Pre-treatment Case Settlement				
Subgroup	Percent	χ^2	df	p
NO	96.60	1.335	4	.855
LOW	96.80			
MEDIUM	98.80			
HIGH	96.90			
VERY HIGH	97.70			
Total Length of Disability (months)				
Subgroup	Mean (SD)	F	df	p
NO	11.08 (10.76)	8.267	4, 961	<.001
LOW	15.18 (17.94)			
MEDIUM	18.65 (19.66)			
HIGH	13.02 (9.13)			
VERY HIGH	15.66 (11.31)			

Table 9 (cont.)

Prior Work-related Injury				
Subgroup	Percent	χ^2	df	p
NO	35.80	8.476	4	.076
LOW	45.50			
MEDIUM	41.80			
HIGH	48.30			
VERY HIGH	43.90			
Pre-treatment Surgery				
Subgroup	Percent	χ^2	df	p
NO	37.70	25.067	4	<.001
LOW	43.10			
MEDIUM	58.10			
HIGH	52.30			
VERY HIGH	64.40			
Attorney Retention				
Subgroup	Percent	χ^2	df	p
NO	18.2	2.561	4	.634
LOW	22.6			
MEDIUM	23.1			
HIGH	20.8			
VERY HIGH	17.5			

Table 9 (cont.)

SSDI/SSI Subgroup	Percent	χ^2	df	p
NO	0.60	8.963	4	.062
LOW	2.80			
MEDIUM	1.00			
HIGH	1.40			
VERY HIGH	3.90			

Table 10

Pre-treatment Psychosocial Variables: Opioid Groups

Variable Mean (SD)	NO	YES
N=1226 n (% of Total)	630 (51.39)	596 (48.61)
BDI	13.71 (9.33)	16.43 (10.07)
HAM-D	13.41 (6.32)	15.41 (5.70)
MVAS	85.77 (26.08)	94.92 (20.51)
OSW	35.32 (14.57)	41.35 (14.86)
SF-36MHS	40.12 (9.61)	38.09 (9.60)
SF-36PHS	30.83 (5.84)	29.61 (5.92)

Table 11

Statistical Analyses of Pre-treatment Psychosocial Variables: Opioid Groups

BDI				
Group	Mean (SD)	F	df	p
NO	13.71 (9.33)	19.88	1, 1008	<.001
YES	16.43 (10.07)			
HAM-D				
Group	Mean (SD)	F	df	p
NO	13.41 (6.32)	27.557	1, 1000	<.001
YES	15.41 (5.70)			
MVAS				
Group	Mean (SD)	F	df	p
NO	85.77 (26.08)	37.849	1, 1006	<.001
YES	94.92 (20.51)			
OSW				
Group	Mean (SD)	F	df	p
NO	35.32 (14.57)	39.22	1, 933	<.001
YES	41.35 (14.86)			

Table 11 (cont.)

SF-36MHS				
Group	Mean (SD)	F	df	p
NO	40.12 (9.61)	10.876	1, 971	.001
YES	38.09 (9.60)			
SF-36PHS				
Group	Mean (SD)	F	df	p
NO	30.83 (5.84)	10.437	1, 971	.001
YES	29.61 (5.92)			

Table 12

Chi-square Analysis of Pre-treatment BDI Categories: Opioid Groups

Pre-treatment BDI Group	n	%	χ^2	df	p
NO	528				
Absence of Depression (<10)		39.6	20.667	3	<.001
Mild to Moderate (10-18)		34.8			
Moderate to Severe (19-29)		19.1			
Severe (>29)		6.4			
YES	482				
Absence of Depression (<10)		27.2			
Mild to Moderate (10-18)		38.4			
Moderate to Severe (19-29)		23.4			
Severe (>29)		11.0			

Table 13

Chi-square Analysis of Pre-treatment HAM-D Categories: Opioid Groups

Pre-treatment HAM-D Group	n	%	χ^2	df	p
NO	524				
None to Minimal (<12)		38.9	22.553	3	<.001
Mild to Moderate (12-20)		49.6			
Moderate to Severe (21-29)		10.7			
Severe (30+)		0.8			
YES	476				
None to Minimal (<12)		26.2			
Mild to Moderate (12-20)		55.4			
Moderate to Severe (21-29)		17.6			
Severe (30+)		0.8			

Table 14

Chi-square Analysis of Pre-treatment MVAS Categories: Opioid Groups

Pre-treatment MVAS Group	n	%	χ^2	df	p
NO	528				
Mildly Disabling (≤40)		5.3	51.130	4	<.001
Moderately Disabling (41-70)		22.2			
Severely Disabling (71-100)		43.0			
Very Severely Disabling (101-130)		26.5			
Extremely Disabling (≥131)		3.0			
YES	480				
Mildly Disabling (≤40)		0.4			
Moderately Disabling (41-70)		11.5			
Severely Disabling (71-100)		46.9			
Very Severely Disabling (101-130)		39.2			
Extremely Disabling (≥131)		2.1			

Table 15

Chi-square Analysis of Pre-treatment MVAS Extremes: Opioid Groups

Pre-treatment MVAS Group	n	%	χ^2	df	p
NO	528				
Not Extreme (0-70)		27.5	38.122	1	<.001
Extreme (71-150)		72.5			
YES	480				
Not Extreme (0-70)		11.9			
Extreme (71-150)		88.1			

Table 16

Chi-square Analysis of Pre-treatment OSW Categories: Opioid Groups

Pre-treatment OSW Group	n	%	χ^2	df	p
NO	475				
Minimal Disability (0-20)		15.4	42.546	4	<.001
Moderate Disability (21-40)		54.3			
Severe Disability (41-60)		25.5			
Crippling Disability (61-80)		4.0			
Bedbound or Exaggerated Disability (81-100)		0.8			
YES	460				
Minimal Disability (0-20)		6.5			
Moderate Disability (21-40)		47.6			
Severe Disability (41-60)		33.7			
Crippling Disability (61-80)		11.7			
Bedbound or Exaggerated Disability (81-100)		0.4			

Table 17

Post-treatment Psychosocial Variables: Opioid Groups (Completers Only)

Variable Mean (SD)	NO	YES
N=954 n (% of Total)	513 (53.77)	441 (46.23)
BDI	8.06 (6.93)	9.79 (7.87)
BDI DELTA	5.72 (7.40)	6.11 (7.81)
HAM-D	7.87 (4.42)	9.62 (4.78)
HAM-D DELTA	5.51 (5.67)	5.59 (5.07)
MVAS	59.41 (28.20)	70.24 (28.15)
MVAS DELTA	26.35 (26.31)	23.04 (25.59)
OSW	20.66 (7.06)	23.56 (7.58)
OSW DELTA	-0.67 (12.33)	-0.50 (10.39)
SF-36MHS	46.67 (8.76)	46.29 (9.20)
SF-36MHS DELTA	-5.24 (10.49)	-7.01 (11.59)
SF-36PHS	36.68 (6.97)	34.84 (6.24)
SF-36PHS DELTA	-4.64 (8.00)	-4.21 (7.72)

Table 18

Statistical Analyses of Post-treatment Psychosocial Variables: Opioid Groups (Completers Only)

BDI				
Group	Mean (SD)	F*	df	p
NO	8.06 (6.93)	3.290	1, 818	.070
YES	9.79 (7.87)			
BDI DELTA				
Group	Mean (SD)	F	df	p
NO	5.72 (7.40)	0.537	1, 815	.464
YES	6.11 (7.81)			
HAM-D				
Group	Mean (SD)	F	df	p
NO	7.87 (4.42)	31.779	1, 879	<.001
YES	9.62 (4.78)			
HAM-D DELTA				
Group	Mean (SD)	F	df	p
NO	5.51 (5.67)	0.043	1, 807	.836
YES	5.59 (5.07)			

*ANCOVA

Table 18 (cont.)

MVAS				
Group	Mean (SD)	F*	df	p
NO	59.41 (28.20)	12.810	1, 817	<.001
YES	70.24 (28.15)			
MVAS DELTA				
Group	Mean (SD)	F	df	p
NO	26.35 (26.31)	3.287	1, 814	.070
YES	23.04 (25.59)			
OSW				
Group	Mean (SD)	F*	df	p
NO	20.66 (7.06)	1.150	1, 168	.268
YES	23.56 (7.58)			
OSW DELTA				
Group	Mean (SD)	F	df	p
NO	-0.67 (12.33)	0.009	1, 167	.923
YES	-0.50 (10.39)			

*ANCOVA

Table 18 (cont.)

SF-36MHS				
Group	Mean (SD)	F	df	p
NO	46.67 (8.76)	.110	1, 241	.741
YES	46.29 (9.20)			
SF-36MHS DELTA				
Group	Mean (SD)	F	df	p
NO	-5.24 (10.49)	1.416	1, 223	.235
YES	-7.01 (11.59)			
SF-36PHS				
Group	Mean (SD)	F	df	p
NO	36.68 (6.97)	4.496	1, 241	.035
YES	34.84 (6.24)			
SF-36PHS DELTA				
Group	Mean (SD)	F	df	p
NO	-4.64 (8.00)	0.157	1, 223	.692
YES	-4.21 (7.72)			

*ANCOVA

Table 19

Chi-square Analysis of Post-treatment BDI Categories: Opioid Groups (Completers Only)

Post-treatment BDI					
Group	n	%	χ^2	df	p
NO	477				
Absence of Depression (<10)		67.5	7.725	3	.052
Mild to Moderate (10-18)		23.1			
Moderate to Severe (19-29)		8.0			
Severe (>29)		1.5			
YES	406				
Absence of Depression (<10)		59.6			
Mild to Moderate (10-18)		25.9			
Moderate to Severe (19-29)		12.1			
Severe (>29)		2.5			

Table 20

Chi-square Analysis of Post-treatment HAM-D Categories: Opioid Groups (Completers Only)

Post-treatment HAM-D Group	n	%	χ^2	df	p
NO	476				
None to Minimal (<12)		79.2	22.849	2	.000
Mild to Moderate (12-20)		20.0			
Moderate to Severe (21-29)		0.8			
Severe (30+)		0.0			
YES	405				
None to Minimal (<12)		65.4			
Mild to Moderate (12-20)		31.6			
Moderate to Severe (21-29)		3.0			
Severe (30+)		0.0			

Table 21

Chi-square Analysis of Post-treatment MVAS Categories: Opioid Groups (Completers Only)

Post-treatment MVAS					
Group	n	%	χ^2	df	p
NO	478				
Mildly Disabling (≤40)		27.0	32.126	4	<.001
Moderately Disabling (41-70)		39.1			
Severely Disabling (71-100)		26.2			
Very Severely Disabling (101-130)		7.1			
Extremely Disabling (≥131)		0.6			
YES	406				
Mildly Disabling (≤40)		16.5			
Moderately Disabling (41-70)		32.0			
Severely Disabling (71-100)		36.7			
Very Severely Disabling (101-130)		14.0			
Extremely Disabling (≥131)		0.7			

Table 22

Chi-square Analysis of Post-treatment MVAS Extremes: Opioid Groups (Completers Only)

Post-treatment MVAS					
Group	n	%	χ^2	df	p
NO	478				
Not Extreme (0-70)		66.1	27.879	1	<.001
Extreme (71-150)		33.9			
YES	406				
Not Extreme (0-70)		48.5			
Extreme (71-150)		51.5			

Table 23

Chi-square Analysis of Post-treatment OSW Categories: Opioid Groups (Completers Only)

Post-treatment OSW Group	n	%	χ^2	df	p
NO	116				
Minimal Disability (0-20)		54.3	8.289	2	.016
Moderate Disability (21-40)		45.7			
Severe Disability (41-60)		0.0			
Crippling Disability (61-80)		0.0			
Bedbound or Exaggerated Disability (81-100)		0.0			
YES	88				
Minimal Disability (0-20)		35.2			
Moderate Disability (21-40)		63.6			
Severe Disability (41-60)		1.1			
Crippling Disability (61-80)		0.0			
Bedbound or Exaggerated Disability (81-100)		0.0			

Table 24

Pre-treatment Psychosocial Variables: Opioid Subgroups

Variable Mean (SD)	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=1146					
n	630	267	112	78	59
(% of Total)	(54.97)	(23.30)	(9.77)	(6.81)	(5.15)
BDI	13.74 (9.32)	15.88 (9.00)	15.10 (9.50)	17.73 (11.27)	23.02 (13.10)
HAM-D	13.43 (6.30)	15.30 (5.46)	15.26 (5.68)	16.5 (6.08)	17.65 (5.99)
MVAS	85.81 (26.09)	93.82 (20.90)	96.34 (18.04)	97.86 (18.60)	101.15 (19.18)
OSW	35.32 (14.57)	39.86 (14.68)	45.58 (15.88)	43.23 (15.87)	44.51 (14.73)
SF-36MHS	40.12 (9.61)	37.86 (9.24)	39.12 (9.98)	39.52 (10.52)	34.91 (8.50)
SF-36PHS	30.83 (5.84)	29.60 (5.94)	29.36 (6.35)	28.62 (5.19)	29.66 (6.72)

Table 25

Statistical Analyses of Pre-treatment Psychosocial Variables: Opioid Subgroups

BDI				
Subgroup	Mean (SD)	F	df	p
NO	13.74 (9.32)	11.030	4, 903	<.001
LOW	15.88 (9.00)			
MEDIUM	15.10 (9.50)			
HIGH	17.73 (11.27)			
VERY HIGH	23.02 (13.10)			
HAM-D				
Subgroup	Mean (SD)	F	df	p
NO	13.43 (6.30)	8.952	4, 895	<.001
LOW	15.30 (5.46)			
MEDIUM	15.26 (5.68)			
HIGH	16.15 (6.08)			
VERY HIGH	17.65 (5.99)			

Table 25 (cont.)

MVAS				
Subgroup	Mean (SD)	F	df	p
NO	85.81 (26.09)	10.840	4, 901	<.001
LOW	93.82 (20.90)			
MEDIUM	96.34 (18.04)			
HIGH	97.86 (18.60)			
VERY HIGH	101.15 (19.18)			
OSW				
Subgroup	Mean (SD)	F	df	p
NO	35.32 (14.57)	13.401	4, 838	<.001
LOW	39.86 (14.68)			
MEDIUM	45.58 (15.88)			
HIGH	43.23 (15.87)			
VERY HIGH	44.51 (14.73)			

Table 25 (cont.)

SF-36MHS				
Subgroup	Mean (SD)	F	df	p
NO	40.12 (9.61)	4.325	4, 873	.002
LOW	37.86 (9.24)			
MEDIUM	39.12 (9.98)			
HIGH	39.52 (10.52)			
VERY HIGH	34.91 (8.50)			
SF-36PHS				
Subgroup	Mean (SD)	F	df	p
NO	30.83 (5.84)	3.471	4, 873	.008
LOW	29.60 (5.94)			
MEDIUM	29.36 (6.35)			
HIGH	28.62 (5.19)			
VERY HIGH	29.66 (6.72)			

Table 26

Chi-square Analysis of Pre-treatment BDI Categories: Opioid Subgroups

Pre-treatment BDI Subgroup	n	%	χ^2	df	p (linear)
NO	527				
Absence of Depression (<10)		39.5	28.607	1	<.001
Mild to Moderate (10-18)		34.9			
Moderate to Severe (19-29)		19.2			
Severe (>29)		6.5			
LOW	195				
Absence of Depression (<10)		25.1			
Mild to Moderate (10-18)		42.1			
Moderate to Severe (19-29)		24.6			
Severe (>29)		8.2			
MEDIUM	86				
Absence of Depression (<10)		37.2			
Mild to Moderate (10-18)		31.4			
Moderate to Severe (19-29)		20.9			
Severe (>29)		10.5			
HIGH	59				
Absence of Depression (<10)		27.1			
Mild to Moderate (10-18)		32.2			
Moderate to Severe (19-29)		27.1			
Severe (>29)		13.6			

Table 26 (cont.)

VERY HIGH	41	
Absence of Depression (<10)		14.6
Mild to Moderate (10-18)		26.8
Moderate to Severe (19-29)		29.3
Severe (>29)		29.3

Table 27

Chi-square Analysis of Pre-treatment HAM-D Categories: Opioid Subgroups

Pre-treatment HAM-D Subgroup	n	%	χ^2	df	p (linear)
NO	523				
None to Minimal (<12)		38.8	22.864	1	<.001
Mild to Moderate (12-20)		49.7			
Moderate to Severe (21-29)		10.7			
Severe (30+)		0.8			
LOW	192				
None to Minimal (<12)		27.1			
Mild to Moderate (12-20)		55.2			
Moderate to Severe (21-29)		17.2			
Severe (30+)		0.5			
MEDIUM	86				
None to Minimal (<12)		30.2			
Mild to Moderate (12-20)		53.2			
Moderate to Severe (21-29)		15.0			
Severe (30+)		1.2			
HIGH	59				
None to Minimal (<12)		23.7			
Mild to Moderate (12-20)		50.8			
Moderate to Severe (21-29)		25.4			
Severe (30+)		0.0			

Table 27 (cont.)

VERY HIGH	40	
None to Minimal (<12)		17.5
Mild to Moderate (12-20)		52.5
Moderate to Severe (21-29)		27.5
Severe (30+)		2.5

Table 28

Chi-square Analysis of Pre-treatment MVAS Categories: Opioid Subgroups

Pre-treatment MVAS					
Subgroup	n	%	χ^2	df	p (linear)
NO	528				
Mildly Disabling (≤40)		5.3	31.338	1	<.001
Moderately Disabling (41-70)		22.2			
Severely Disabling (71-100)		43.0			
Very Severely Disabling (101-130)		26.5			
Extremely Disabling (≥131)		3.0			
LOW	193				
Mildly Disabling (≤40)		0.5			
Moderately Disabling (41-70)		11.9			
Severely Disabling (71-100)		46.6			
Very Severely Disabling (101-130)		38.9			
Extremely Disabling (≥131)		2.1			
MEDIUM	86				
Mildly Disabling (≤40)		0.0			
Moderately Disabling (41-70)		9.3			
Severely Disabling (71-100)		51.2			
Very Severely Disabling (101-130)		38.4			
Extremely Disabling (≥131)		1.2			

Table 28 (cont.)

HIGH	59	
Mildly Disabling (≤40)		0.0
Moderately Disabling (41-70)		8.5
Severely Disabling (71-100)		44.1
Very Severely Disabling (101-130)		45.8
Extremely Disabling (≥131)		1.7
VERY HIGH	41	
Mildly Disabling (≤40)		0.0
Moderately Disabling (41-70)		4.9
Severely Disabling (71-100)		46.3
Very Severely Disabling (101-130)		46.3
Extremely Disabling (≥131)		2.4

Table 29

Chi-square Analysis of Pre-treatment MVAS Extremes: Opioid Subgroups

Pre-treatment MVAS Subgroup	n	%	χ^2	df	p
NO	528				
Not Extreme (0-70)		27.5	41.695	4	<.001
Extreme (71-150)		72.5			
LOW	193				
Not Extreme (0-70)		12.4			
Extreme (71-150)		87.6			
MEDIUM	86				
Not Extreme (0-70)		9.3			
Extreme (71-150)		90.7			
HIGH	59				
Not Extreme (0-70)		8.5			
Extreme (71-150)		91.5			
VERY HIGH	41				
Not Extreme (0-70)		4.9			
Extreme (71-150)		95.1			

Table 30

Chi-square Analysis of Pre-treatment OSW Categories: Opioid Subgroups

Pre-treatment OSW Group	n	%	χ^2	df	p (linear)
NO	475				
Minimal Disability (0-20)		15.4	38.039	1	<.001
Moderate Disability (21-40)		54.3			
Severe Disability (41-60)		25.5			
Crippling Disability (61-80)		4.0			
Bedbound or Exaggerated Disability (81-100)		.8			
LOW	192				
Minimal Disability (0-20)		8.3			
Moderate Disability (21-40)		49.5			
Severe Disability (41-60)		31.3			
Crippling Disability (61-80)		10.9			
Bedbound or Exaggerated Disability (81-100)		0.0			
MEDIUM	76				
Minimal Disability (0-20)		6.6			
Moderate Disability (21-40)		35.5			
Severe Disability (41-60)		42.1			
Crippling Disability (61-80)		13.2			
Bedbound or Exaggerated Disability (81-100)		2.6			

Table 30 (cont.)

HIGH	57	
Minimal Disability (0-20)		1.8
Moderate Disability (21-40)		52.6
Severe Disability (41-60)		26.3
Crippling Disability (61-80)		19.3
Bedbound or Exaggerated Disability (81-100)		0.0
VERY HIGH	73	
Minimal Disability (0-20)		4.7
Moderate Disability (21-40)		41.9
Severe Disability (41-60)		34.9
Crippling Disability (61-80)		18.6
Bedbound or Exaggerated Disability (81-100)		0.0

Table 31

Post-treatment Psychosocial Variables: Opioid Subgroups (Completers Only)

Variable Mean (SD)	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=887					
n	513	205	75	53	41
(% of Total)	(57.84)	(23.11)	(8.46)	(5.98)	(4.62)
BDI	8.07	9.67	9.74	9.66	12.58
	(6.93)	(7.28)	(7.60)	(8.78)	(12.19)
BDI DELTA	5.72	5.75	4.79	8.32	8.93
	(7.40)	(7.64)	(6.55)	(8.50)	(9.42)
HAM-D	7.88	9.76	9.39	9.43	10.81
	(4.41)	(4.74)	(4.93)	(4.81)	(5.95)
HAM-D DELTA	5.51	5.48	5.77	6.88	5.29
	(5.67)	(5.14)	(4.98)	(5.27)	(4.90)
MVAS	59.48	71.85	68.79	67.75	69.58
	(28.19)	(28.25)	(27.35)	(29.72)	(31.99)
MVAS DELTA	26.35	20.78	24.54	29.80	28.57
	(26.31)	(25.83)	(24.16)	(29.68)	(28.75)
OSW	20.66	22.68	20.38	22.50	21.00
	7.059	6.540	6.865	7.750	3.665
OSW DELTA	-0.67	-0.038	6.17	7.20	-2.14
	12.33	10.62	10.21	10.73	10.07
SF-36MHS	46.67	46.34	47.76	49.99	50.72
	(8.76)	(9.07)	(7.00)	(8.83)	(6.83)
SF-36MHS DELTA	-5.24	-6.24	-11.18	-8.45	-10.61
	(10.49)	(10.54)	(15.90)	(11.37)	(12.76)
SF-36PHS	36.68	34.34	34.43	33.37	34.10
	(6.97)	(6.26)	(3.72)	(4.91)	(4.00)
SF-36PHS DELTA	-4.64	-5.64	-0.80	-1.54	-1.04
	(8.00)	(7.60)	(8.87)	(6.48)	(7.10)

Table 32

Statistical Analyses of Post-treatment Psychosocial Variables: Opioid Subgroups (Completers Only)

BDI				
Subgroup	Mean (SD)	F*	df	p
NO	7.95 (6.89)	1.213	4, 732	.304
LOW	9.64 (7.23)			
MEDIUM	9.48 (7.69)			
HIGH	9.66 (8.78)			
VERY HIGH	12.60 (12.49)			

BDI DELTA				
Subgroup	Mean (SD)	F	df	p
NO	5.72 (7.40)	2.720	4, 731	.029
LOW	5.75 (7.64)			
MEDIUM	4.79 (6.55)			
HIGH	8.32 (8.50)			
VERY HIGH	8.93 (9.42)			

*ANCOVA

Table 32 (cont.)

HAM-D				
Subgroup	Mean (SD)	F	df	p
NO	7.88 (4.41)	8.267	4, 785	<.001
LOW	9.76 (4.74)			
MEDIUM	9.39 (4.93)			
HIGH	9.43 (4.81)			
VERY HIGH	10.81 (5.95)			
HAM-D DELTA				
Subgroup	Mean (SD)	F	df	p
NO	5.51 (5.67)	0.698	4, 723	.594
LOW	5.48 (5.14)			
MEDIUM	5.77 (4.98)			
HIGH	6.88 (5.27)			
VERY HIGH	5.29 (4.90)			

Table 32 (cont.)

MVAS				
Subgroup	Mean (SD)	F*	df	p
NO	58.73 (28.49)	3.278	4, 731	.011
LOW	70.87 (28.21)			
MEDIUM	67.43 (26.97)			
HIGH	67.75 (29.72)			
VERY HIGH	68.80 (30.39)			
MVAS DELTA				
Subgroup	Mean (SD)	F	df	p
NO	26.35 (26.31)	1.779	4, 730	.131
LOW	20.78 (25.83)			
MEDIUM	24.54 (24.16)			
HIGH	29.80 (29.68)			
VERY HIGH	28.57 (28.75)			

*ANCOVA

Table 32 (cont.)

OSW				
Subgroup	Mean (SD)	F	df	p
NO	20.66 (7.059)	0.690	4, 171	.600
LOW	22.68 (6.540)			
MEDIUM	20.38 (6.865)			
HIGH	22.50 (7.750)			
VERY HIGH	21.00 (3.665)			
OSW DELTA				
Subgroup	Mean (SD)	F	df	p
NO	-0.67 (12.33)	1.471	1, 139	.214
LOW	-0.038 (10.62)			
MEDIUM	6.17 (10.21)			
HIGH	7.20 (10.73)			
VERY HIGH	-2.14 (10.07)			

Table 32 (cont.)

SF-36MHS				
Subgroup	Mean (SD)	F	df	p
NO	46.67 (8.76)	.863	4, 208	.487
LOW	46.34 (9.07)			
MEDIUM	47.76 (7.00)			
HIGH	49.99 (8.83)			
VERY HIGH	50.72 (6.83)			
SF-36MHS DELTA				
Subgroup	Mean (SD)	F	df	p
NO	-5.24 (10.49)	1.018	4, 191	.399
LOW	-6.24 (10.54)			
MEDIUM	-11.18 (15.90)			
HIGH	-8.45 (11.37)			
VERY HIGH	-10.61 (12.76)			

Table 32 (cont.)

SF-36PHS				
Subgroup	Mean (SD)	F	df	p
NO	36.68 (6.97)	1.780	4, 208	.134
LOW	34.34 (6.26)			
MEDIUM	34.43 (3.72)			
HIGH	33.37 (4.91)			
VERY HIGH	34.10 (4.00)			
SF-36PHS DELTA				
Subgroup	Mean (SD)	F	df	p
NO	-4.64 (8.00)	1.319	4, 191	.264
LOW	-5.64 (7.60)			
MEDIUM	-0.80 (8.87)			
HIGH	-1.54 (6.48)			
VERY HIGH	-1.04 (7.10)			

Table 33

Chi-square Analysis of Post-treatment BDI Categories: Opioid Subgroups (Completers Only)

Post-treatment BDI					
Subgroup	n	%	χ^2	df	p (linear)
NO	476				
Absence of Depression (<10)		67.4	12.407	1	<.001
Mild to Moderate (10-18)		23.1			
Moderate to Severe (19-29)		8.0			
Severe (>29)		1.5			
LOW	177				
Absence of Depression (<10)		60.5			
Mild to Moderate (10-18)		26.6			
Moderate to Severe (19-29)		10.7			
Severe (>29)		2.3			
MEDIUM	62				
Absence of Depression (<10)		54.8			
Mild to Moderate (10-18)		25.8			
Moderate to Severe (19-29)		19.4			
Severe (>29)		0.0			
HIGH	44				
Absence of Depression (<10)		59.1			
Mild to Moderate (10-18)		25.0			
Moderate to Severe (19-29)		11.4			
Severe (>29)		4.5			

Table 33 (cont.)

VERY HIGH	33	
Absence of Depression (<10)		54.5
Mild to Moderate (10-18)		18.2
Moderate to Severe (19-29)		18.2
Severe (>29)		9.1

Table 34

Chi-square Analysis of Post-treatment HAM-D Categories: Opioid Subgroups (Completers Only)

Post-treatment HAM-D					
Subgroup	n	%	χ^2	df	p (linear)
NO	475				
None to Minimal (<12)		79.2	23.095	1	<.001
Mild to Moderate (12-20)		20.0			
Moderate to Severe (21-29)		0.8			
Severe (30+)		0.0			
LOW	177				
None to Minimal (<12)		63.8			
Mild to Moderate (12-20)		33.3			
Moderate to Severe (21-29)		2.8			
Severe (30+)		0.0			
MEDIUM	62				
None to Minimal (<12)		64.5			
Mild to Moderate (12-20)		32.3			
Moderate to Severe (21-29)		3.2			
Severe (30+)		0.0			
HIGH	44				
None to Minimal (<12)		63.6			
Mild to Moderate (12-20)		34.1			
Moderate to Severe (21-29)		2.3			
Severe (30+)		0.0			

Table 34 (cont.)

VERY HIGH	32	
None to Minimal (<12)		56.3
Mild to Moderate (12-20)		34.4
Moderate to Severe (21-29)		9.4
Severe (30+)		0.0

Table 35

Chi-square Analysis of Post-treatment MVAS Categories: Opioid Subgroups (Completers Only)

Post-treatment MVAS					
Subgroup	n	%	χ^2	df	p (linear)
NO	478				
Mildly Disabling (≤40)		27.0	14.943	1	<.001
Moderately Disabling (41-70)		39.1			
Severely Disabling (71-100)		26.2			
Very Severely Disabling (101-130)		7.1			
Extremely Disabling (≥131)		0.6			
LOW	177				
Mildly Disabling (≤40)		15.8			
Moderately Disabling (41-70)		30.5			
Severely Disabling (71-100)		37.3			
Very Severely Disabling (101-130)		16.4			
Extremely Disabling (≥131)		0.0			
MEDIUM	62				
Mildly Disabling (≤40)		17.7			
Moderately Disabling (41-70)		30.6			
Severely Disabling (71-100)		38.7			
Very Severely Disabling (101-130)		11.3			
Extremely Disabling (≥131)		1.6			

Table 35 (cont.)

HIGH	44	
Mildly Disabling (≤40)		22.7
Moderately Disabling (41-70)		29.5
Severely Disabling (71-100)		29.5
Very Severely Disabling (101-130)		18.2
Extremely Disabling (≥131)		0.0
VERY HIGH	33	
Mildly Disabling (≤40)		12.1
Moderately Disabling (41-70)		45.5
Severely Disabling (71-100)		24.2
Very Severely Disabling (101-130)		15.2
Extremely Disabling (≥131)		3.0

Table 36

Chi-square Analysis of Post-treatment MVAS Extremes: Opioid Subgroups (Completers Only)

Post-treatment MVAS					
Subgroup	n	%	χ^2	df	p
NO	478				
Not Extreme (0-70)		66.1	25.499	4	<.001
Extreme (71-150)		33.9			
LOW	177				
Not Extreme (0-70)		46.3			
Extreme (71-150)		53.7			
MEDIUM	62				
Not Extreme (0-70)		48.4			
Extreme (71-150)		51.6			
HIGH	44				
Not Extreme (0-70)		52.3			
Extreme (71-150)		47.7			
VERY HIGH	33				
Not Extreme (0-70)		57.6			
Extreme (71-150)		42.4			

Table 37

Chi-square Analysis of Post-treatment OSW Categories: Opioid Subgroups (Completers Only)

Post-treatment OSW					
Group	n	%	χ^2	df	p (linear)
NO	116				
Minimal Disability (0-20)		54.3	1.197	1	.274
Moderate Disability (21-40)		45.7			
Severe Disability (41-60)		0.0			
Crippling Disability (61-80)		0.0			
Bedbound or Exaggerated Disability (81-100)		0.0			
LOW	34				
Minimal Disability (0-20)		35.3			
Moderate Disability (21-40)		64.7			
Severe Disability (41-60)		0.0			
Crippling Disability (61-80)		0.0			
Bedbound or Exaggerated Disability (81-100)		0.0			
MEDIUM	8				
Minimal Disability (0-20)		62.5			
Moderate Disability (21-40)		37.5			
Severe Disability (41-60)		0.0			
Crippling Disability (61-80)		0.0			
Bedbound or Exaggerated Disability (81-100)		0.0			

Table 37 (cont.)

HIGH	10	
Minimal Disability (0-20)		60.0
Moderate Disability (21-40)		40.0
Severe Disability (41-60)		0.0
Crippling Disability (61-80)		0.0
Bedbound or Exaggerated Disability (81-100)		0.0
VERY HIGH	8	
Minimal Disability (0-20)		25.0
Moderate Disability (21-40)		75.0
Severe Disability (41-60)		0.0
Crippling Disability (61-80)		0.0
Bedbound or Exaggerated Disability (81-100)		0.0

Table 38

Statistical Analyses of MMPI Validity and Clinical Scale Scores: Opioid Groups

Scale Mean (SD)	NO	YES	F	df	p*
N=768 n (% of Total)	398 (51.8)	370 (48.2)			
L	59.94 (10.21)	59.40 (10.24)	.536	1, 764	.464
F	58.63 (15.45)	60.41 (16.16)	2.413	1, 764	.121
K	51.02 (10.73)	49.30 (11.05)	4.820	1, 766	.028
1	72.19 (11.84)	76.38 (10.50)	26.809	1, 766	<.001
2	68.02 (13.89)	71.77 (14.26)	13.661	1, 766	<.001
3	70.83 (13.43)	74.67 (14.25)	14.779	1, 766	<.001
4	58.42 (11.34)	60.16 (12.85)	3.959	1, 766	.047
5	50.08 (10.39)	50.59 (10.07)	.468	1, 766	.494
6	56.74 (13.67)	58.23 (15.41)	2.005	1, 766	.157
7	61.07 (13.18)	64.02 (13.88)	9.169	1, 766	.003
8	62.03 (14.13)	65.10 (14.80)	8.688	1, 766	.003
9	51.97 (10.27)	53.42 (11.22)	3.490	1, 766	.062
0	53.88 (11.47)	54.66 (11.93)	.871	1, 766	.351

* alpha = .005

Table 39

Chi-square Analyses of MMPI Clinical Scale Elevations: Opioid Groups

Scale % with Elevations	NO	YES	χ^2	df	p	OR	CI (95%)
N=768 n	398	370					
1	78.6	88.1	12.291	1	<.000	2.012	1.355, 2.989
2	55.8	61.9	2.956	1	.086	1.288	.965, 1.718
3	68.8	77.6	7.411	1	.006	1.565	1.132, 2.163
4	27.1	32.7	2.840	1	.092	1.305	.957, 1.779
5	10.3	11.9	.493	1	.483	1.175	.748, 1.845
6	23.6	29.5	3.365	1	.067	1.351	.979, 1.863
7	35.7	47.3	10.679	1	.001	1.618	1.211, 2.161
8	40.7	45.4	1.730	1	.188	1.212	.910, 1.613
9	13.3	16.8	1.782	1	.182	1.310	.881, 1.950
0	20.4	22.2	.376	1	.540	1.114	.788, 1.575

Table 40

Chi-Square Analysis of MMPI Clusters: Opioid Groups

	NO		YES				
Cluster	n	%*	n	%*	χ^2	df	p
Defended	144	36.4	102	27.7	13.26	3	.004
Distressed	31	7.8	55	14.9			
Somatoform	114	28.8	113	30.7			
Depressed	107	27.0	98	26.6			

* of 764 classifiable patients

Table 41

Chi-Square Analysis of Four MMPI Profiles: Opioid Groups

	NO		YES				
Profile	n	%*	n	%*	χ^2	df	p
Normal	26	8.9	15	5.3	8.195	3	.042
Conversion V	52	17.8	36	12.6			
Neurotic Triad	28	9.6	22	7.7			
Disability Profile	186	63.7	212	74.4			

*of 577 classifiable patients

Table 42

Statistical Analyses of WAIS-R Index Scores: Opioid Groups

Index Mean (SD)	NO	YES	F	df	p
N=1030 n=	536	494			
V-IQ	86.75 (13.93)	85.95 (12.31)	.930	1, 1028	.335
P-IQ	87.29 (15.26)	86.02 (11.35)	2.267	1, 1027	.076
FSIQ	86.53 (13.99)	85.09 (11.70)	3.165	1, 1026	.076

Table 43

Statistical Analyses of MMPI Validity and Clinical Scale Scores: Opioid Subgroups

Scale Mean (SD)	NO	LOW	MED	HIGH	VERY HIGH	F	df	p*
N=684 n=	397	148	57	47	35			
L	58.89 (10.18)	59.51 (9.76)	60.18 (11.78)	60.68 (10.59)	57.31 (10.26)	.653	4, 677	.625
F	58.68 (15.45)	58.73 (15.52)	61.72 (17.74)	64.28 (18.27)	65.91 (18.35)	3.002	4, 677	.018
K	50.99 (10.73)	49.41 (10.97)	50.44 (10.46)	49.74 (11.88)	45.23 (10.77)	2.599	4, 679	.035
1	72.22 (11.84)	75.24 (9.81)	75.77 (10.24)	78.87 (10.72)	80.17 (11.61)	8.179	4, 679	<.001
2	68.08 (13.85)	70.72 (13.45)	71.07 (14.81)	75.02 (15.84)	79.54 (14.60)	7.590	4, 679	<.001
3	70.88 (13.41)	72.58 (13.57)	74.58 (14.93)	77.57 (12.54)	80.71 (15.48)	6.539	4, 679	<.001
4	58.43 (11.36)	58.45 (12.13)	61.42 (13.71)	62.87 (13.22)	66.51 (13.95)	5.348	4, 679	<.001
5	50.08 (10.41)	50.36 (11.03)	49.61 (10.71)	50.21 (8.24)	51.14 (8.58)	.140	4, 679	.967
6	56.79 (13.67)	56.96 (15.14)	57.91 (14.87)	60.89 (14.75)	66.17 (16.83)	4.103	4, 679	.003
7	61.09 (13.18)	62.68 (12.64)	62.33 (16.06)	67.83 (13.61)	69.60 (16.24)	5.318	4, 679	<.001
8	62.05 (14.15)	62.19 (13.06)	64.86 (16.64)	68.96 (16.19)	73.00 (18.05)	6.548	4, 679	<.001
9	51.97 (10.29)	52.32 (10.73)	53.93 (11.53)	55.19 (11.99)	52.86 (11.10)	1.269	4, 679	.281
0	53.92 (11.44)	54.15 (10.74)	53.42 (12.17)	55.81 (12.46)	62.37 (14.09)	4.601	4, 679	.001

* alpha = .005

Table 44

Chi-square Analyses of MMPI Clinical Scale Elevations: Opioid Subgroups

Scale % with Elevation	NO	LOW	MED	HIGH	VERY HIGH	χ^2	df	p
N=684 n=	397	148	57	47	35			
1	78.8	87.7	86.0	91.5	91.4	11.776	4	.019
2	55.9	60.1	61.4	68.1	80.1	9.747	4	.045
3	69.0	77.7	73.7	83.0	88.6	11.595	4	.021
4	27.2	26.4	36.8	38.3	60.0	20.105	4	<.001
5	10.3	15.5	8.8	6.4	5.7	5.670	4	.225
6	23.7	28.4	26.3	36.2	45.7	10.625	4	.031
7	35.8	44.6	42.1	59.6	57.1	15.792	4	.003
8	40.8	40.5	40.4	57.4	68.6	14.604	4	.006
9	13.4	14.2	17.5	25.5	11.4	5.694	4	.223
0	20.4	18.9	21.1	25.5	48.6	16.216	4	.003

Table 45

Chi-Square Analysis of MMPI Clusters: Opioid Subgroups

Cluster %*	NO	LOW	MED	HIGH	VERY HIGH	χ^2	df	p
n=	395	148	56	47	34			
Defended	36.2	27.7	33.9	17.0	17.6	41.331	12	<.001
Distressed	7.8	9.5	17.9	23.4	35.3			
Somato-form	28.9	33.1	25.0	27.7	20.6			
Depressed	27.1	29.7	23.2	31.9	26.5			

* of 680 classifiable patients

Table 46

Chi-Square Analysis of Four MMPI Profiles: Opioid Subgroups

Profile %*	NO	LOW	MED	HIGH	VERY HIGH	χ^2	df	p (linear)
n=	292	115	41	41	30			
Normal	8.9	6.1	7.3	4.9	6.7	4.278	1	.039
Conversion V	17.8	12.2	17.1	14.6	3.3			
Neurotic Triad	9.6	7.8	9.8	7.3	6.7			
Disability Profile	63.7	73.9	65.9	73.2	83.3			

* of 519 classifiable patients

Table 47

Statistical Analyses of WAIS-R Index Scores: Opioid Subgroups

Index	NO	LOW	MED	HIGH	VERY HIGH	F	df	p
N=923								
n=	535	210	79	58	41			
V-IQ	86.71 (13.92)	84.80 (11.27)	85.01 (15.50)	86.43 (12.16)	88.29 (10.10)	1.205	4, 918	.307
P-IQ	87.25 (15.25)	85.40 (11.91)	86.49 (12.56)	86.29 (9.92)	83.93 (7.58)			
FSIQ	86.49 (13.98)	84.10 (11.68)	84.89 (13.37)	85.38 (10.57)	85.37 (8.94)			

Table 48

Pre-treatment Physical Variables: Opioid Groups

Variable Mean (SD)	NO	YES
N=1226 n (% of Total)	630 (51.39)	596 (48.61)
Cumulative Physical Score	45.88 (17.69)	37.53 (17.44)
QPD-Trunk	8.33 (7.83)	11.29 (9.31)
QPD-Extremity	11.20 (14.10)	15.14 (17.86)
Pain Intensity	6.27 (1.96)	6.61 (1.69)

Table 49

Statistical Analyses of Pre-treatment Physical Variables: Opioid Groups

Cumulative Physical Score				
Group	Mean (SD)	F	df	p
NO	45.88 (17.69)	68.02	1, 1202	<.001
YES	37.53 (17.44)			
QPD-Trunk				
Group	Mean (SD)	F	df	p
NO	8.33 (7.83)	29.93	1, 1004	<.001
YES	11.29 (9.31)			
QPD-Extremity				
Group	Mean (SD)	F	df	p
NO	11.20 (14.10)	15.18	1, 1004	<.001
YES	15.14 (17.86)			
Pain Intensity				
Group	Mean (SD)	F	df	p
NO	6.27 (1.96)	8.75	1, 1008	.003
YES	6.61 (1.69)			

Table 50

Chi-square Analysis of Pre-treatment Pain Intensity Categories: Opioid Groups

Pain Intensity Group	n	%	χ^2	df	p
NO	530				
Not Extreme (0-7)		69.6	2.903	1	.088
Extreme (8-10)		30.4			
YES	480				
Not Extreme (0-7)		64.6			
Extreme (8-10)		35.4			

Table 51

Post-treatment Physical Variables: Opioid Groups (Completers Only)

Variable Mean (SD)	NO	YES
N=954 n (% of Total)	513 (53.77)	441 (46.23)
Cumulative Physical Score	79.09 (14.49)	76.82 (15.59)
Cumulative Score Delta	-31.74 (19.93)	-37.84 (20.09)
QPD-Trunk	7.02 (7.68)	9.04 (8.46)
QPD-Trunk Delta	1.18 (5.45)	1.86 (6.66)
QPD-Extremity	8.98 (11.25)	10.72 (13.49)
QPD-Extremity Delta	2.18 (8.76)	3.70 (14.48)
Pain Intensity	4.38 (2.13)	4.91 (2.14)
Pain Intensity Delta	1.87 (2.19)	1.59 (2.19)

Table 52

Statistical Analyses of Post-treatment Physical Variables: Opioid Groups (Completers Only)

Cumulative Physical Score				
Group	Mean (SD)	F*	df	p
NO	79.09 (14.49)	0.244	1, 878	.622
YES	76.82 (15.59)			
Cumulative Score Delta				
Group	Mean (SD)	F	df	p
NO	-31.74 (19.93)	20.33	1, 879	<.001
YES	-37.84 (20.09)			
QPD-Trunk				
Group	Mean (SD)	F	df	p
NO	7.02 (7.68)	13.84	1, 877	<.001
YES	9.04 (8.46)			
QPD-Trunk Delta				
Group	Mean (SD)	F	df	p
NO	1.18 (5.45)	2.59	1, 815	.108
YES	1.86 (6.66)			

*ANCOVA

Table 52 (cont.)

QPD-Extremity				
Group	Mean (SD)	F	df	p
NO	8.98 (11.25)	4.33	1, 876	.038
YES	10.72 (13.49)			
QPD-Extremity Delta				
Group	Mean (SD)	F	df	p
NO	2.18 (8.76)	3.40	1, 814	.065
YES	3.70 (14.48)			
Pain Intensity				
Group	Mean (SD)	F*	df	p
NO	4.38 (2.13)	13.26	1, 882	<.001
YES	4.91 (2.14)			
Pain Intensity Delta				
Group	Mean (SD)	F	df	p
NO	1.87 (2.19)	3.24	1, 815	.072
YES	1.59 (2.19)			

*ANCOVA

Table 53

Chi-square Analysis of Post-treatment Pain Intensity Categories: Opioid Groups (Completers Only)

Pain Intensity					
Group	n	%	χ^2	df	p
NO	478				
Not Extreme (0-7)		91.0	3.326	1	.068
Extreme (8-10)		9.0			
YES	406				
Not Extreme (0-7)		87.2			
Extreme (8-10)		12.8			

Table 54

Pre-treatment Physical Variables: Opioid Subgroups

Variable Mean (SD)	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=1146					
n	630	267	112	78	59
(% of Total)	(54.97)	(23.30)	(9.77)	(6.81)	(5.15)
Cumulative Physical Score	45.86 (17.71)	38.31 (16.84)	34.31 (18.00)	34.28 (15.72)	35.96 (17.08)
QPD-Trunk	8.34 (7.83)	10.70 (9.23)	12.34 (9.83)	10.34 (6.76)	12.39 (9.81)
QPD-Extremity	11.22 (14.10)	14.12 (17.22)	15.02 (19.70)	15.75 (16.97)	20.76 (18.13)
Pain Intensity	6.26 (1.96)	6.52 (1.73)	6.69 (1.57)	6.86 (1.63)	7.00 (1.61)

Table 55

Statistical Analyses of Pre-treatment Physical Variables: Opioid Subgroups

Cumulative Physical Score				
Subgroup	Mean (SD)	F	df	p
NO	45.86 (17.71)	20.10	4, 1079	<.001
LOW	38.31 (16.84)			
MEDIUM	34.31 (18.00)			
HIGH	34.28 (15.71)			
VERY HIGH	35.96 (17.08)			

QPD-Trunk				
Subgroup	Mean (SD)	F	df	p
NO	8.34 (7.83)	7.31	4, 900	<.001
LOW	10.70 (9.23)			
MEDIUM	12.34 (9.83)			
HIGH	10.34 (6.76)			
VERY HIGH	12.39 (9.81)			

Table 55 (cont.)

QPD-Extremity				
Subgroup	Mean (SD)	F	df	p
NO	11.22 (14.10)	5.19	4, 900	<.001
LOW	14.12 (17.22)			
MEDIUM	15.02 (19.70)			
HIGH	15.75 (16.97)			
VERY HIGH	20.76 (18.13)			
Pain Intensity	Mean (SD)	F	df	p
NO	6.26 (1.96)	3.45	4, 903	.008
LOW	6.52 (1.73)			
MEDIUM	6.69 (1.57)			
HIGH	6.86 (1.63)			
VERY HIGH	7.00 (1.61)			

Table 56

Chi-square Analysis of Pre-treatment Pain Intensity Categories: Opioid Subgroups

Pain Intensity Group	n	%	χ^2	df	p
NO	530				
Not Extreme (0-7)		69.6	4.515	4	.341
Extreme (8-10)		30.4			
LOW	193				
Not Extreme (0-7)		65.3			
Extreme (8-10)		34.7			
MEDIUM	86				
Not Extreme (0-7)		64.0			
Extreme (8-10)		36.0			
HIGH	59				
Not Extreme (0-7)		59.3			
Extreme (8-10)		40.7			
VERY HIGH	41				
Not Extreme (0-7)		61.0			
Extreme (8-10)		39.0			

Table 57

Post-treatment Physical Variables: Opioid Subgroups (Completers Only)

Variable Mean (SD)	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=887					
n=	513	205	75	53	41
(% of Total)	(57.84)	(23.11)	(8.46)	(5.98)	(4.62)
Cumulative Physical Score	79.07 (14.49)	77.25 (16.94)	74.00 (15.35)	77.40 (12.94)	72.82 (12.86)
Cumulative Physical Score Delta	-31.83 (19.87)	-38.24 (21.04)	-37.40 (20.52)	-40.00 (16.41)	-37.50 (19.87)
QPD-Trunk	7.03 (7.68)	9.24 (8.10)	8.73 (8.28)	8.16 (6.78)	8.39 (8.60)
QPD-Trunk Delta	1.19 (5.46)	1.43 (5.86)	2.90 (7.46)	2.32 (6.35)	2.97 (7.37)
QPD-Extremity	9.00 (11.25)	10.82 (13.14)	9.44 (11.52)	11.23 (13.48)	13.00 (16.78)
QPD-Extremity Delta	2.18 (8.77)	2.78 (14.35)	3.84 (14.58)	5.27 (14.16)	5.03 (14.32)
Pain Intensity	4.39 (2.13)	5.07 (2.14)	4.81 (1.99)	4.84 (2.20)	4.94 (2.15)
Pain Intensity Delta	1.87 (2.19)	1.37 (2.24)	1.65 (2.10)	2.02 (2.19)	2.00 (2.33)

Table 58

Statistical Analyses of Post-treatment Physical Variables: Opioid Subgroups (Completers Only)

Cumulative Physical Score				
Subgroup	Mean (SD)	F*	df	p
NO	79.07 (14.49)	1.005	4, 786	.404
LOW	77.25 (16.94)			
MEDIUM	74.00 (15.35)			
HIGH	77.40 (12.94)			
VERY HIGH	72.82 (12.86)			

Cumulative Physical Delta				
Subgroup	Mean (SD)	F	df	p
NO	-31.83 (19.87)	5.03	4, 787	.001
LOW	-38.24 (21.04)			
MEDIUM	-37.40 (20.52)			
HIGH	-40.00 (16.41)			
VERY HIGH	-37.50 (19.87)			

*ANCOVA

Table 58 (cont.)

QPD-Trunk				
Subgroup	Mean (SD)	F*	df	p
NO	7.03 (7.68)	0.567	4, 731	.687
LOW	9.24 (8.10)			
MEDIUM	8.73 (8.28)			
HIGH	8.16 (6.78)			
VERY HIGH	8.39 (8.60)			

QPD-Trunk Delta				
Subgroup	Mean (SD)	F	df	p
NO	1.19 (5.46)	1.83	4, 731	.121
LOW	1.43 (5.86)			
MEDIUM	2.90 (7.46)			
HIGH	2.32 (6.35)			
VERY HIGH	2.97 (7.37)			

*ANCOVA

Table 58 (cont.)

QPD-Extremity				
Subgroup	Mean (SD)	F	df	p
NO	9.00 (11.25)	1.57	4, 785	.181
LOW	10.82 (13.14)			
MEDIUM	9.44 (11.52)			
HIGH	11.23 (13.48)			
VERY HIGH	13.00 (16.78)			
QPD-Extremity Delta				
Subgroup	Mean (SD)	F	df	p
NO	2.18 (8.77)	1.28	4, 730	.277
LOW	2.78 (14.35)			
MEDIUM	3.84 (14.58)			
HIGH	5.27 (14.16)			
VERY HIGH	5.03 (14.32)			

*ANCOVA

Table 58 (cont.)

Pain Intensity				
Subgroup	Mean (SD)	F*	df	p
NO	4.39 (2.13)	2.934	4, 731	.020
LOW	5.07 (2.14)			
MEDIUM	4.81 (1.99)			
HIGH	4.84 (2.20)			
VERY HIGH	4.94 (2.15)			

Pain Intensity Delta				
Subgroup	Mean (SD)	F	df	p
NO	1.87 (2.19)	1.79	4, 731	.130
LOW	1.37 (2.24)			
MEDIUM	1.65 (2.10)			
HIGH	2.02 (2.19)			
VERY HIGH	2.00 (2.33)			

*ANCOVA

Table 59

Chi-square Analysis of Post-treatment Pain Intensity Categories: Opioid Subgroups (Completers Only)

Pain Intensity Group	n	%	χ^2	df	p
NO	478				
Not Extreme (0-7)		91.0	4.040	4	.401
Extreme (8-10)		9.0			
LOW	177				
Not Extreme (0-7)		87.6			
Extreme (8-10)		12.4			
MEDIUM	62				
Not Extreme (0-7)		90.3			
Extreme (8-10)		9.7			
HIGH	44				
Not Extreme (0-7)		84.1			
Extreme (8-10)		15.9			
VERY HIGH	33				
Not Extreme (0-7)		84.8			
Extreme (8-10)		15.2			

Table 60

One-year Socioeconomic Outcomes: Opioid Groups (Completers Only)

Variable	NO	YES
N=954 n (% of Total)	513 (53.77)	441 (46.23)
Work Return (%)	93.7	88.1
Work Retention (%)	85.3	68.8
40+ hours/week (%)	77.8	74.8
New Surgery to the Original Site of Injury (%)	2.1	4.4
Seeking Treatment from a New Provider (%)	14.0	29.9
Recurrent Injury to the Same Body Part (%)	4.4	6.8
Case Settlement (%)	97.2	98.0
SSDI or SSI (%)	1.9	5.2

Table 61

Statistical Analyses of Socioeconomic Outcomes: Opioid Groups (Completers Only)

Work Return						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	93.7	8.045	1	.005	2.00	1.23, 3.25
YES	88.1					
Work Retention						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	85.3	32.094	1	.000	2.62	1.87, 2.62
YES	68.8					
40+ Hours/Week						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	77.8	.868	1	.352	1.19	0.83, 1.70
YES	74.8					
Surgery to the Same Body Part						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	2.1	3.205	1	.073	2.18	.911, 5.20
YES	4.4					

Table 61 (cont.)

Seeking Treatment from a New Provider						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	14.0	27.420	1	.000	2.63	1.82, 3.81
YES	29.9					
New Injury to the Same Body Part						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	4.4	1.775	1	.183	1.57	0.81, 3.07
YES	6.8					
Case Settlement						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	97.2	.485	1	.486	1.04	0.54, 3.66
YES	98.0					
SSDI or SSI						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	1.9	6.309	1	.012	2.81	1.21, 6.49
YES	5.2					

Table 62

One-year Socioeconomic Outcomes: Opioid Subgroups (Completers Only)

Variable	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=887					
n	513	205	75	53	41
(% of Total)	(57.84)	(23.11)	(8.46)	(5.98)	(4.62)
Work Return (%)	93.7	88.7	89.5	90.7	75.9
Work Retention (%)	85.2	70.1	63.0	69.0	55.2
40+ hours/week (%)	77.8	68.9	76.2	78.1	83.3
Surgery to Same Body Part (%)	2.1	5.5	2.1	7.7	7.4
Seeking Treatment from a New Provider (%)	14.0	28.8	36.7	28.2	29.6
New Injury to the Same Body Part (%)	4.4	3.8	13.0	6.3	4.2
Case Settlement (%)	97.2	98.6	98.0	95.0	100.0
SSDI or SSI (%)	1.9	5.7	3.9	4.5	18.5

Table 63

Statistical Analyses of Socioeconomic Outcomes: Opioid Subgroups (Completers Only)

Work Return				
Subgroup	Percent	χ^2	df	p
NO	93.7	13.816	4	.008
LOW	88.7			
MEDIUM	89.5			
HIGH	90.7			
VERY HIGH	75.9			
Work Retention				
Subgroup	Percent	χ^2	df	p
NO	85.2	37.748	4	<.001
LOW	70.1			
MEDIUM	63.0			
HIGH	69.0			
VERY HIGH	55.2			

Table 63 (cont.)

40+ Hours/Week				
Subgroup	Percent	χ^2	df	p
NO	77.8	5.166	4	.271
LOW	68.9			
MEDIUM	76.2			
HIGH	78.1			
VERY HIGH	83.3			
Surgery to the Same Body Part				
Subgroup	Percent	χ^2	df	p
NO	2.1	7.719	4	.102
LOW	5.5			
MEDIUM	2.1			
HIGH	7.7			
VERY HIGH	7.4			
Seeking Treatment from a New Provider				
Subgroup	Percent	χ^2	df	p
NO	14.0	26.831	4	<.001
LOW	28.8			
MEDIUM	36.7			
HIGH	28.2			
VERY HIGH	29.6			

Table 63 (cont.)

New Injury to the Same Body Part				
Subgroup	Percent	χ^2	df	p
NO	4.4	7.082	4	.132
LOW	3.8			
MEDIUM	13.0			
HIGH	6.3			
VERY HIGH	4.2			
Case Settlement				
Subgroup	Percent	χ^2	df	p
NO	97.2	2.750	4	.601
LOW	98.6			
MEDIUM	98.0			
HIGH	95.0			
VERY HIGH	100.0			
SSDI or SSI				
Subgroup	Percent	χ^2	df	p
NO	1.9	22.063	4	<.001
LOW	5.7			
MEDIUM	3.9			
HIGH	4.5			
VERY HIGH	18.5			

Table 64

One-year Socioeconomic Outcomes: Opioid Groups (Non-completers Only)

Variable	NO	YES
N=272 n (% of Total)	117 (43.0)	155 (57.0)
Work Return (%)	71.2	53.5
Work Retention (%)	58.9	44.2
40+ hours/week (%)	82.0	84.5
New Surgery to the Original Site of Injury (%)	10.1	12.8
Seeking Treatment from a New Provider (%)	34.6	49.5
Recurrent Injury to the Same Body Part (%)	21.7	9.3
Case Settlement (%)	97.1	94.8
SSDI or SSI (%)	3.5	14.5

Table 65

Statistical Analyses of Socioeconomic Outcomes: Opioid Groups (Non-completers Only)

Work Return						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	71.2	5.258	1	.022	2.16	1.11, 4.17
YES	53.5					
Work Retention						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	58.9	3.422	1	.064	1.81	0.96, 3.40
YES	44.2					
40+ Hours/Week						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	82.0	.119	1	.730	1.19	0.43, 3.29
YES	84.5					
Surgery to the Same Body Part						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	10.1	.292	1	.589	1.30	0.50, 3.36
YES	12.8					

Table 65 (cont.)

Seeking Treatment from a New Provider						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	34.6	3.903	1	.048	1.85	1.00, 3.42
YES	49.5					
New Injury to the Same Body Part						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	21.7	3.034	1	.082	2.72	0.86, 8.62
YES	9.3					
Case Settlement						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	97.1	.487	1	.485	1.84	0.33, 10.35
YES	94.8					
SSDI or SSI						
Group	Percent	χ^2	df	p	OR	CI (95%)
NO	3.5	4.289	1	.038	4.67	0.96, 22.63
YES	14.5					

Table 66

One-year Socioeconomic Outcomes: Opioid Subgroups (Non-completers Only)

Variable	NO	LOW	MEDIUM	HIGH	VERY HIGH
N=246					
n	117	60	31	23	15
(% of Total)	(47.6)	(24.4)	(12.6)	(9.3)	(6.1)
Work Return (%)	71.2	54.8	38.1	53.8	66.7
Work Retention (%)	58.9	54.8	28.6	53.8	33.3
40+ hours/week (%)	82.0	75.0	100.0	75.0	100.0
Surgery to Same Body Part (%)	10.1	20.6	0.0	8.3	0.0
Seeking Treatment from a New Provider (%)	34.6	54.3	45.8	46.2	25.0
New Injury to the Same Body Part (%)	21.7	9.5	21.4	0.0	0.0
Case Settlement (%)	97.1	93.1	89.5	100.0	100.0
SSDI or SSI (%)	3.5	13.0	12.5	11.1	20.0

Table 67

Statistical Analyses of Socioeconomic Outcomes: Opioid Subgroups (Non-completers Only)

Work Return				
Subgroup	Percent	χ^2	df	p
NO	71.2	8.708	4	.069
LOW	54.8			
MEDIUM	38.1			
HIGH	53.8			
VERY HIGH	66.7			
Work Retention				
Subgroup	Percent	χ^2	df	p
NO	58.9	6.968	4	.138
LOW	54.8			
MEDIUM	28.6			
HIGH	53.8			
VERY HIGH	33.3			

Table 67 (cont.)

40+ Hours/Week				
Subgroup	Percent	χ^2	df	p
NO	82.0	5.306	4	.257
LOW	75.0			
MEDIUM	100.0			
HIGH	75.0			
VERY HIGH	100.0			
Surgery to the Same Body Part				
Subgroup	Percent	χ^2	df	p
NO	10.1	7.536	4	.110
LOW	20.6			
MEDIUM	0.0			
HIGH	8.3			
VERY HIGH	0.0			
Seeking Treatment from a New Provider				
Subgroup	Percent	χ^2	df	p
NO	34.6	5.083	4	.279
LOW	54.3			
MEDIUM	45.8			
HIGH	46.2			
VERY HIGH	25.0			

Table 67 (cont.)

New Injury to the Same Body Part				
Subgroup	Percent	χ^2	df	p
NO	21.7	3.419	4	.490
LOW	9.5			
MEDIUM	21.4			
HIGH	0.0			
VERY HIGH	0.0			
Case Settlement				
Subgroup	Percent	χ^2	df	p
NO	97.1	3.081	4	.544
LOW	93.1			
MEDIUM	89.5			
HIGH	100.0			
VERY HIGH	100.0			
SSDI or SSI				
Subgroup	Percent	χ^2	df	p
NO	3.5	3.810	4	.432
LOW	13.0			
MEDIUM	12.5			
HIGH	11.1			
VERY HIGH	20.0			

Table 68

Extreme Group Comparisons of MMPI Scale Scores

Variable % OR CI (95%) p-value	Comparison Subgroup NO Subgroup n=398	HIGH n=47	VERY HIGH n=35	HIGH/ VERY HIGH n=82
Scale 1 Scores in the Normal Range	21.40	8.50 2.92 1.02-8.36 .037	8.60 2.90 0.87-9.69 .072	8.50 2.91 1.29-6.55 .007
Scale 2 Scores in the Normal Range	44.20	31.90 1.69 0.89-3.22 .107	20.00 3.171 1.35-7.43 .005	26.80 2.162 1.28-3.66 .004
Scale 3 Scores in the Normal Range	31.20	17.00 2.21 1.00-4.86 .045	11.40 3.51 1.21-10.15 .014	14.60 2.64 1.38-5.05 .003
Scale 4 Score Elevations	27.10	38.30 1.67 .89-3.12 .108	60.00 4.03 1.98-8.20 .000	47.60 2.44 1.50-3.96 .000
Scale 6 Score Elevations	23.60	36.20 1.83 0.97-3.47 .060	45.70 2.72 1.35-5.51 .004	40.20 2.18 1.32-3.59 .002
Scale 7 Score Elevations	35.70	59.60 2.66 1.43-4.927 .004	57.10 2.40 1.19-4.84 .012	58.50 2.55 1.57-4.13 .000
Scale 8 Score Elevations	40.70	57.40 1.97 1.07-3.63 .028	68.60 3.18 1.52-6.67 .001	62.20 2.40 1.47-3.91 .000
Scale 9 Score Elevations	13.30	25.50 2.23 1.09-4.57 .025	11.40 0.840 0.29-2.48 .751	19.50 1.58 0.85-2.93 .145
Scale 0 Score Elevations	20.40	25.50 1.34 0.67-2.70 .409	48.60 3.70 1.82-7.49 .000	35.40 2.14 1.28-3.58 .003

Table 69

Pre-treatment Opioid Medications

Variable	LOW	MED	HIGH	VERY HIGH	LOW through VERY HIGH Combined
n	250	99	72	51	472
Daily Morphine Units					
Mean (SD)	17.65	45.51	85.77	205.31	54.16
Median	16.67	45.00	90.00	180.00	30.00
Mode	22.50	45.00	90.00	138.46	22.5
Minimum	0.09	32.73	61.15	120.81	0.09
Maximum	30.00	60.00	120.00	390.00	390.00
Taking ≥ 2 Opioid Medications n (%)	11 (4.40)	11 (11.11)	19 (26.39)	25 (49.02)	66 (13.98)

Table 70

Frequency of Pre-treatment Opioid Medications

Variable	LOW	MED	HIGH	VERY HIGH	LOW through VERY HIGH Combined
n	250	99	72	51	472
hydrocodone	92	35	21	14	162
Vicodin	61	20	12	8	101
Darvocet	19	14	25	22	80
Lortab	35	16	16	9	76
Tylenol w/ Codeine	19	6	4	0	29
oxycodone	3	4	1	15	23
Norco	6	4	6	6	22
Vicoprofen	5	5	1	2	13
pentazocine	5	4	1	0	10
Other	16	3	4	10	33

Table 71

Frequency of Pre-treatment Non-opioid Analgesics

Variable	NO	LOW	MED	HIGH	VERY HIGH	LOW through VERY HIGH Combined
n	628	250	99	72	51	472
Celebrex	141	60	22	15	12	109
rofecoxib	86	43	12	3	9	67
ibuprofen	141	41	13	8	4	66
naproxen	103	33	13	8	4	58
arthrotec	23	8	3	2	2	15
Daypro (oraprozin)	56	20	4	10	4	38
Lodine (etodolac)	58	27	8	4	6	45
Relafen (meclofenamate)	83	41	14	15	4	74
acetaminophen	62	25	8	2	3	38
Ultram (tramadol)	66	9	4	4	4	21
Other	91	27	12	15	6	60

Table 72

Frequency of Pre-treatment Muscle Relaxants

Variable	NO	LOW	MED	HIGH	VERY HIGH	LOW through VERY HIGH Combined
n	628	250	99	72	51	472
Soma (carisoprodol)	28	37	21	14	6	78
Flexeril (cyclobenzaprine)	28	37	15	8	5	65
Valium (diazepam)	8	11	6	4	4	25
Robaxin (methocarbamol)	7	9	3	2	4	18
Zanaflex (tizanidine)	10	11	3	0	4	18
Skelaxin (metaxalone)	26	9	4	3	1	17
Other	4	4	3	2	2	11

APPENDIX C
IRB Approval

UT SOUTHWESTERN
MEDICAL CENTER

Institutional Review Board

TO: Cindy Kidner
Psychiatry - 9149

FROM: George Buchanan, MD
Institutional Review Board 2 Chairperson
IRB - 8843

DATE: March 9, 2007

RE: **Expedited Approval of Protocol and HIPAA Waiver**
IRB Number: 022007-025
Title: Pre-treatment Level of Opioid Use as a Predictor of Chronic Pain Rehabilitation Outcome

An IRB Chair has approved this protocol on March 7, 2007 via the expedited review procedure in accordance with 45 CFR 46.110(a)-(b)(1), 63 FR 60364, and 63 FR 60353. IRB approval of this research lasts until February 20, 2008. If the research continues beyond twelve months, you must apply for updated approval of the protocol one month before the date of expiration noted above. **The use of consent form is waived in accordance with 45 CFR 46.116(d). Your approved subject sample size is 1400 subjects.**

The IRB requires that you report to the Board any unexpected adverse events that occur during the study. In the future, if you require a modification to the protocol, obtain review and approval by the Board prior to implementing any changes except when necessary to eliminate apparent immediate hazards to a subject.

If you have any questions related to this approval or the IRB, you may telephone Kathryn Jacobson at 214.648.3696.

Enc: HIPAA Waiver
Project Summary
NR1-EXP copy

GB/as

5323 Harry Hines Boulevard, C1.206 / Dallas, Texas 75390-8843 / 214-648-3060 Fax 214-648-2171
www.utsouthwestern.edu

APPENDIX D
Materials

Case Number:	**123456789**	**Episode:**	**1**
Last Name:	**Smith**	**Discharge Date:**	**15-Feb-02**
First Name:	**John**	**Completer Status:**	**1**

Medication		Face Sheet-Initial	Face Sheet-Adm	Face Sheet-DC	Oral Med-Adm	Oral Med-DC	1st MD Note	Psych MD Note	Psych Eval

BECK INVENTORY

Name_____ Date_____

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. **Be sure to read all the statements in each group before making your choice.**

1 0 I do not feel sad.
1 I feel sad.
2 I am sad all the time and I can't snap out of it.
3 I am so sad or unhappy that I can't stand it.

2 0 I am not particularly discouraged about the future.
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to.
3 I feel that the future is hopeless and that things cannot improve.

3 0 I do not feel like a failure.
1 I feel I have failed more than the average person.
2 As I look back on my life, all I can see is a lot of failures.
3 I feel I am a complete failure as a person.

4 0 I get as much satisfaction out of things as I used to.
1 I don't enjoy things the way I used to.
2 I don't get real satisfaction out of anything anymore.
3 I am dissatisfied or bored with everything.

5 0 I don't feel particularly guilty.
1 I feel guilty a good part of the time.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6 0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7 0 I don't feel disappointed in myself.
1 I am disappointed in myself.
2 I am disgusted with myself.
3 I hate myself.

8 0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weaknesses or mistakes.
2 I blame myself all the time for my faults.
3 I blame myself for everything bad that happens.

9 0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10 0 I don't cry any more than usual.
1 I cry more now than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.

11 0 I am no more irritated now than I ever am.
1 I get annoyed or irritated more easily than I used to.
2 I feel irritated all the time now.
3 I don't get irritated at all by the things that used to irritate me.

12 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.

14 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds. I am purposely trying to lose we
2 I have lost more than 10 pounds. by eating less. Yes_____ No__
3 I have lost more than 15 pounds.

20 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think about anything else.

21 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION (HAM-D)

PATIENT NAME: _____

INTERVIEWER'S NAME TODAY'S DATE (Mo/Da/Yr)

☐ 1. **DEPRESSED MOOD** (Sadness, hopeless, gloomy, pessimistic, weeping, worthless. Behavior: Facies, postures, weeping, voice.)
0 - NOT DEPRESSED
1 - DOUBTFUL or TRIVIAL: Behavioral evidence and feeling elicited only on direct questioning.
2 - MILD: Occasional weeping. Feeling states elicited only on direct questioning
3 - MODERATE: Obvious behavioral evidence, frequent weeping and behavioral state comprises a large part of spontaneous communication.
4 - SEVERE: Exhibits virtually only this feeling state in spontaneous verbal and non-verbal behavior in the presence of severe retardation.

☐ 7. **WORK AND INTEREST:** (Apathy: loss of pleasure and interest in work, hobbies, social activities, recreations. Inability to obtain satisfaction, decreased performance at work and in home duties. Do not rate fatigue or loss of energy.)
0 - NO DISTURBANCE
1 - DOUBTFUL or TRIVIAL: Feels incapable, listless, is less efficient, distinguish from fatigue and loss of energy.
2 - MILD: Has to push self to undertake normal activities. Loss of interest, sees no point, gets less satisfaction.
3 - MODERATE: Clearly decreased efficiency, gets less done, e.g. at work or home spends less time at working usual chores or recreations. Rate 3 if patient does not engage in activities spontaneously, marked loss of personal tidiness.
4 - SEVERE: Stopped working because of illness. Does not shave, bathe, etc. Does not take part in usual activities even when urged by others or does not perform routine chores unassisted.

☐ 14. **LOSS OF LIBIDO:** (Rate only definite changes associated with illness)
0 - ABSENT: No loss or inadequate information.
1 - MILD to INFREQUENT: Loss of libido; impaired sexual performance.
2 - OBVIOUS and SEVERE: Complete loss of sexual appetite.

☐ 12. **LOSS OF APPETITE:**
0 - ABSENT
1 - MILD: Infrequent symptoms, eats without encouragement from others, food-intake about normal.
2 - OBVIOUS AND SEVERE: Marked reduction of appetite and food intake, difficulty eating without urging from others

☐ 17. **WEIGHT LOSS:** (Since onset of illness, or since last visit.)
0 - ABSENT:
1 - DOUBTFUL or TRIVIAL: Less than 5 pounds.
2 - OBVIOUS and SEVERE: Greater than or equal to 5 pounds

☐ 4. **INITIAL INSOMNIA:** (Difficulty getting to sleep after going to bed)
0 - ABSENT
1 - MILD, TRIVIAL, INFREQUENT: Less than 30 minutes.
2 - OBVIOUS AND SEVERE: More than 30 minutes on most nights.

☐ 5. **MIDDLE INSOMNIA:** (Difficulty staying asleep 12 midnight - 3 am)
0 - ABSENT
1 - MILD, INFREQUENT: Patient complains of being restless and disturbed during the night. If wakes to void, unable to return to sleep quickly.
2 - OBVIOUS AND SEVERE: Patient wakes once or more after being asleep and has difficulty sleeping again. Any getting out of bed (other than to void) rates 2. (Same for smoking or reading in bed on waking).

6. **DELAYED INSOMNIA:** (Early morning awakening.)
0 - ABSENT
1 - MILD, INFREQUENT: Wakes earlier than usual but eventually sleeps again until normal time of rising.
2 - OBVIOUS AND SEVERE: Wakes 1-3 hours before usual time and is unable to sleep again.

13. **ANERGIA:** (Fatigability; feels tired or exhausted; loss of energy; heavy, dragging feelings in arms or legs.)
0 - ABSENT
1 - MILD: Infrequent, feelings noted but not marked
2 - OBVIOUS AND SEVERE: Tires very quickly; exhausted much of the time, spontaneously mentions these symptoms.

2. **GUILT FEELINGS:** (Pathological guilt not rationalizing self-blame.)
0 - ABSENT
1 - DOUBTFUL OR TRIVIAL: Feelings of self-reproach or letting people down.
2 - MILD: Ideas of guilt spontaneously expressed.
3 - MODERATE: Belief that illness may be punishment, ruminations over past errors or sins, may state that illness and suffering are deserved.
4 - SEVERE: Guilty delusions, accuses self of unlikely or impossible blame, asks to be killed because of delusional thoughts, may have accusing and denouncing auditory or visual hallucinations, or conviction of imminent executions. May be convinced that presence is making others ill.

3. **SUICIDE:** (Rate for feeling or behavior during past week.)
0 - ABSENT
1 - DOUBTFUL or TRIVIAL: In response to direct questioning says life is empty, not worth living.
2 - MILD: Recurrent thoughts of death, death wishes spontaneously given or elicited only by questioning.
3 - MODERATE: Includes (2) together with active suicidal thoughts, or behavior indicative of same, e.g. isolation, suicide gestures or threats or discussions with others.
4 - SEVERE: Suicide attempt.

10. **ANXIETY (PSYCHOLOGICAL):** (Tense, unable to relax, irritable, easily started, worrying over trivia (distinguish from morbid depressive ruminations); Phobic symptoms: apprehensive of impending doom; fear of loss of control; panic episodes.)
0 - ABSENT
1 - DOUBTFUL OR TRIVIAL: Expresses feeling states only on direct questioning. Few symptoms and low frequency.
2 - MILD: Spontaneously expresses feeling states. Good control and non incapacitating.
3 - MODERATE: Behavioral evidence of anxiety (distinguish from agitation); spontaneous expression of feeling states in significant number of frequency.
4 - SEVERE: Feeling states comprise large part of spontaneous verbal and non-verbal communication, panic episodes observed.

11. **ANXIETY (SOMATIC):** (Physiological concomitants of anxiety, (i.e. effects of autonomic overactivity, "butterflies", indigestion, stomach cramps, belching, diarrhea, palpitations, hyperventilation, paresthesia, sweating, flushing, tremor, headache, urinary frequency.) Panic symptoms should be rated.)
0 - ABSENT
1 - DOUBTFUL or TRIVIAL: Minor symptoms elicited by direct questioning.
2 - MILD: Spontaneously describes symptoms, which are not marked or incapacitating.
3 - MODERATE: Greater number and frequency of symptoms than (2). Accompanied by more subjective distress and serve to impair normal functioning.
4 - SEVERE: Symptoms are numerous, persistent and incapacitating much of the time, or panic attacks almost daily.

15. **HYPOCHONDRIASIS:** (Morbid preoccupation with real or imagined bodily symptoms or functions.) 0 -
0 - ABSENT
1 - MILD: Some preoccupation with bodily functions and physical symptoms. (Trivial or doubtfully pathological; score 1).
2 - MODERATE: Much attention given to physical symptoms. Patient expresses thoughts of organic disease with tendency to "somatize" clinical presentation.
3 - SEVERE: Convictions or organic disease to explain present condition e.g. brain tumor, cancer.
4 - EXTREME: Hypochondriacal delusions, often with guilty association, e.g. of syphilus, worms eating head, rotting inside, bowels blocked and will never function again, infecting other persons, etc.

16. **LOSS OF INSIGHT:** (Denial of "nervous" illness. Attributes illness to virus, overwork, climate, physical symptoms. Does not recognize symptoms as "nervous" in origin.)
0 - ABSENT
1 - DOUBTFUL,MILD: Some denial.
2 - OBVIOUS and SEVERE: Denies being ill at all; strong conviction that illness is not nervous in origin. Delusional patients (guilty, hypochondriasis) rate (2) by definition, as do those with fixed depressive ideation.

8. **RETARDATION:** (Psychomotor. Slowing of thought, speech and movement should be rated by both observation and self-report.)
0 - ABSENT
1 - MILD: Slight flattening of affect, fixity of expression
2 - MODERATE: Monotonous voice, delayed in answering questions, tends to sit motionless.
3 - SEVERE: Retardation prolongs interview to a marked degree, slowness of movement and gait, with diminished associated movement. Abnormal time to complete self-ratings.
4 - EXTREME: Depressive stupor, interview impossible.

9. **AGITATION** (Psychomotor, in mild form can be present together with mild retardation. May also have motor agitation with verbal retardation. Rate by both observing self report.)
0 - ABSENT
1 - MILD: Fidgety at interview, clenching fists or side of chair, kicking feet
2 - MODERATE: Wringing hands, biting lips, pulling hair, gesturing with arms, picking at hands and clothes, restlessness with some pacing.
3 - SEVERE: Includes features of (2). In addition cannot stay in chair during interview. Much pacing.

17 ITEM TOTAL SCORE_____

DATE:_____ NAME:_____

MILLION VISUAL ANALOGUE SCALE

PLEASE MAKE AN "X" ALONG THE LINE TO SHOW HOW FAR FROM NORMAL TOWARD THE WORST POSSIBLE SITUATION YOUR PAIN PROBLEM HAS TAKEN YOU DURING THE PAST WEEK.

1. How bad is your pain?

no pain — worst possible

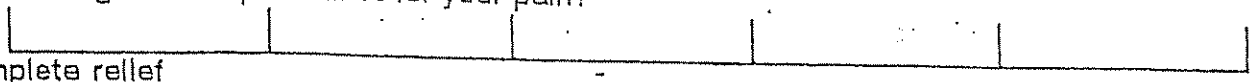
2. How bad is the pain at night?

no pain — worst possible

3. Does the pain interfere with your lifestyle?

no problem — total change in lifestyle

4. How good are pain killers for your pain?

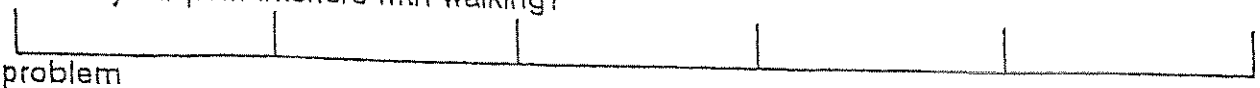


complete relief — no relief

5. How stiff is your injured area?

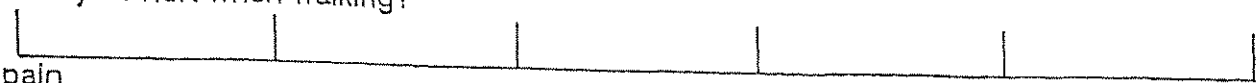
no stiffness — worst possible stiffness

6. Does your pain interfere with walking?



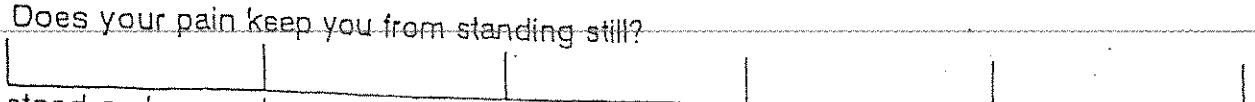
no problem — cannot walk

7. Do you hurt when walking?



no pain — worst possible pain

8. Does your pain keep you from standing still?

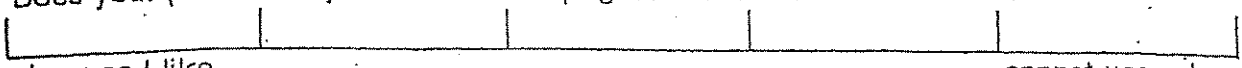


can stand as long as I want — cannot stand at all

9. Does your pain keep you from twisting?

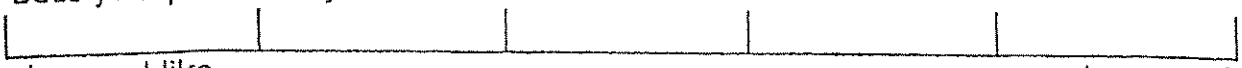
no problem — cannot twist

10. Does your pain allow you to sit in an upright hard chair?



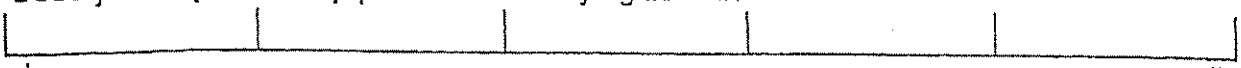
sit as long as I like — cannot use a hard chair at all

11. Does your pain allow you to sit in a soft arm chair?



sit as long as I like — cannot use a soft chair at all

12. Does your injured body part hurt when lying in bed?



no pain — no relief at all

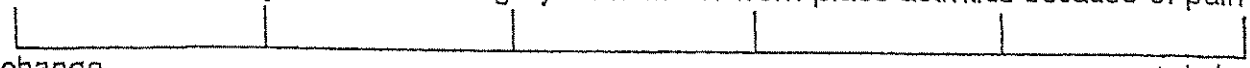
13. How much does your pain limit your normal lifestyle?

no limit — cannot do anything

14. Does your pain interfere with your activities of daily living or work?

no problem — major problem

15. How much have you had to change your home or work place activities because of pain?



no change — a great deal of change

OSWESTRY

NAME: _____ DATE: _____

How long have you had your pain? _____Years _____Months _____Weeks

Please read:
This questionnaire has been designed to give the doctor information as to how your pain has affected your ability to manage in everyday life. Please answer every section, and mark in each section only the *one box* which applies to you. We realize you may consider that two of the statements in any one section relate to you, but please just *mark the box which most closely describes your problem.*

Section 1 – Pain Intensity
- ☐ I can tolerate the pain I have without having to use painkillers.
- ☐ The pain is bad but I manage without taking pain killers.
- ☐ Pain killers give complete relief from pain.
- ☐ Pain killers give moderate relief from pain.
- ☐ Pain killers give very little relief from pain.
- ☐ Pain killers have no effect on the pain and I do not use them.

Section 2 – Personal Care (Washing, Dressing, etc)
- ☐ I can look after myself normally without causing extra pain.
- ☐ I can look after myself normally but it causes extra pain.
- ☐ It is painful to look after myself and I am slow and careful.
- ☐ I need some help but manage most of my personal care.
- ☐ I need help every day in most aspects of self-care.
- ☐ I do not get dressed, wash with difficulty and stay in bed.

Section 3 - Lifting
- ☐ I can lift heavy weights without extra pain.
- ☐ I can lift heavy weights but it gives extra pain.
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, e.g., on a table.
- ☐ Pain prevents me from lifting heavy weights but I can manage light to medium weights if they are conveniently positioned.
- ☐ I can lift only very light weights.
- ☐ I cannot lift or carry anything at all.

Section 4 - Walking
- ☐ Pain does not prevent me from walking any distance.
- ☐ Pain prevents me walking more than a mile
- ☐ Pain prevents me walking more than ½ mile.
- ☐ Pain prevents me walking more than ¼ mile.
- ☐ I can only walk using a stick or crutches.
- ☐ I am in bed most of the time and have to crawl to the toilet.

Section 5 - Sitting
- ☐ I can sit in any chair as long as I like
- ☐ I can only sit in my favorite chair as long as I like
- ☐ Pain prevents me sitting more than 1 hour.
- ☐ Pain prevents me sitting more than ½ hour.
- ☐ Pain prevents me sitting more than 10 minutes.
- ☐ Pain prevents me sitting at all.

Section 6 - Standing
- ☐ I can stand as long as I want without extra pain.
- ☐ I can stand as long as I want but it gives me extra pain.
- ☐ Pain prevents me from standing for more than 1 hour.
- ☐ Pain prevents me from standing for more than 30 minutes.
- ☐ Pain prevents me from standing for more than 10 minutes.
- ☐ Pain prevents me from standing at all.

Section 7 - Sleeping
- ☐ Pain does not prevent me from sleeping well.
- ☐ I can sleep well only by using tablets.
- ☐ Even when I take tablets I have less than six hours sleep.
- ☐ Even when I take tablets I have less than four hours sleep.
- ☐ Even when I take tablets I have less than two hours sleep.
- ☐ Pain prevents me from sleeping at all.

Section 8 – Sex Life
- ☐ My sex life is normal and causes no extra pain.
- ☐ My sex life is normal but causes some extra pain.
- ☐ My sex life is nearly normal but is very painful.
- ☐ My sex life is severely restricted by pain.
- ☐ My sex life is nearly absent because of pain.
- ☐ Pain prevents any sex life at all.

Section 9 – Social Life
- ☐ My social life is normal and gives me no extra pain.
- ☐ My social life is normal but increases the degree of pain.
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests (e.g., dancing, etc.)
- ☐ Pain has restricted my social life and I do not go out as often.
- ☐ Pain has restricted my social life to my home.
- ☐ I have no social life because of pain.

Section 10 - Traveling
- ☐ I can travel anywhere without extra pain.
- ☐ I can travel anywhere, but it gives me extra pain.
- ☐ Pain is bad but I manage journeys over two hours.
- ☐ Pain restricts me to journeys of less than one hour.
- ☐ Pain restricts me to short necessary journeys under 30 minutes.
- ☐ Pain prevents me from traveling except to the doctor or hospital.

COMMENT: _____

AME: _____ DATE: _____

_____ DATE OF BIRTH: _____

ICLE ONE: MALE FEMALE

SF-36 HEALTH SURVEY

INSTRUCTIONS: This survey asks for your views about your health. This information will help keep track of how you feel and how well you are able to do your usual activities.

Answer every question by marking the answer as indicated. If you are unsure about how to answer a question, please give the best answer you can.

1. In general, would you say your health is:

(circle one)

Excellent 1

Very good 2

Good 3

Fair 4

Poor 5

2. Compared to one week ago, how would you rate your health in general now?

(circle one)

Much better now than one week ago 1

Somewhat better now than one week ago 2

About the same as one week ago 3

Somewhat worse now than one week ago 4

Much worse now than one week ago 5

3. The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

(circle one number on each line)

ACTIVITIES	Yes, Limited A Lot	Yes, Limited A Little	No, Not Limited At All
a. **Vigorous activities**, such as running, lifting heavy objects, participating in strenuous sports	1	2	3
b. **Moderate activities**, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf	1	2	3
c. Lifting or carrying groceries	1	2	3
d. Climbing **several** flights of stairs	1	2	3
e. Climbing **one** flight of stairs	1	2	3
f. Bending, kneeling, or stooping	1	2	3
g. Walking **more than a mile**	1	2	3
h. Walking **several blocks**	1	2	3
i. Walking **one block**	1	2	3
j. Bathing or dressing yourself	1	2	3

4. During the past week, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

(circle one number on each line)

	YES	NO
a. Cut down on the **amount of time** you spent on work or other activities	1	2
b. **Accomplished less** than you would like	1	2
c. Were limited in the **kind** of work or other activities	1	2
d. Had **difficulty** performing the work or other activities (for example, it took extra effort)	1	2

5. During the past week, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

(circle one number on each line)

	YES	NO
a. Cut down the **amount of time** you spent on work or other activities	1	2
b. **Accomplished less** than you would like	1	2
c. Didn't do work or other activities as **carefully** as usual	1	2

6. During the past week, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbors, or groups?

(circle one)

Not at all 1

Slightly 2

Moderately 3

Quite a bit 4

Extremely 5

7. How much bodily pain have you had during the past week?

(circle one)

None 1

Very mild 2

Mild 3

Moderate 4

Severe 5

Very severe 6

8. During the past week, how much did pain interfere with your normal work (including both work outside the home and housework)?

(circle one)

Not at all 1

A little bit 2

Moderately 3

Quite a bit 4

Extremely 5

9. These questions are about how you feel and how things have been with you during the past week. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past week -

(circle one number on each line)

	All of the Time	Most of the Time	A Good Bit of the Time	Some of the Time	A Little of the Time	None of the Time
a. Did you feel full of pep?	1	2	3	4	5	6
b. Have you been a very nervous person?	1	2	3	4	5	6
c. Have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
d. Have you felt calm and peaceful?	1	2	3	4	5	6
e. Did you have a lot of energy?	1	2	3	4	5	6
f. Have you felt downhearted and blue?	1	2	3	4	5	6
g. Did you feel worn out?	1	2	3	4	5	6
h. Have you been a happy person?	1	2	3	4	5	6
i. Did you feel tired?	1	2	3	4	5	6

10. During the past week, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)?

(circle one)

All of the time 1

Most of the time 2

Some of the time 3

A little of the time 4

None of the time 5

11. How TRUE or FALSE is each of the following statements for you?

(circle one number on each line)

	Definitely True	Mostly True	Don't Know	Mostly False	Definitely False
a. I seem to get sick a little easier than other people	1	2	3	4	5
b. I am as healthy as anybody I know	1	2	3	4	5
c. I expect my health to get worse	1	2	3	4	5
d. My health is excellent	1	2	3	4	5

ame:_____ Date:_____

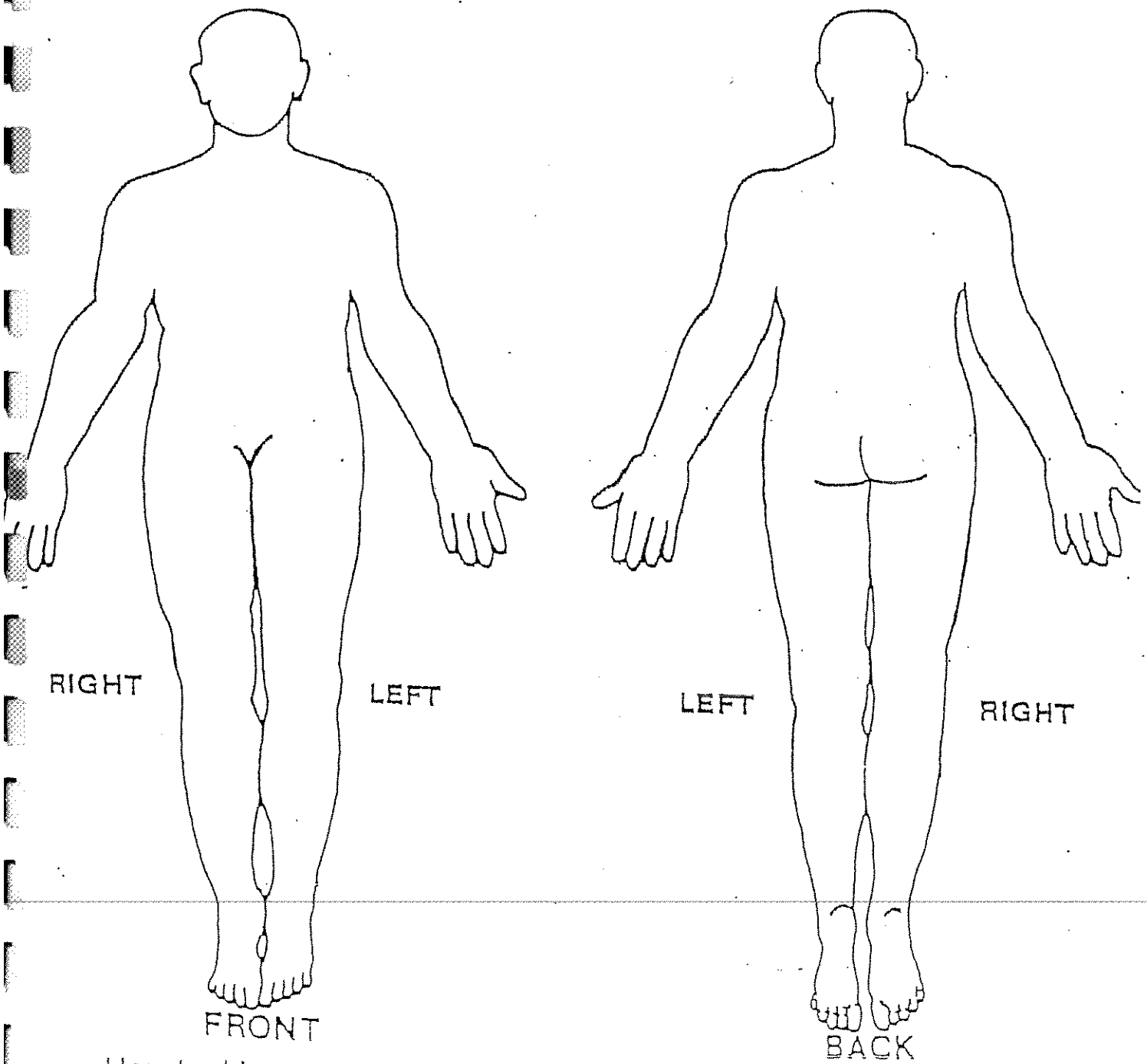
QUANTIFIED PAIN DRAWING (PAIN INTENSITY)

THINKING ABOUT YOUR AVERAGE PAIN IN THE LAST 7 DAYS, ANSWER THE FOLLOWING QUESTIONS.

Draw the location of your pain on the body outlines and mark whether it is all back/neck or all arm/leg.

ALL
ACK/NECK

ALL
ARM/LEG



How bad is your pain?

NO PAIN

WORST POSSIBLE

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VITAE

Cindy Lee Kidner was born in St. Charles, Missouri on April 20, 1972, the daughter of Judy Gail Kidner and Raymond Lee Kidner. After graduating from Grove City High School, Grove City, Ohio in 1990, she attended Ohio University in Athens, Ohio. Following her freshman year, she transferred to The Ohio State University in Columbus, Ohio. She received the degree of Bachelor of Science with a major in psychology and a minor in zoology in June, 1994. In August of that year, she moved to Denton, Texas, where she attended the University of North Texas. In August, 1997 she received the degree of Master of Arts in Counseling Psychology. During the following three years, she was employed as a research coordinator in the Department of Psychiatry at the University of Texas-Southwestern Medical Center at Dallas and at The Folgelson Neuroscience Center at Presbyterian Hospital in Dallas. In August, 2000, she entered the Graduate School of Biomedical Sciences at the University of Texas-Southwestern Medical Center at Dallas. In August, 2004, she completed her doctoral internship through the University of Texas-Southwestern Medical Center at Dallas, with rotations at Southern Methodist University and Parkland Memorial Hospital. Since August, 2004, she has been employed as the project coordinator at the Psychosocial Research and Depression Clinic in the Department of Psychiatry at the University of Texas-Southwestern Medical Center. In April, 2006, she became engaged to Christopher Michael Pilcher of Richardson, Texas. She was awarded the degree of Doctor of Philosophy in Clinical Psychology in December, 2007.

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